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Chapter 6

Prevention, Diagnosis and Treatment of Caries and Non-Carious Lesions in Orthodontic Patients

Andréa Cristina Barbosa da Silva,
Diego Romário da Silva, Ana Marly Araújo Maia,
Pierre Andrade Pereira de Oliveira,
Daniela Correia Cavalcante Souza and
Fábio Correia Sampaio

Additional information is available at the end of the chapter

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1. Introduction

1.1. Caries risks and orthodontic treatment

Although orthodontic treatment has many recognized benefits, including improvement in dental health, function, appearance, and self-esteem; nevertheless, orthodontic appliances can cause unwanted complications if adequate care is not taken during the treatment.

It is important that patients are aware of these potential risks so that they can know their responsibilities and expectations on them during the treatment. This ensures in achieving successful results without any adverse effects after the completion of orthodontic treatment [1].

Some of potential intraoral risks that orthodontic treatment carries with it are enamel demineralization/caries, adverse effects to the periodontal tissues, tissues damage, enamel fractures, enamel wear (abrasion/erosion), root resorption, pulp reactions, and allergic reactions to nickel and latex.

These potential complications are easily avoidable by undertaking certain precautions and timely interventions by both the orthodontist and the patient [1]. However, the lack of patient's cooperation and consequent poor oral hygiene is one of the main challenges in orthodontic practice [2].

The irregular surfaces of brackets, bands, wires and other attachments also limit naturally occurring self-cleansing mechanisms, such as the movement of the oral musculature and saliva [3, 4, 5]. The insertion of fixed orthodontic appliances creates stagnation areas for plaque and makes tooth cleaning more difficult [6, 7]. These promote retention niches that pose an increased risk of caries [5, 8].

Enamel demineralization around the brackets and bands is one adverse side effect of major clinical relevance [4, 7, 9, 10, 11]. White spot lesions develop as a result of prolonged plaque accumulation on the affected surface, commonly due to inadequate oral hygiene [6].

All these changes in the local environment appear to favor colonization of aciduric bacteria such as *Streptococcus mutans* and *lactobacilli* [3].

Clinically, formation of white spots around orthodontic attachments can occur as early as 4 weeks into treatment [6, 12] and their prevalence among orthodontic patients ranges from 2% to 96% [4, 5, 8, 12, 13].

Several studies have reported significant increase in the prevalence and severity of demineralization in patients after orthodontic treatment [12, 14, 15, 16].

Whilst the demineralized surface remains intact, there is a possibility of remineralization and lesion reversal [17]. It is generally believed that, when the appliances are removed and oral hygiene is restored, these lesions regress [9].

Some white spot lesions may remineralize and return to normal conditions or at least to a visually acceptable appearance. However, white spot lesions may also persist, resulting in aesthetically unacceptable result, as opaque and/or hypoplastic areas [18, 19]. In severe cases, restorative treatment may be required [20].

Besides incipient carious lesions, common damage that also needs attention from the orthodontist includes enamel wear by abrasion and erosion. Wear of enamel against both metal and ceramic brackets may occur. Stainless steel brackets tend to induce less enamel abrasion than ceramic brackets [21]. In patients with deep bite, use of bite planes is advocated to minimize interference and the subsequent risk of enamel abrasion [22]. Any enamel erosion must be recorded prior to treatment commencing and appropriate dietary advice should be given to minimize further tooth substance loss. Carbonated drinks and acid pure juices are the commonest causes of erosion and should be avoided in patients with fixed appliances [17].

Treatment can only be justified and success can only be ensured thanks to thorough understanding and controlled management of the potential associated risks, including those of carious and non-carious lesions. Preventive measures are available and have been scientifically validated and therefore, they should be implemented before and during orthodontic treatment [20].

Among the most common and effective preventive measures are special oral hygiene instructions, including a recommendation for using high-fluoride toothpaste, fluoride mouth rinse, and products with high-fluoride content [23].

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Just importantly as preventing, intercepting and correcting problems related to tooth alignment and bone growth, is to provide patients undergoing orthodontic treatment, the maintenance of their oral health as a whole [18].

The provision of oral hygiene instruction, monitoring and motivation of all patients under orthodontic treatment is the responsibility of the orthodontist, the General practitioner and his/her entire team.

In this context, professionals in the field of orthodontics should be prepared to prevent and recognize the risks and diseases that may arise due to the use of orthodontic apparatus for their patients, as well as properly dealing with possible complications with appropriate measures.

2. Etiology and diagnosis of carious and non-carious lesions

Despite the still high prevalence of dental caries in the world population [24], increase in the prevalence of non-carious lesions has also been observed in recent years [25]. Especially, the carious white spot lesions may present increased risk of developing in teeth during orthodontic treatment due to the installation of devices in orthodontic patients [26].

The carious and non-carious lesions differ in their etiology, basically due to the former being associated with bacteria, while the latter have no relationship with microorganisms. Thus, the term dental caries is used to describe the results of a chemical dissolution of tooth structure caused by the metabolic events that occur in the biofilm, which covers the affected areas [27]. In non-carious lesions, loss of tooth structure is caused by multiple factors and of complex diagnosis due to difficulty in identifying the primary cause; however, it is not associated with the presence of microorganisms [28, 29].

In other words, the dental caries result from a change in the ecology and metabolic activity of the biofilm, in which a balance between tooth mineral and biofilm is established. Importantly, the biofilm formed that grows ubiquitously in solid surfaces does not necessarily result in the development of clinically visible caries. However, the biofilm is a prerequisite for the occurrence of these injuries. In general, the dental caries are result of physiological imbalance between the tooth mineral and biofilm fluid [27].

Initially, carious lesions can develop into any dental site of the oral cavity, where a biofilm can develop and remain for a period. It is erroneous talk on surfaces more or less susceptible to decay, since it can generate the belief that certain tooth parts are "tougher" or "less prone" to developing dental caries in function of variations in the chemical and structural composition [30, 31].

However, it is worth noting that the risk factor to the onset of tooth decay is higher in orthodontic patients, due to mechanical propensity of greater accumulation of waste associated with the presence of brackets and bands in the development of lesions. These "protected" sites are relatively protected areas of the mechanical influence of the tongue, cheeks, abrasive foods

and brushing. Thus, these are the most likely sites to develop the lesion, because biofilms can stagnate for a long time [27]. Thus, some tests for risk assessment of caries can be useful in these patients, as the computerized program Cariograma® [32], as well as others developed especially for these patients, as an immediate chair-side test, displaying the intraoral lactic acid production of cariogenic bacteria. Recently, this test was considered useful to evaluate the risk for dental caries in patients with orthodontic treatment. Furthermore, specific clinical approaches for this group of patients are required [33].

In this context, it becomes important for the patient, prior to installation of the appliance, to have a dental and periodontal examination adequate for a correct diagnosis of dental and/or periodontal injuries [34], with referral for some specialists in periodontics and/or dentistry, if necessary.

Overall, the tactile–visual examination of the teeth is currently considered sufficient for a proper diagnosis of caries. Thus, it should be conducted systematically after each quadrant of the mouth has been isolated with cotton rolls and with a suction device for preventing the teeth wetting by saliva. This type of examination requires good lighting and clean and dry teeth. The teeth can be examined with the help of a mirror and a probe with a blunt tip, whose purpose is to remove biofilm and verify the lesion surface texture [35].

Moreover, the increased knowledge of the dynamic chemical processes of caries has stimulated the development of a new method for visual–tactile diagnosis of caries based on verification of the lesion activity. This method evaluates the surface features of the lesion: the activity, reflected by the surface texture, and surface integrity, expressed by the presence and absence of a cavity or microcavity on the surface [36]. The pathobiological principle of the method is based on the observation that enamel surface characteristics change in response to changes in the biofilm activity covering the tooth surface [37].

It is noteworthy that one should not force the tooth tissue with the probe, as there is a risk of causing irreversible damage to the surface area of incipient lesion [38] damage. Additional resources in tactile–visual diagnosis of caries include fiber optic transillumination, tooth separation and teeth expansion, especially for professionals in the labor market for longer [35].

Regarding the use of radiography in the diagnosis of carious lesions, the most commonly used is the *bitewing*. Although routine use of bitewing radiography could help identify teeth with hidden caries to allow early and conservative treatment, it is inaccurate in diagnosing occlusal enamel caries [39]. However, small mineral loss cannot be detected in the radiographs, and the additional diagnostic yield in bitewing radiography is confined to lesions diagnosed at the level of cavity\dentin. In addition, the radiographic examination fails to determine the lesion activity and cavity formation, and suffers from a high number of false-positive diagnoses [35].

Among the unknown carious lesions, the most important and can be associated to orthodontic treatment is tooth erosion that is caused by dissolution and removal of an ultrathin layer of enamel whenever exposed to the acidic challenge. In early enamel caries with the tipical subsurface lesion, mineral loss and changes predominantly occur in the body of the lesion below the pseudo-intact surface layer [40].

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Only the surfaces directly exposed to acid are affected. The teeth are eroded and where foods and fresh and acidic beverages reach the enamel surfaces prior to being neutralized and diluted by saliva [40]. In addition, tooth erosion can also be caused by acid stomach content that can return to the mouth due to an eating disorder or gastroesophageal reflux [41, 42]. Vomit may be a feature of an eating disorder, which can cause erosion and bulimia [43].

Early identification of patients with erosion is important, and thus the dentist may assist the patient in establishing the cause. Sometimes, the patient's complaint about tooth sensitivity directs attention to the problem because the dentin is exposed. However, the dentist may have diagnosed even early lesions. In particular, lingual surfaces of the maxillary incisors should be examined because they are the most eroded. Thus, a careful anamnesis is essential to try to determine the probable cause, and counseling is mandatory [40]. Even if the cause is not identified in the anamnesis, for being difficult to determine [44], counseling is provided to the patient [40].

Enamel erosion (decalcification) around orthodontic brackets bonded to the teeth has been a concern for orthodontists, since this decalcification is the first step to more greater damage to the enamel. These erosive lesions can also lead to infiltration of resin restorations and shear of orthodontic brackets [45].

Finally, it is interesting to note that despite the self-ligated brackets being considered more hygienic than those with elastic, no differences in terms of white spot lesion formation were found between conventional and self-ligating wire brackets straight; no differences in terms of white spot lesion formation were found between conventional straight wire and self-ligating brackets; and white spot lesion formation does depend largely on patients' oral hygiene status, the type of bracket or ligation used [46].

3. Control of dental biofilm associated with orthodontic treatment

It is known that the goal of orthodontic treatment is to make corrections to the occlusion and also provide improvements in dental aesthetics. However, this treatment can provide increased chances of developing dental caries and/or periodontal disease, which can harm the patient and the progress of treatment. The development of these diseases may be mainly associated with a deficient biofilm removal due to the impediment of a proper hygiene, caused by intraoral devices [47]. Wires, brackets and orthodontic bands serve as a biofilm reserve and source of infections since it provides increased biofilm retainer niches due to its configuration and surface. Factors such as the type of material of the bracket, the type of ligature used, taping or gluing of orthodontic accessories, diet and biofilm control can influence the capacity of proliferation of microorganisms and subsequent development of caries [48, 49, 50].

Wires, brackets and bands can become centers of tooth decay if the patient fails to maintain oral hygiene. Studies have shown increased prevalence of great severity of enamel demineralization after orthodontic treatment when compared to individuals not subjected to this treatment. However, once active orthodontic treatment has been completed, the

demineralization process is normally expected to decelerate due to a change in local environmental factors [9].

Diet can be defined as the usual intake of food and drinks daily taken by any person, and may have a local effect in the mouth by its reaction with the tooth enamel surface, serving as a substrate for cariogenic microorganisms, particularly if it is rich in sucrose [51]. The frequency of intake, the sugar content and consistency of meals are decisive for caries formation [52]. On this basis, it is easy to observe that a poor diet associated with poor hygiene and the use of orthodontic appliance may be potential factors that influence the development of caries and inflammatory processes. It is the responsibility of the orthodontist to instruct the patient regarding these factors, especially at the point that relates to oral hygiene and plaque control. In addition, the orthodontist must have diagnostic accuracy to know when to refer the patient to the periodontist, when necessary.

Regarding the biofilm control, it is known that this procedure may be performed by mechanical and chemical means. The mechanical control consists of the biofilm removal using a proper brushing technique associated with the use of toothpaste and auxiliary materials, such as dental floss or tape [53]. Brushing, although extremely important, it is a relatively simple technique. There is no ideal brushing; however, there are features that facilitate oral hygiene procedures, such as the presence of multi-tufts, small head, rounded soft bristles, according to [54]. Brushing is extremely effective in controlling biofilm and reducing the risk of caries; however, to obtain maximum effectiveness in hygiene is necessary to consider factors such as the frequency and technique of brushing, hand skills and motivation of patients [55]. It is for the orthodontist to motivate the patient, who is considered to have a potential for developing caries and inflammation, to have a proper hygiene for oral health during and after treatment. The plaque disclosure can be a valuable alternative to help this motivation.

Chemical control is only an adjunct to mechanical control. Thus, it should not be used as a substitute for mechanical control. Chemical control can be performed in the prophylactic or therapeutic sense, depending on the patient's oral health status [56]. Generally, mouthwashes containing sodium fluoride [57], chlorhexidine [58], cetylpyridinium chloride [59] and essential oils [60] are used. Studies have shown that the use of commercial mouthwashes along with mechanical oral hygiene, guidance and motivation is the most appropriate management for maintenance of oral health in orthodontic patients [61]. Furthermore, the use of fluorides, in its various forms of presentation, is extremely important for dental remineralization and prevention of demineralization that might cause carious lesions [62]. It is important that the orthodontist can make use of these resources, adapting to the reality of each patient to provide orthodontic treatment free of complications.

4. Treatment of carious and non-carious lesions in orthodontics patients

Regarding all factors previously discussed, it is also important to the orthodontist to be aware of techniques and products that can prevent and treat, as a nonoperative procedure, the development of carious and non-carious lesions during orthodontics treatment. Management

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of white spot lesions begins with a good oral hygiene regime and needs to be associated with use of fluoride agents, as fluoridated toothpaste, fluoride-containing mouth rinse, gel, varnish, bonding materials, elastic ligature, CPP-ACP, antiseptics, LASER, tooth whitening, resin infiltration and micro-abrasion [63]. As demonstrated, the patient's behavior before the increased risk is very important, although the professional should supervise and also choose the better technique for each patient.

One of the first important points, which only depends on the orthodontist technique, is to choose the best adhesive composite type to bracket bonding, and also control and reduce the adhesive composite excess around the brackets. Incorporating preventive agents in orthodontic bonding composite seems to be a potential method to reduce white spot lesions during orthodontic treatment [64]. Bioavailable minerals from cement-containing amorphous calcium phosphate (ACP), as the Aegis–Ortho with 28% ACP fillers (Bosworth, Skokie Companies) [65], have been described to facilitate remineralization and also inhibit lesion development. There are also other cements-containing fluorides, as Pro-seal and Quick Cure (Reliance Orthodontic Products), and Light cure composite without fluoride or ACP, as Transbond XT (3M Unitek) has been also used followed by application of Varnish fluoride [66].

Researches are fundamental to compare and establish better options for each patient, [64], comparing different cements under similar in-vitro conditions to prevent incipient caries lesions next to brackets on teeth, observed that ACP into orthodontics adhesive material provided reduction in bacterial adhesion and lesion depth formation, with similar results to the Quick Cure adhesive with fluoride. Although Behnan et al., (2010) [66] having demonstrated that both light-cured filled resin (Pro-seal) and fluoride varnish (Varnish) might prevent enamel demineralization, even positive effects of Aegis-Ortho and MI Paste were not significant. The development of cements with preventive agents that can be effectively delivered in the long term encourages further investigation and also *in vivo* performance to evaluate these materials in clinical application, and also their importance to be applied in the orthodontic patient who lacks compliance with oral hygiene.

Attention is fundamental to position braces accurately, but it is also fundamental to remove composite excess between brackets and teeth with a scalar or another instrument, avoiding adding more area to plaque retention. One other option to this objective, commonly used when braces are placed on the lingual surface, is the indirect transfer system. This technique was evaluated in vivo during 4 months, if it could help reducing plaque accumulation around the brackets and consequently, the surrounding enamel demineralization. Concerning the differences between direct and indirect bonding, there was a significantly greater presence of white spots at the end of the treatment and a greater plaque accumulation around the braces where the direct bonding was used [67]. It was concluded by authors that, in a clinical point of view, the indirect bonding technique could help to reduce the enamel demineralization.

The bracket model has been also changed through the years, and the use of elastic ligatures has been also discussed about the association with white spot lesions development. The self-ligation bracket that does not require elastic ligatures was firstly introduced also as a hygienic option to minimize dental plaque retention and microbial flora. Although a clinical trial that evaluated 20 boys about plaque index, bleeding on probing and others, before bonding, after

1 week and 3 months after bonding, found that all clinical parameters showed statistically significant increases from before bonding, although self-ligation brackets and conventional brackets ligated with stainless steel ligatures did not differ with regard to dental plaque retention [68]. These results are similar to [46], that also revealed no statistically significant differences on white spot lesion development between conventional straight wire and self-ligating brackets, and also highlighted that WSL formation does depend largely on patients' oral hygiene status, not the type of bracket or ligation used.

In the light of the difficulties involved in clinically detecting, monitoring and managing dental demineralization, researchers are actively searching for new agents for prevention as preventive programs should be implemented to avoid the potentially detrimental [69]. The basic home care oral hygiene with manual toothbrush and fluoride toothpaste (1100ppm of fluoride), dental floss use, and patient's compliance seem to be an efficient preventive procedure, although it is also necessary to individualize and consider high risk of patients that even before and after some months do not have a satisfactory oral hygiene. For these patients, it is necessary to add some other alternatives such as the association of antimicrobial therapies.

The application of antimicrobial therapies with common commercially produced antibacterial agents such as varnishes, mouthwashes, or dentifrice gel formulations, particularly with chlorhexidine (CHX), have been found as beneficial preventive strategy for reducing undesirable outcomes of fixed orthodontic appliances [70, 71]. Some studies investigated the effects of various concentrations of chlorhexidine on oral microflora [72], and also fluoride varnish [73]. The combination of chlorhexidine–fluoride-containing dentifrice and fluoride varnish has also been investigated, and shown to be successful and promising alternative for improving oral hygiene features of patients lacking sufficient oral hygiene characteristics during orthodontic treatment. It was also observed that a one-visit chlorhexidine varnish treatment combined with a home-based chlorhexidine–fluoride gel formulation may be implemented as a caries-prevention strategy for patients undergoing fixed appliance treatment and particularly under high risk of caries [74].

The application of clinical procedures of each material is also fundamental to effectiveness. The Cervitec®Plus (1% chlorhexidine diacetate and 1% thymol, Ivoclar Vivadent) application procedures recommends that tooth surfaces should be cleaned thoroughly dry with air syringe and isolated with cotton rolls. Three drops of Cervitec®Plus poured into a dapper dish and a thin coat of varnish applied to all of the surfaces of the bonded teeth (buccal, lingual, occlusal and proximal areas). The varnish should be dispersed with air and allowed to dry, and the cotton rolls were removed after 30 seconds. After application, patients should be instructed not to rinse, eat/drink or brush for one hour. The Cervitec Plus can also be associated with Cervitec®Gel (Ivoclar Vivadent), 0.2% chlorhexidine digluconate and 0.2% sodium fluoride (900 ppm fluoride) to brush their teeth with as a dentifrice for four weeks at home, three times a day for 2 min.

The Fluor Protector (Ivoclar Vivadent) contents 5 wt% difluorosilane corresponding to 0.7 wt % F-; application procedure also consists of tooth surface cleaned thoroughly and dried with air syringe and isolated with cotton rolls. A thin layer of Fluor Protector should be applied to all tooth surfaces (buccal, lingual, occlusal and proximal areas) using applicator, dispersed

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with air and allowed to dry, and cotton rolls but just removed after 60 seconds. After the application of varnish, patients should be told not to eat/drink or brush for 45 minutes.

Many studies have investigated the protective effect of fluoride on erosion using fluoride compounds that are widely used in caries prevention such as NaF, AmF, SnF_2, or acidulated phosphate fluoride [75]. Fluorides has also been associated to ACP (amorphous calcium phosphate), commercially as MI Paste Plus(GC Corporation, Tokyo, Japan), that use the nano-complex of the milk protein casein phosphor-peptide (CPP); this product has demonstrated preventive and remineralization properties in the caries process [76] and suggested as a mechanism to prevent or reduce enamel demineralization also in orthodontics patients [77], that following for 3 months, demonstrated that the use of MI Paste Plus for a minimum of 3 to 5 minutes each day at night after brushing helped prevent the development of new white spot lesions during orthodontics treatment and decreased the number of white spot lesions already present.

Another technique to prevent enamel demineralization for the full duration of orthodontics treatment with fixed appliances is the tooth sealant polish, as Biscover LV (Bisco), and a clinically small but statistically significant ability to prevent white spot lesion was demonstrated [66]. To apply BisCover LV, the cleaned tooth surface should be etched for 15 seconds, rinsed copiously, air-dry thoroughly. After, one thin coat of BisCover LV should be applied, evaporated for 15 seconds without air, and then light-cured for 30 seconds with a LED.

The high risk of permanent damage to the enamel structure makes the orthodontic bracket debonding and subsequent clean-up extremely important to maintain the integrity of the enamel surface [78]. Many instruments have been proposed for bracket debonding and subsequent enamel clean-up, such as Arkansas points, diamond points, multiblade burs, pliers and others. However, once none of these instruments can achieve complete clean-up without affecting the enamel surface, several protocols have been proposed [79]. Instruments and protocols must be developed and tested in order to avoid possible iatrogenic damage to the enamel structure, leaving it as close as possible to its pretreatment condition. [80].

Associated to the risk, the presence of white spot or non-carious lesion appearance around brackets increased the risk of damage and also became a disappointment to the patient after debonding. Even with all homemade care, sometimes preventive procedures are not sufficient at all. Thus, the first option after debonding brackets that should be applied as soon as possible is remineralization agent procedures. There are also several techniques that have been evaluated by in vitro and in vivo experiments that each orthodontist should be aware, although no systematic review specifically addressing remineralization of WSLs after orthodontic treatment has been published [81]. Even not being possible to review all options, this chapter presents a small review about this topic and aims at discussing the strengths and limitations of the techniques and certainly helping clinicians weigh the effectiveness of various remineralization agents.

One of the most used preventive agents is the sodium fluoride, common used in toothpaste and also in varnish. To remineralize effects, the fluoride varnish Duraphat (5 % sodium fluoride) has been applied and analyzed through a clinical trial of 110 participants followed

by 3 and 6 months, and it has been demonstrated that its effective in reversing WSLs after debonding every month during the first 6 months after debonding [16]. Authors also commented on the topical fluoride varnish that should be advocated as a routine caries prevention during orthodontic treatment.

The fluoride Varnish Duraphat application consists of this sequence, as preconized by [16]. Firstly, the subject's teeth should be clean with a toothbrush, especially the surfaces with WSLs. Secondly, excessive saliva in one or two quadrants of the mouth should be removed by cotton rolls or by using air syringe. It was not necessary to keep the tooth surface extremely dry because Duraphat could set in the presence of saliva. Thirdly, fluoride varnish should be applied onto the tooth surfaces with WSLs using a miniature cotton swab or brush, with the applicator dabbed repeatedly onto the tooth surface without contacting soft tissues. After a few minutes, a thin and clear layer is formed. Then, the next quadrants were treated in the same manner. After application, the patient should be advised not brushing their teeth or chewing food for at least 4 h after treatment; during this time, soft food and liquid might be consumed.

The casein phosphopeptide-amorphous calcium phosphate previously discussed, associated in cements and as a preventive treatment, has been also recommended to post–orthodontic population [82]. The product applied twice daily after fluoride toothpaste use for 12 weeks showed significantly regressed post–orthodontic white spot lesions [82]. The application preconized in this clinical test is very simple; after normal oral hygiene procedures using fluoridated dentifrice and a soft texture toothbrush, the patient uses a finger to apply 1g of the cream to the buccal/labial surfaces of teeth in the morning and night for 12 consecutive weeks.

However, the recently conducted research of [81] comparing the effectiveness of MI Paste Plus and PreviDent fluoride varnish (22,600 ppm of fluoride) with a standard oral hygiene regimen using toothpaste (1100 ppm of fluoride; Colgate Oral Pharmaceuticals) in terms of improving the appearance of WSLs after orthodontic treatment, found no difference of MI Paste Plus or PreviDent fluoride varnish compared to a standard oral hygiene and toothpaste regimen for ameliorating WSLs during an 8-week period. For more details, see [83].

Another new approach in treating incipient caries lesions and also non-carious lesion is the resin infiltration technique that became a treatment option for non-cavitated lesions not expected to arrest or remineralize it [45]. Infiltrate caries with low viscosity penetrate the porous body and are optimized for rapid capillary penetration [84]. The resin completely fills the pores within the tooth, replacing lost tooth structure and stopping caries and erosion progression by blocking further introduction of any nutrients into the pore system [85]. The resin infiltration is recommended for teeth that present obvious area of WSL found around the fixed orthodontic brackets but non-cavitated enamel surface. The applications procedures of the resin infiltrate (Icon caries infiltrant-Smooth Surfaces), according to the manufacturer's instructions and described by Hammad and Enan (2013) [45], recommend that firstly, a rubber dam as well as the OptraGate Lip and Cheek Retractor should be placed to protect the gingiva and to provide a dry working field to ensure the best treatment result as possible. Mylar strips should be placed securely with wedges distal to protect the neighboring teeth. Next, the pseudo-intact enamel surface layer is eroded using a 15% HCl gel (Icon-Etch), which is applied

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with a specially developed smooth surface applicator tip (mini-brush head), attached to the syringe containing the etching gel. By twisting the syringe top, an ample amount of the acid (Icon- Etch) is dispensed through an opening in the center of the applicator. The flocked tip is used to spread the gel evenly on the enamel surface by moving it in a circular motion, resulting in a homogenous etching pattern, extending 2 mm beyond the edges of the lesion. After 2 minutes of contact time, the etchant was rinsed off with water spray for at least 30 seconds, and dried with oil- and water-free air, resulting in a chalky white appearance of the etched area. To free the enamel microporosities of any remaining water, a 99% ethanol (Icon-Drya) should be applied, using roughly half the syringe content, onto the lesion site surface and left for 30 seconds, after which it is air dried. The resin infiltrate should be applied in two steps: a clean Smooth Surface Tip was attached onto the Icon-Infiltrate syringe, and an ample amount of the Icon-Infiltrate should be applied onto the etched lesion surface and left for 3 minutes to infiltrate and spread into the microporosities and then light-cured (output of 450 nm and a light intensity of 800 mW/cm2 for 40 seconds). A second layer of Icon-Infiltrate should be applied, for an additional minute, and then excess material removed and light-cured again for 40 seconds. Finally, after removing the rubber dam, polishing cups were used to finish the surface to a smooth luster. For further information see [86].

Another prevention method involves improving the effect of different fluoride compounds by applying high-intensity (~100–200 J/cm^2) laser radiation. [87]. According to [88], laser irradiation can significantly alter the permeability, crystallinity and acid solubility of enamel, and lead to increased resistance to demineralization. Furthermore, the absorption of fluoride is improved by laser irradiation [89].

As it was discussed, conservative treatment options for WSLs aim at bypassing the superficial layer and remineralizing the subsurface zone of the lesion. This is accomplished in two distinct ways: with the application of low levels of fluoride and calcium ions, which can penetrate deep into the WSL [90], or by reactivating the superficial enamel substrate via mechanical and chemical abrasion [91].

Considered as a minimally invasive treatment, microabrasion consists of the application of acidic and abrasive compound to the enamel surface and white spot lesion [92]. The microabrasion process abrades the surface enamel while also polishing it, causing it to reflect light differently than natural enamel. The application of a microabrasion technique results in significant regression of WSLs. [93].

One option of microabrasion procedure, preconized experimentally by [93], suggested applying 2-minute 35% phosphoric acid etch applied with Gel-Etch Semi Gel (Temrex Corp, Freeport, NY) and rinse, followed by a 20-second pumice with Topex Prep & Polish Paste (Sultan Dental Products Inc, Englewood, NJ) with a rubber cup, attached to a slow-speed handpiece, in a clockwise direction followed by a rinse. The same microabrasion application can be followed by twice-daily application of 1:1 diluted MI Paste and deionized water for 20 seconds.

The procedures preconized by Akin and Basciftci (2012) [94], recommends the application of 18% of hydrochloric acid mixed with fine pumice powder to obtain a slurry form to be applied

on the affected teeth, by using a rubber cup in a contra angle handpiece. A rubber dam should be used to isolate these teeth from the rest so as to eliminate the chemical effects of microabrasion. The slurry should be agitated into the tooth surface for 30 seconds and then washed off with an air-water spray. The cycle of microabrasion procedure and washing can be repeated three to four times on each affected tooth. The patients underwent four or five sessions of the microabrasion therapy at 2-week interval when necessary.

The association between microabrasion and subsequently, the application of CPP-ACP cream has also been described, and suggested microabrasion to remove the hypermineralized superficial layer of enamel, followed by daily home application of CPP-ACP, could eliminate WSLs without involving invasive restorative procedures [91]. Akin and Basciftci (2012) [94], also concluded that CPP-ACP and fluoride agents support increased remineralization of the demineralized enamel, but the microabrasion is the best method for the cosmetic treatment of these post–orthodontic demineralized white spot lesions.

5. Final remarks

The demineralization occurring in teeth during orthodontic treatment is a significant clinical problem. Thus, in view of the etiological factors related to the development of carious and non-carious lesions, the best preventive strategy seems to be assessing the risk factors prior to banding, coupled with fluoride rinses, regular reinforcement of oral hygiene and dietary advice throughout the course of treatment.

Author details

Andréa Cristina Barbosa da Silva[1*], Diego Romário da Silva[1], Ana Marly Araújo Maia[1], Pierre Andrade Pereira de Oliveira[1], Daniela Correia Cavalcante Souza[2] and Fábio Correia Sampaio[2]

*Address all correspondence to: andreacbsilva@gmail.com

1 Center of Sciences, Technology and Health, State University of Paraiba, UEPB, Paraíba, Brazil

2 Center of Health Sciences, Federal University of Paraiba, Paraíba, Brazil

References

[1] Meeran NA. Iatrogenic possibilities of orthodontic treatment and modalities of prevention. J Orthod Sci 2013;2(3) 73-86.

[2] White LW. Oral hygiene for orthodontic patients. J Clin Orthod 1996;30 340-341.

[3] Rosenbloom RG, Tinanoff N. Salivary *Streptococcus mutans* levels in patients before, during, and after orthodontic treatment. Am J Orthod Dentofacial Orthop 1991;100(1) 35-37.

[4] Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. Am J Orthod Dentofacial Orthop 1982;81(2) 93–98.

[5] Mizrahi, E. Enamel demineralization following orthodontic treatment. Am J Orthod 1982;82(1) 62-67.

[6] O'Reilly MM, Featherstone JD. Demineralization and remineralization around orthodontic appliances: an in vivo study. Am J Orthod Dentofacial Orthop 1987;92(1) 33-40.

[7] Gontijo L, Cruz RA, Brandão PRG. Dental enamel around fixed orthodontic appliances after fluoride varnish application. Braz Dent J 2007;18(1) 49-53.

[8] 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) 353-356.

[9] Sudjalim TR, Woods MG, Manton DJ. Prevention of white spot lesion in orthodontics practice: a contemporary review. Aust Dent J 2006;51(4) 284-289.

[10] Olympio KPK, Bardal PAP, Henriques JFC, Bastos JRM. Prevenção de cáries dentária e doença periodontal em ortodontia: uma necessidade imprescindível. Revista Dental Press Ortop Facial 2006;11(2) 110-119.

[11] Chapman JA, Roberts WE, Eckert GJ, Kula KS, González-Cabezas C. Risk factors for incidence and severity of white spot lesions during treatment with fixed orthodontic appliances. Am J Orthod Dentofacial Orthop 2010;138(2) 188-194.

[12] Ogaard B, Rølla G, Arends J. Orthodontic appliances and enamel demineralization. Part 1. Lesion development. Am J Orthod Dentofacial Orthop 1988;94(1) 68-73.

[13] Enaia M, Bock N, Ruf S. White spot lesions during multibracket appliance treatment: A challenge for clinical excellence. Am J Orthod Dentofacial Orthop 2011;140(1) 17-24.

[14] Øgaard B. Prevalence of white spot lesions in 19-year-old: a study on untreated and orthodontically treated persons 5 years after treatment. Am J Orthod Dentofacial Orthop 1989;96(5) 423-427.

[15] Øgaard B, Larsson E, Henriksson T et al. Effects of combined application of antimicrobial and fluoride varnishes in orthodontic patients. Am J Orthod Dentofacial Orthop 2001;120(1) 28-35.

[16] Du M, Cheng N, Tai B, Jiang H, Li J, Bian Z. Randomized-controlled trial on fluoride varnish application for treatment of white spot lesion after fixed orthodontic treatment. Clin Oral Investig. 2012;16(2) 463-68. doi: 10.1007/s00784-011-0520-4.

[17] Travess H, Roberts-Harry D, Sandy J. Orthodontics. Part 6: Risks in orthodontic treatment. Br Dent J 2004;196(2) 71-77.

[18] Consolaro A, Consolaro MFM-O. Lesões cariosas incipientes e formação de cavidades durante o tratamento ortodôntico. É papel do ortodontista diagnosticar, prevenir e até tratar manchas brancas cariosas e não cariosas no esmalte? Rev Clín Ortodon Dental Press 2006;5(4) 104-111.

[19] Willmot D. White spot lesions after orthodontic treatment. Semin Orthod 2008;14(3) 209-219.

[20] Vital SO, Haignere-Rubinstein C, Lasfargues JJ, Chaussain C. Caries risk and orthodontic treatment. Int Orthod 2010;8(1) 28-45.

[21] Viazis AD, DeLong R, Bevis RR, Rudney JD, Pintado MR. Enamel abrasion from ceramin orthodontic brackets under an artificial oral environment. Am J Orthod 1990;98(2) 103-109.

[22] Chen YJ, Yao CC, Chang HF. Nonsurgical correction of skeletal deep overbite and class II division 2 malocclusion in an adult patient. Am J Orthod Dentofacial Orthop 2004;126(3) 371–378.

[23] Alexande SA, Ripa LW. Effects of self-applied topical fluoride preparations in orthodontic patients. Angle Orthod 2000;70(6) 424-430.

[24] Bagramian RA, Garcia-godoy F, Volpe AR. The global increase in dental caries. A pending public health crisis. Am J Dent 2009;21(1) 1-6.

[25] Caldeira EM, Fidalgo TKS, Passalini P, Marquezan M, Maia LC, Nojima MCG. Effect of fluoride on tooth erosion around orthodontic brackets. Braz Dent J 2012;23(5) 581-585.

[26] Julien KC, Buschang PH, Campbell PM. Prevalence of white spot lesion formation during orthodontic treatment. Angle Orthod 2013;83(4) 641-647.

[27] Fejerskov O, Kidd, E. Cárie dentária: a doença e seu tratamento clínico. 2 ed. São Paulo: Santos, 2011.

[28] Davies SJ, Gray RJ, Qualtrough AJ. Management of tooth surface loss. Br Dent J 2002;192(1) 11-16, 19-23.

[29] Hobkirk JA. Tooth surface loss: causes and effects. Int J Prosthodont 2007;20(4) 340-341.

[30] Black GV. Operative dentistry, Vol. 1 Pathology of the hard tissues of the teeth. London: Claudius Ash, Sons & Co., 1914.

[31] Weatherell JA, Robinson C, Hallsworth AS. The concept of enamel resistance: a critical review. In: Guggenheim B (ed) Cariology today. Basel: Karger, 1984, pp. 223–230.

[32] Bratthall D, Hänsel Petersson G. Cariogram: a multifactorial risk assessment model for a multifactorial disease. Community Dent Oral Epidemiol 2005;33(4) 256-264.

[33] Chaussain C, Opsahl VS, Viallon V, Vermelin L, Haignere C, Sixou M, Lasfargues JJ. Interest in a new test for caries risk in adolescents undergoing orthodontic treatment. Clin Oral Investig 2010;14(2) 177-185.

[34] Meade MJ, Millett DT. An audit of the caries status of patients about to start orthodontic treatment. J Ir Dent Assoc 2011;57(3) 156-160.

[35] Nyvad B, Machiulskiene V, Baelun V. Construct and predictive validity of clinical caries diagnostic criteria assessing lesion activity. J Dent Res 2003;82(2) 117-122.

[36] Nyvad B, Machiulskiene V, Baelum V. Reliability of a new caries diagnostic system differentiating between active and inactive caries lesions. Caries Res 1999;33(4) 252-260.

[37] Thylstrup A, Bruun C, Holmen L. In vivo caries models: mechanisms for caries initiation and arrestment. Adv Dent Res 1994;8(2) 144-157.

[38] Ekstrand KR, Qvist V, Thylstrup A. Light microscope study of the effect of probing in occlusal surfaces. Caries Res 1994;21(4) 368-374.

[39] Mejàre I, Kidd EAM. Radiography for caries diagnosis. In: Fejerskov O, Kidd EAM (Eds) Dental caries: the disease and its clinical management. Oxford: Blackwell Munksgaard, 2008, pp. 69-89.

[40] Fejerskov O, Nyvad B, Kidd EAM. Clinical and histological manifestations of dental caries. In: Fejerskov O, Kidd EAM (eds): Dental caries. The disease and its clinical management. Oxford: Blackwell Munksgaard, 2003, pp. 71–98.

[41] Holst JJ, Lange F. Perimylolysis. A contribution towards the genesis of tooth wasting from non-mechanical causes. Acta Odontol Scand 1939;1(1) 36–47.

[42] Scheutzel, P. Etiology of dental erosion – intrinsic factors. Eur J Oral Sci 1996;104 178-190.

[43] Corega C, Vaida L, Festila DG, Rigoni G, Albanese M, D'Agostino A, Pardo A, Rossetto A, Nocini PF, Bertossi D. Dental white spots associated with bulimia nervosa in orthodontic patients. Minerva Stomatol 2014; Jan 14. [Epub ahead of print].

[44] Kidd EAM et al. Occlusal caries diagnosis: a changing challenge for clinicians and epidemiologists. J Dent 1993;21(6) 323-337.

[45] Hammad SM, Enan ET. In vivo effects of two acidic soft drinks on shear bond strength of metal orthodontic brackets with and without resin infiltration treatment. Angle Orthod 2013;83(4) 648-652.

[46] 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(2) e46-e50.

[47] Bourzgui F, Sebbar M, Hamza M. Orthodontics and caries. In: Naretto S (Ed) Principles in contemporary orthodontics. InTech, 2011. ISBN: 978-953-307-687-4. Available from: http://www.intechopen.com/books/principles-in-contemporary-orthodontics/orthodontics-and-caries.

[48] Brusca M, Chara O, Sterin-Borda L, Rosa AC. Influence of different orthodontic brackets on adherence of microorganisms in vitro. Angle Orthod 2007;77(2) 331-336.

[49] Souza RA, Magnani MBBA, Nouer DF, Silva CO, Klein MI, Sallum EA et al. Periodontal and microbiologic evaluation of 2 methods of archwire ligation: ligature wires and elastomeric rings. Am J Orthod Dentofacial Orthop 2008;134(4) 506-512.

[50] Lidel ID, Elter C, Heuer W, Heidenblut T, Stiesch M, Schwestka- Polly R et al. Comparative analysis of long-term biofilm formation on metal and ceramic brackets. Angle Orthod 2011;81(5) 907-914.

[51] Newbrun E. Açúcar, substitutos do açúcar e agentes adoçantes não calóricos. In: Newbrun E (Ed) Cariologia. São Paulo: Santos, 1988.

[52] Dresti DVW, Waes HV. Prevenção coletiva, semicoletiva e individual em crianças e adolescentes. In: Waes HJMV, Stockli PW (Ed) Odontopediatria. Porto Alegre: Artmed, 2002. cap. 7, pp. 133-150.

[53] Owens J, Addy M, Faulkner J, Lockwood C, Adair R. A short-term clinical study design to investigate the chemical plaque inhibitory properties of mouthrinses when used as adjuncts to toothpastes: applied to chlorhexidine. J Clin Periodontol 1997;24(10) 732–737.

[54] Panzeri H, Lara EHG, Zaniquelli O, Schiavetto F. Avaliação de algumas características das escovas dentais do mercado nacional. Rev ABO Nac 1993;1(1) 23-29.

[55] Halla DA. Propósito das escovas dentárias. Rev Paul Odontol 1982;4(2) 42-47.

[56] Marsh PD. Microbiological aspects of the chemical control of plaque and gingivitis. J Dent Res 1992;71(7) 1431-1438.

[57] Bijella MFTB, Zanella NLM, Tarzia O, Bijella VT. Avaliação do efeito inibidor de bochechos diários com fluoreto de sódio neutro e acidulado a 0, 05 por cento sobre o metabolismo da placa dentária humana (fermentação e síntese de polissacarídeos extracelulares). Cecade News 1994;2(2) 1-19.

[58] Rodrigues LG, Zawadski PT, Calvete E. O efeito do plax na formação da placa bacteriana. Rev Periodontia1999;8(1) 39-44.

[59] Monfrin R, Ribeiro MC. Avaliação in vitro de anti-sépticos bucais sobre a microbiota da saliva. Rev Assoc Paul Cir Dent 2000;54(5) 400-407.

[60] Fine DH et al. Reducing bacteria in dental aerosols: pre-procedural use of an antiseptic mouth rinse. J Am Dent Assoc 1993;124(5) 56–58.

[61] Alves KM, Goursand D, Zenobio EG, Cruz RA. Effectiveness of procedures for the chemical-mechanical control of dental biofilm in orthodontic patients. J Contemp Dent Pract 2010;11(2) 41-48.

[62] Adriaens ML, Dermaut LR, Verbeeck RM. The use of 'Fluor Protector', a fluoride varnish, as a caries prevention method under orthodontic molar bands. Eur J Orthod 1990;12(3) 316-319.

[63] Morrier JJ. White spot lesions and orthodontic treatment. Prevention and treatment. Orthod Fr 2014;85(3) 235-244.

[64] Chow CKW, Wu CD, Evans CA. In vitro properties of orthodontic adhesives with fluoride or amorphous calcium phosphate. Int J Dent 2011; 2011 1-8.

[65] Rao A, Malhotra N. The role of remineralizing agents in dentistry: a review. Compend Contin Educ Dent 2011;32(6) 26-33.

[66] Behnan SM, Arruda AO, González-Cabezas C, Sohn W, Peters MC. In-vitro evaluation of various treatments to prevent demineralization next to orthodontic brackets. Am J Orthod Dentofacial Orthop 2010;138(6) 712.e1-712.e7; discussion 712-3. doi: 0.1016/j.ajodo.2010.05.014.

[67] Dalessandri D, Dalessandri M, Bonettic S, Viscontid L, Paganellie C. Effectiveness of an indirect bonding technique in reducing plaque accumulation around braces, Angle Orthod 2012;82(2) 313–318.

[68] Baka ZM, Basciftci FA, Arslan U. Effects of 2 bracket and ligation types on plaque retention: a quantitative microbiologic analysis with real-time polymerase chain reaction. Am J Orthod Dentofacial Orthop 2013;144(2) 260-267.

[69] Oltramari-Navarro PV, Titarelli JM, Marsicano JA, Henriques JF, Janson G, Lauris JR et al. Effectiveness of 0.50% and 0.75% chlorhexidine dentifrices in orthodontic patients: a double-blind and randomized controlled. Am J Orthod Dentofacial Orthop 2009;136(5) 651-656.

[70] Øgaard B, Alm AA, Larsson E, Adolfsson U. A prospective, randomized clinical study on the effects of an amine fluoride/stannous fluoride toothpaste/mouthrinse on plaque, gingivitis and initial caries lesion development in orthodontic patients. Eur J Orthod 2006;28(1) 8-12.

[71] Jenatschke F, Elsenberger E, Welte HD, Schlagenhauf U. Influence of repeated chlorhexidine varnish applications on mutans streptococci counts and caries increment in patients treated with fixed orthodontic appliances. J Orofac Orthop 2001;62(1) 36-45.

[72] Sari E, Birinci I. Microbiological evaluation of 0.2% chlorhexidine gluconate mouth rinse in orthodontic patients. Angle Orthod 2007;77(5) 881-884.

[73] Gatti A, Camargo LB, Imparato JC, Mendes FM, Raggio DP. Combination effect of fluoride dentifrices and varnish on deciduous enamel demineralization. Braz Oral Res 2011;25(5) 433-438.

[74] Baygin O, Tuzuner T, Ozel MB, Bostanoglu O. Comparison of combined application treatment with one-visit varnish treatments in an orthodontic population. Med Oral Patol Oral Cir Bucal 2013;18(2) 362-370.

[75] Schlueter N, Duran A, Klimek J, Ganss C. Investigation of the effect of various fluoride compounds and preparations thereof on erosive tissue loss in enamel in vitro. Caries Res 2009;43(1) 10-16.

[76] Reynolds EC. Calcium phosphate-based remineralization systems: scientific evidence? Aust Dent J 2008;53(3) 268-278.

[77] Robertson MA, Kau CH, English JD, Lee RP, Powers J, Nguyen JT. MI Paste Plus to prevent demineralization in orthodontic patients: a prospective randomized controlled trial. Am J Orthod Dentofacial Orthop 2011;140(5) 660-668.

[78] Eliades T, Gioka C, Eliades G, Makou M. Enamel surface roughness following debonding using two resin grinding methods. Euro J Orthod 2004;26(3) 333-338.

[79] Bonetti GA, Zanarini M, Incerti Parenti S, Lattuca M, Marchionni S, Gatto MR. Evaluation of enamel surfaces after bracket debonding: an in-vivo study with scanning electron microscopy. Am J Orthod Dentofacial Orthop 2011;140(5) 696-702.

[80] Karan S, Kircelli BH, Tasdelen B. Enamel surface roughness after debonding. Angle Orthod 2010;80(6) 1081-1088.

[81] Huang GJ, Roloff-Chiang B, Mills BE, Shalchi S, Spiekerman C, Korpak AM, Starrett JL, Greenlee GM, Drangsholt RJ, Matunas JC. Effectiveness of MI Paste Plus and PreviDent fluoride varnish for treatment of white spot lesions: a randomized controlled trial. Am J Orthod Dentofacial Orthop 2013;143(1) 31–41.

[82] Bailey DL, Adams GG, Tsao CE, Hyslop A, Escobar K, Manton DJ, Reynolds EC, Morgan MV. Regression of post-orthodontic lesions by a remineralizing cream. J Dent Res 2009;88(12) 1148-1153.

[83] Llena-Puy C. MI Paste Plus and PreviDent fluoride varnish appear no more effective than normal home care for improving the appearance of white spot lesions. J Evid Based Dent Pract 2013;13(3) 114-116.

[84] Kielbassa AM, Muller J, Gernhardt CR. Closing the gap between oral hygiene and minimally invasive dentistry: a review on the resin infiltration technique of incipient (proximal) enamel lesions. Quintessence Int 2009;40(8) 663-681.

[85] Attin R, Stawarczyk B, Kecik D, Knosel M, Wiechmann D, Attin T. Shear bond strength of brackets to demineralize enamel after different pretreatment methods. Angle Orthod 2012;82(1) 56-61.

[86] Shivanna V, Shivakumar B. Novel treatment of white spot lesions: a report of two cases Conserv Dent 2011;14(4) 423-426.

[87] Rios D, Magalhães AC, Machado MAAM, Silva SMB, Lizarelli RFZ, Bagnato VS, Buzalaf MAR. In vitro evaluation of enamel erosion after Nd:YAG laser irradiation and fluoride application. Photomed Laser Surg 2009;27(5) 743-747.

[88] Zezell DM, Boari HGD, Ana PA, Eduardo CP, Powell GL. Nd:YAG laser in caries prevention: a clinical trial. Lasers Surg Med 2009;41(1) 31–35.

[89] Tagomori S, Morioka T. Combined effects of laser and fluoride on acid resistance of human dental enamel. Caries Res 1989;23(4) 225–231.

[90] Bishara S, Ostby A. White spot lesions: formation, prevention, and treatment. Semin Orthod 2008;14(3) 174-182.

[91] Ardu S, Castioni NV, Benbachir N, Krejci I. Minimally invasive treatment of white spot enamel lesions. Quintessence Int 2007;38(8) 633-636.

[92] Murphy TC, Willmot DR, Rodd HD. Management of postorthodontic demineralized white lesions with microabrasion: a quantitative assessment. Am J Orthod Dentofacial Orthop 2007;131(1) 27-33.

[93] Pliska BT, Warner GA, Tantbirojn D, Larson BE. Treatment of white spot lesions with ACP paste and microabrasion. Angle Orthod 2012;82(5).

[94] Akin A, Basciftci FA. Can white spot lesions be treated effectively? Angle Orthod 2012;82(5) 770-775.

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