

Orthodontic Scars : A topical Review

Dr. Sanjeev Kumar Sharma
Senior Resident

Dr. Anushree Chaurasia
Junior Resident

Dr. Pradeep Tandon
Professor & HOD

Department of Orthodontics and Dentofacial Orthopaedics
Faculty of Dental Sciences
KGMU, Lucknow, U.P., India

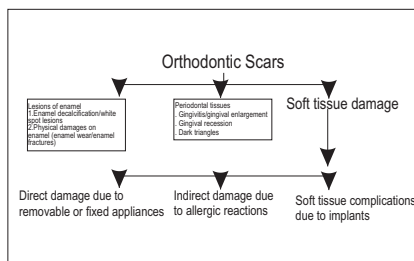
Introduction

Since long the aim of orthodontics has been to achieve functional efficiency, structural balance and esthetic harmony. Orthodontics as a science has flourished from the times of Angle, Tweed, Strang, Andrews to the present modern era. An important esthetic goal in orthodontic treatment planning is to achieve a balanced smile. However the actual benefit of any treatment modality whether in the dental or medical speciality can only be evaluated by also analysing its harmful affects and various methods to overcome it. We orthodontist are lucky enough as the benefits of orthodontic treatment, far outweigh the risk factors or harm caused by orthodontic appliances and materials.

In the medical speciality the term "scar" generally refers to a soft tissue manifestation on the skin or other body tissue in the region of a wound, burn or sore, seen during the healing process. Similarly Orthodontic scars may be defined as a reversible or irreversible, soft or hard tissue damage, that are expressed clinically either intra/extraorally, during or after the orthodontic treatment. The purpose of this article is to provide an overview of various types of orthodontic scars and methods to prevent or treat them.

It should be the prime duty of the operator to carry out a thorough examination of the oral cavity of the patient at the onset of treatment and also during subsequent visits to assess, identify and manage orthodontic scars to avoid the adverse effects and achieve a successful final result. It is also essential to inform the patient about such possibilities prior to commencement of orthodontic treatment.

Orthodontic Scars can be broadly classified into:



Lesions of Enamel

Enamel decalcification/ white spot lesions Fejerskov and Kidd defined WSL as the "first sign of a caries lesion on enamel that can be detected with the naked eye." (Figure 1) The characteristic opaque, chalky, white appearance of WSL is due to difference in refractive index due to loss of mineral from enamel surface or subsurface, this appearance can easily be visualised by drying the tooth surface.² Being one of the most common adverse effects of orthodontic treatment, they can hamper the dental esthetics. Prevalence has been reported to range from 2% to 96%.²⁻⁵

Prevalence prior to orthodontic treatment ranges from 15.5% to 40%^{2,6,7} whereas incidence of new clinically visible WSL occurring during orthodontic treatment ranges between 30% to 70%^{4,5,6,8-10}. Maxillary anterior teeth are most commonly affected, with the order of incidence being lateral incisors, canines, premolars, and central incisors^{2,6,11}; however, all teeth are potentially at risk.

Risk factors include: poor pre-treatment and intra-treatment oral hygiene, age (preadolescent at start of treatment), increased decayed, missing, or filled teeth, etching time and surface area, and caries/restorative status of first molars^{10,11}. One important feature is the potential for rapid formation, with clinically visible lesions developing in as little as 4 weeks.⁵

It has been documented that patients, parents, general dentists, and orthodontists perceive that the responsibility for prevention of WSL lies with the patient.¹² A modified fluoride toothpaste technique involving twice daily brushing for 2 minutes followed by vigorous swishing of the toothpaste slurry for 30 seconds without rinsing with water, and avoidance of eating or drinking for 2 hours, has also been shown to reduce the incidence of new caries in orthodontic patients.¹³ Daily 0.5% sodium fluoride rinse in conjunction with fluoridated dentifrice is perhaps the most common fluoride regimen recommended by orthodontists¹⁴. Other measures include use of fluoride-releasing bonding material, fluoride varnish and amorphous calcium phosphate. For management of

postorthodontic lesions best course is to wait and watch since most lesions tend to improve in their appearance over the first 1 to 2 years after debonding^{15,16}. Other methods to improve the appearance of WSL include resin infiltration, vital bleaching, microabrasion and indirect restorations.

Enamel wear /fracture

At the time of bracket removal, enamel loss can occur and depends largely on the bracket material and method of debond. This is less common with metal brackets where a peeling force is applied to bracket base, leading to bond failure at the bracket-adhesive interface, without damaging the enamel.¹⁹ However a number of reports of enamel fracture and loss at debond of ceramic brackets have been reported^{17,18}. Four debonding techniques including mechanical debonding, electrothermal debonding, laser debonding and ultrasonic debonding have been developed^{20,21}.

Mechanical debonding depends on either the deformation of the bracket or stressing the adhesive to cause adhesive failure. Electro-thermal and laser debonding attempt to dissolve the bonding cement through heat generation. Ultrasonic debonding uses ultrasonic vibrations to break the adhesive interlocking²². Surface enamel cracks and wear provide stagnation areas for the development of caries, cause partial tooth fracture, or may cause unaesthetic discoloration. Higher prevalence of cracks is found in debonded teeth compared to untreated teeth.¹⁹

Restorative procedures can be carried out to manage the tooth fracture. Applying debonding forces lower than 13 MPa and adhering to proper debonding techniques can help avoid the incidence of accidental tooth fracture^{19,22}.

Periodontal Tissues

Gingivitis/Gingival Enlargement

Some amount of gingival inflammation is associated with fixed orthodontic mechanotherapy. (Figure 2) But this is usually transient and does not lead to attachment loss.²³ Hyperplasia of gingiva is commonly found around orthodontic bands leading to formation of pseudo-pockets. However, this usually resolves within weeks of debanding.

The most important factor in the initiation, progression and recurrence of periodontal disease is the presence of microbial plaque.^{24,25} Light orthodontics forces are recommended for adult patients. Utmost care should be taken particularly in patients with systemic diseases, such as epilepsy and patient on calcium channel blockers.²⁶

Gingival Recession

The risk of attachment loss can be anticipated when iatrogenic irritations are present.²⁷ (Figure 3) Patients with good oral hygiene and absence of any periodontal disorders are at minimal periodontal risk when optimum forces are used.²⁸ However, in the presence of poor oral hygiene and preexisting untreated periodontal disorders, fixed orthodontic appliances and tooth movement can contribute to significant and permanent periodontal damage.²⁹ This is more common in Adult patients.^{29,30}

Orthodontic treatment is not contraindicated in this group, provided the disease is well-controlled and the patient maintains excellent oral hygiene throughout the treatment.³⁰

It is important to assess periodontal status prior to treatment and any pre-existing problems must be treated before initiating the treatment. Regular periodontal checks and routine scaling and polishing are highly advisable to prevent the aggravation of periodontal problems.^{30,31}

Dark Triangles

Open gingival embrasures also referred to as black triangles exist when the embrasure space is not completely filled by the gingival tissue. It is mainly associated with low esthetics and serve as area of plaque deposition, thus affecting health of the periodontium. Such a condition is more common in adult patients (1/3rd of patients) with bone loss.³³

This potential complication should be discussed with patients prior to initiating orthodontic treatment.^{32,35} These unesthetic areas can be treated by removing enamel at the contact point so that the teeth can be moved closer to minimize the space between them.

However, care should be taken not to distort the proportional relationships of the teeth to each other in terms of their connector heights.

Soft Tissue

Direct Damage caused by Removable and/or Fixed Appliance Components Removable Appliances: May be used as active appliances during the treatment for the management of minor orthodontic problems which require a simple tipping or in the form of retainers at the end of fixed orthodontic treatment.^{35,36} Damage is mainly caused by the wire components (retentive clasps, springs, canine retractors etc.) which may cause tissue impingement. Undercuts should be carefully evaluated in the plaster model and blocked out prior to acrylisation and care should be taken to avoid any sharp edges and nodules in the appliance to avoid trauma during the insertion and removal of the appliance. Patients should be recalled a

few days after appliance delivery (approx..7 days) to check for any tissue impingement or trauma.³⁶

Fixed Appliance and Its Components Archwire, Brackets, Bands, Transpalatal Arch

Damage to the oral mucosa is common during orthodontic treatment due to rubbing of the lips and cheeks on the archwire, brackets, bands and hooks³⁵ (Figure 4). Although the oral mucosa quickly keratinize and get accustomed to the new appliance relatively fast, this damage can be prevented by:

1. Use of dental wax over the bracket
2. Rubber tubing on the unsupported archwire may serve to reduce the initial trauma and discomfort.
3. The distal ends of the archwire should be cut off flush with the molar tube or cinched toward the tooth to avoid mucosal trauma.³⁶

Loops, Utility Arches

Loops and utility arches are required for space closure, space maintenance or intrusion. Main consideration during their fabrication is vestibular extension. Over extension may cause blanching, tissue impingement, ulcerations, tissue hyperplasia and other types of tissue damage³⁵. In extreme situations, the loop may become completely embedded in the hyperplastic tissue requiring surgical excision for removal of the hyperplastic tissue (Figure 5). This can be prevented by proper fabrication taking into consideration the sulcus depth and periodic monitoring of such components.^{35,36}

Headgear Injury

Samuels and Jones³⁸ classified the types of headgear injuries based of percentage occurrences:

1. Accidental disengagement of head strap while playing (27%)
2. Incorrect handling (27%)
3. Disengagement by another child (19%)
4. Disengagement while asleep (27%)

The headgear bow is sharp and covered by oral bacteria.³⁷ There is a potential risk of a bilateral injury to the eyes because the inner arms of the face-bow are of the same width as the eyes. A penetrating eye injury might not cause immediate pain, but the oral bacteria multiply and the eye can be lost due to overwhelming infection.³⁸ The face bow can also get dislocated during sleep and cause injury to the soft tissues.

Headgear injury can be prevented by:

1. Use of headgear with safety modules that stop it from being accidentally displaced or recoiling back into the face or eyes (Figure 6)
2. Proper patient and parent instructions regarding the safe use of headgear.^{37,38} During the initial days parents should be present to monitor the patient when they wear and remove the headgear. The patient should first remove the head strap before proceeding to remove the face bow, as directly pulling the face bow without loosening the head strap might result in eye injury due to the recoil.
3. Patients should be advised not to wear their headgear while playing.
4. An immediate ophthalmologic

examination, in case an eye injury is suspected.

Indirect Damage by Allergic Reactions Nickel Allergy

Nickel present in orthodontic appliances like brackets, bands, and archwires is responsible for causing allergic reactions in some patients.³⁹ Nickel hypersensitivity affects three in ten of the general population, but the symptoms are very mild and unnoticed. These include loss of taste or metallic taste, numbness, burning sensation, soreness at the side of the tongue, angular cheilitis and erythematous areas or severe gingivitis in the absence of plaque. Nickel-induced contact dermatitis is a Type IV delayed hypersensitivity immune response, occurring 24 hours after exposure.³⁹ It has been found that females are most susceptible to nickel allergy. Patients allergic to nickel can be treated by alternatives like:

1. Use of Titanium wires and brackets⁴⁰.
2. Use of epoxy coated nickel titanium wires.
3. Ceramic brackets or clear aligners can also be used in such patients.

Latex Allergy

Latex sensitivity may occur in response to contact with latex gloves, elastomeric ligatures or intra- and extraoral elastics. The commonest sites affected are the gingivae and tongue, but the perioral region may also be affected.⁴¹

In the latex sensitive patient, steel ligatures or self-ligating brackets may be preferred. Mechanics should be modified to avoid class II and class III elastics.

Allergy to Bonding Agents

Composite and acrylic can cause allergic reactions in some orthodontic patients. Toxicity is due to unpolymerized material (methylmethacrylate) and is greatest immediately after polymerization although cytotoxicity is still evident 2 years after polymerization.⁴² Chemical cured liquid-paste materials are more cytotoxic than light-cured and chemically cured 2-paste materials. No-mix adhesives have been found to be more toxic than two-paste adhesives and must be avoided.⁴³

Soft Tissue Complications related to Implants Impingements and Trauma to Soft Tissue Overlying the Implant

Impingements and trauma to soft tissue overlying the implant is fairly common causing soft tissue damage to the buccal mucosa and attached gingiva related to the implant site.

Peri-implantitis

Peri-implantitis (inflammation of the gingiva around the implant) is as a result of improper oral hygiene maintenance. Patients should be counseled to maintain high level of oral hygiene throughout treatment.

Screw Fracture during Removal Applying lateral forces during implant removal can cause fracture. It is rare if taken out straight..It is important not to wiggle the screwdriver when removing it from the screw head. If the micro-implants are left in place for a very long time, this could also lead to fracture on removal as a result of

partial or full osseointegration.⁴⁴ It is preferable to remove the micro implants as soon as their need is fulfilled rather than waiting for the completion of the entire orthodontic treatment and their removal along with the arch wires and brackets during debonding procedure.

References

1. Fejerskov ONB, Kidd E. Dental caries: the disease and its clinical management, 2nd ed. Copenhagen: Blackwell Munksgaard; 2003.
2. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. Am J Orthod 1982;81:93-8.
3. Mitchell L. Decalcification during orthodontic treatment with fixed appliances—an overview. Br J Orthod 1992;19:199-205.
4. Mizrahi E. Enamel demineralization following orthodontic treatment. Am J Orthod 1982;82:62-7.
5. Ogaard B, Rolla G, Arends J, ten Cate JM. Orthodontic appliances and enamel demineralization. Part 2. Prevention and treatment of lesions. Am J Orthod Dentofacial Orthop 1988;94:123-8.
6. Artun J, Brobakken BO. Prevalence of carious white spots after orthodontic treatment with multibonded appliances. Eur J Orthod 1986;8:229-34.
7. Lovrov S, Hertrich K, Hirschfelder U. Enamel demineralization during fixed orthodontic treatment—incidence and correlation to various oral-hygiene parameters. J Orofac Orthop 2007;68:353-63.
8. Enaia M, Bock N, Ruf S. White-spot lesions during multibracket appliance treatment: a challenge for clinical excellence. Am J Orthod Dentofacial Orthop 2011;14:17-24.
9. Richter AE, Arruda AO, Peters MC, Sohn W. Incidence of caries lesions among patients treated with comprehensive orthodontics. Am J Orthod Dentofacial Orthop 2011;139:657-64.
10. Tufekci E, Dixon JS, Gunsolley JC, Lindauer SJ. Prevalence of white spot lesions during orthodontic treatment with fixed appliances. Angle Orthod 2011;81:206-10.
11. Chapman JA, Roberts WE, Eckert GJ, et al. Risk factors for incidence and severity of white spot lesions during treatment with fixed orthodontic appliances. Am J Orthod Dentofacial Orthop 2010;138:188-94.
12. Maxfield BJ, Hamdan AM, Tufekci E, et al. Development of white spot lesions during orthodontic treatment: perceptions of patients, parents, orthodontists, and general dentists. Am J Orthod Dentofacial Orthop 2012;141:337-44.
13. Al Mulla AH, Kharsa SA, Birkhed D. Modified fluoride toothpaste technique reduces caries in orthodontic patients: a longitudinal, randomized clinical trial. Am J Orthod Dentofacial Orthop 2010;138:285-91.
14. Derks A, Kuijpers-Jagtman AM, Frencken JE, et al. Caries preventive measures used in orthodontic practices: an evidence-based decision? Am J Orthod Dentofacial Orthop 2007;132:165-70.
15. Al-Khateeb S, Forsberg CM, de Josselin de Jong E, Angmar-Mansson B. A longitudinal laser fluorescence study of white spot lesions in orthodontic patients. Am J Orthod Dentofacial Orthop 1998;113:595-602.
16. Mattousch TJ, van der Veen MH, Zentner A. Caries lesions after orthodontic treatment followed by quantitative light-induced fluorescence: a 2-year follow-up. Eur J Orthod 2007;29:294-8.
17. Joseph VP, Rossouw PE. The shear bond strengths of stainless steel orthodontic brackets bonded to teeth with orthodontic composite resin and various fissure sealants. Am J Orthod Dentofacial Orthop 1990;98:66-71.
18. Jeroudi MT. Enamel fracture caused by ceramic brackets. Am J Orthod Dentofacial Orthop 1991;99:97-9.
19. Zachrisson BU, Skogan O, Höymyhr S. Enamel cracks in debonded, debanded, and orthodontically untreated teeth. Am J Orthod. 1980;77:307-19.
20. Artun J. A post-treatment evaluation of multibonded ceramic brackets in orthodontics. Eur J Orthod. 1997;19:219-28.
21. Eslamian L, Borzabadi-Farahani A, Mousavi N, Ghasemi A. A comparative study of shear bond strength between metal and ceramic brackets and artificially aged composite restorations using different surface treatments. Eur J Orthod. 2012;34:610-7.
22. Naimi FB, Gill DS. Tooth fracture associated with debonding a metal orthodontic bracket: A case report. World J Orthod. 2008;9:32-6.
23. Polson AM, Subtelny JD, Meitner SW, Polson AP, Sommers EW, Iker HP, et al. Long-term periodontal status after orthodontic treatment. Am J Orthod. 1988;93:51-8.
24. Loe H, Theilade E, Jensen SB. Experimental gingivitis in man. J Periodontol. 1965;36:177-87.
25. Ericsson I, Thilander B. Orthodontic forces and recurrence of periodontal disease: An experimental study in the dog. Am J Orthod. 1978;74:41-50.
26. Devanna R, Asif K. Interdisciplinary management of a patient with a drug-induced gingival hyperplasia. Contemp Clin Dent 2010 Jul;1:171-76.
27. Alexander SA. Effects of orthodontic attachments on the gingival health of permanent 2nd molars. Am J Orthod Dentofacial Orthop. 1991;199:337-40.
28. Sanders NL. Evidence-based care in orthodontics and periodontics: A review of the literature. J Am Dent Assoc. 1999;130:521-7.
29. Eliasson LA, Hugoson A, Kurol J, Siwe H. The effects of orthodontic treatment on periodontal tissues in patients with reduced periodontal support. Eur J Orthod. 1982;4:1-9.
30. Steffensen B, Storey AT. Orthodontic intrusive forces in the treatment of periodontally compromised incisors: A case report. Int J Perio Rest Dent. 1993;13:433-41.
31. McComb JL. Orthodontic treatment and isolated gingival recession: A review. Br J Orthod. 1994;21:151-9.
32. Ko-Kimura N, Kimura-Hayashi M, Yamaguchi M, Ikeda T, Meguro D, Kanekawa M, et al. Some factors associated with open gingival embrasures following orthodontic treatment. Aust Orthod J. 2003;19:19-24.
33. Tarnow DP, Magner AW, Fletcher P. The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal dental papilla. J Periodontol. 1992;63:995-6.
34. Kurth JR, Kokich VG. Open gingival embrasures after orthodontic treatment in adults: Prevalence and etiology. Am J Orthod Dentofacial Orthop. 2001;120:116-23.
35. Zachrisson BU. Causes and prevention of injuries to teeth and supporting structures during orthodontic treatment. Am J Orthod. 1976;69:285-300.
36. Kerosuo HM, Dahl JE. Adverse patient reactions during orthodontic treatment with fixed appliances. Am J Orthod. 2007;132:789-95.
37. Booth-Mason S, Birnie D. Penetrating eye injury from orthodontic headgear: A case report. Eur J Orthod. 1988;10:111-4.
38. Samuels RH, Jones ML. Orthodontic facebow injuries and safety equipment. Eur J Orthod. 1994;16:385-94.
39. Al-Tawil NG, Marcusson JA, Möller E. Lymphocyte transformation test in patients with nickel sensitivity: An aid to diagnosis. Acta Derm Venereol. 1981;61:511-5.
40. Kim H. Corrosion of Stainless Steel, Niti, coated nickel titanium and titanium orthodontic wires. J Dent Res. 1999;69:39-44.
41. Hain LA, Longman LP, Field EA, Harrison JE. Natural rubber latex allergy: Implications for the orthodontist. J Orthod. 2007;34:6-11.
42. Tang AT, Li J, Ekstrand J, Liu Y. Cytotoxicity tests of in situ polymerized resins: Methodological comparisons and introduction of a tissue culture insert as a testing device. J Biomed Mater Res. 1999;45:214-22.
43. Gioka C, Bouraue C, Hiskia A, Kletsas D, Eliades T, Eliades G. Light-cured or chemically cured orthodontic adhesive resins? A selection based on the degree of cure, monomer leaching, and cytotoxicity. Am J Orthod. 1999;127:413-9.
44. Nienkemper M, Wilmes B, Renger S, Mazaud-Schmelter M, Drescher D. Improvement of mini-implant stability in orthodontics. Orthod Fr. 2012;83:201-7.

