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Review Article

Scars in orthodontics: A critical review of iatrogenic effects and clinical prevention strategies

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Abstract

Orthodontic treatment plays a vital role in improving dental aesthetics, occlusion, and overall oral health. While its benefits are well-documented and widely accepted, it is equally important to acknowledge and understand the potential risks and limitations associated with such treatment—particularly those related to tissue damage. One of the lesser-discussed but clinically significant outcomes of orthodontic therapy is the development of scars or injuries to both soft and hard oral tissues. These iatrogenic effects, although relatively rare, can have lasting consequences on oral health and aesthetics if not identified and managed appropriately.

During orthodontic procedures, various components such as brackets, wires, and bands come into close contact with oral tissues. This prolonged contact, combined with mechanical forces applied to teeth and supporting structures, can sometimes lead to complications. These include soft tissue injuries like mucosal ulcerations, gingival recession, or hypertrophy, and hard tissue damage such as enamel demineralization, decalcification, and root resorption. In some cases, improper appliance placement, excessive orthodontic force, or poor oral hygiene can contribute to more permanent tissue damage, including scarring of gingival or labial tissues.

Moreover, certain patients may be predisposed to developing tissue injuries due to individual anatomical or behavioral factors, such as thin biotype gingiva, parafunctional habits, or inadequate plaque control. Temporomandibular joint (TMJ) discomfort and relapse of tooth positions after treatment are also considered potential long-term risks. Hence, a comprehensive understanding of these possibilities is essential when formulating an orthodontic treatment plan. To minimize these complications, preventive strategies—including careful appliance design, controlled force application, routine oral hygiene reinforcement, and timely intervention—must be integrated into every phase of treatment. Ultimately, the goal is to ensure that the advantages of correcting malocclusion significantly outweigh the risks, thereby safeguarding the integrity of both hard and soft tissues throughout and beyond the duration of orthodontic care.

Keywords: Orthodontic complications, iatrogenic effects, enamel demineralization, root resorption, pulp vitality, gingival recession, TMJ dysfunction.

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1. Introduction

Orthodontic treatment, though helpful, includes possible dangers and restrictions concerning tissue injury. In orthodontics, such risks are thankfully generally rare and minimal now. Nonetheless, weighing all of the possible risks as well as limitations is important. Consideration about proceeding with orthodontic care is a requirement. Risks may damage soft tissues and hard tissues like enamel that demineralizes or lacerates or ulcerates in order to disorder the temporomandibular joint and treatment that possibly fails. To ensure that benefits of malocclusion correction outweigh harm that is potential, people should maintain health preventively of these tissues during treatment and after.[1]

Misalignment, crowding, spacing, and the protrusion of teeth have been persistent aesthetic issues for ages. Orthodontic treatment has thus become an essential component of aesthetic dentistry in the pursuit of a perfect smile. Beyond aesthetics, it significantly aids some patients by improving chewing, speech, appearance, overall dental health, comfort, and self-esteem However, orthodontic devices can sometimes harm the surrounding hard and soft tissues during and after treatment if the orthodontist is not

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careful. It is crucial to identify and manage this potential risk early to avoid such adverse effects.[2]

Damage to the soft and hard tissues, which may be visible both inside and outside the mouth, are referred to as orthodontic scars. These scars may appear during and after treatment and are rarely severe enough to outweigh the benefits of the treatment. However, damage to the hard tissue can be permanent, necessitating further treatment after the completion of orthodontic therapy, as seen in cases of enamel decalcification.[3]

2. Discussion

2.1. Classification of orthodontic scars

Orthodontic scars can be broadly categorized based on the tissues affected

1. Enamel lesions: Demineralization, white spot lesions, abrasions, fractures
2. Dentin lesions: Dentin hypersensitivity, deep demineralization
3. Pulpal changes: Inflammation, ischemia, necrosis
4. Periodontal effects: Gingivitis, gingival enlargement, recession, dark triangles
5. Soft tissue trauma: Ulceration, impingement, allergic reactions
6. Root-related complications: Root resorption
7. TMJ disorders: Joint stress, clicking, or discomfort

2.2. Effects on enamel

1. Enamel etching
2. Intentional enamel reduction (stripping)
3. Enamel colour alteration after the orthodontic treatment
4. Enamel abrasion from brackets of the opposing teeth
5. Debonding
6. Demineralisation

2.3. Effects on enamel

Orthodontic treatment can significantly affect enamel integrity through processes such as etching, enamel reduction, bonding procedures, and bracket removal.

2.3.1. EnamelEtching

Phosphoric acid etching (30–50%) for 15–90 seconds is a conventional method that removes 1.11 to 20 μm of enamel,[2] creating microporosities that enhance resin penetration but can lead to demineralization. Self-etching primers provide a gentler alternative, reducing enamel alteration and residual resin, though they may slightly compromise bond strength. Bharnhat et al. (2021) highlighted the prevalence of aprismatic enamel in cervical areas, contributing to poor etch quality and susceptibility to white spot lesions.

2.3.2. Intentional enamel reduction (stripping)

Interproximal enamel reduction is commonly employed to alleviate crowding or manage tooth-size discrepancies. Techniques range from abrasive strips to rotary diamond burs. While effective, stripping increases enamel surface roughness, potentially enhancing plaque retention and caries risk.[4] Studies suggest that post-stripping polishing using Sof-lex discs or fine burs mitigates this effect. Despite concerns, long-term follow-up studies show no strong link between stripping and interproximal caries.[5] Immediate fluoride application and good oral hygiene are essential for minimizing risk.[6]

2.3.3. Enamel colour alteration

Tooth color changes post-treatment may result from resin infiltration, irreversible tag penetration,[7] or staining from composite degradation. While subjective shade matching is common, instrumental methods offer greater reliability. Studies report that residual resin and adhesive discoloration can alter L* values and esthetics.[8] These changes are influenced by the chemical composition of adhesives and their interaction with dietary chromogens.

2.3.4. Enamel abrasion from brackets

Contact between opposing teeth and brackets, particularly ceramic ones, may lead to enamel abrasion. Areas prone to this include upper canines and anterior incisors in deep bite or crossbite cases. Ceramic brackets, composed of aluminum oxide, exhibit high hardness and abrasiveness. Preventive strategies include occlusal adjustment, bite planes, and cautious bracket placement.[9]

2.3.5. Debonding and enamel damage

Ceramic brackets present higher risk of enamel fracture due to their bond strength. Safe debonding involves peeling forces at the bracket-adhesive interface, often leaving some residual resin for controlled removal. Mechanical, electrothermal, laser, and ultrasonic techniques have been developed, each with advantages and limitations.[10] While diamond burs are effective, they tend to leave rough surfaces prone to plaque retention.[11]

2.3.5.1. Enamel fractures during debonding

Metal brackets typically debond with minimal enamel damage. Ceramic brackets, however, may cause enamel fractures if not carefully handled.[12] Zachrisson reported higher enamel crack prevalence with chemically bonded ceramic brackets. Forces below 13 MPa and proper debonding techniques reduce the risk.[13] A good debonding technique is to squeeze the bracket at the base so that the bracket comes off leaving some residual composite at the enamel surface, which can be cleaned up later with a carbide bur.[14]

2.3.5.2. Evaluation and Management of Enamel post-debonding

Various indices—such as the Enamel Surface Index,[15] Enamel Damage Index,[16] and Surface Roughness Index[17] help assess enamel condition post-cleanup. Studies using profilometry and microscopy confirm that tungsten carbide burs create less roughness than diamond burs or lasers. Ultrasonic and slow-speed rotary methods are preferred for minimal enamel loss. Preventive strategies include immediate fluoride application and dietary counselling

2.4. Management of enamel cracks/fractures[18]

1. Resin-modified glass cement provides minimal damage to the enamel surface.
2. Debonding at the bracket-resin interface with Peeling off stroke is the supported method and removal of residual bonding agents to minimize the risk of enamel fracture and it leaves the enamel surface intact.
3. Debonding force lower than 13Mpa is advocated for debonding technique.
4. Appropriate dietary advice should be given to minimize tooth substance loss

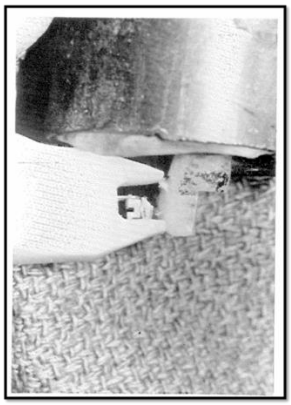

Figure 1: Orthodontic bracket debonding with plier

2.5. Effects on dentin

2.5.1. White spot lesions (WSLs)

WSLs are subsurface enamel demineralizations caused by prolonged plaque retention around orthodontic appliances. The irregular bracket surfaces impede self-cleansing, promoting acidogenic bacterial growth. WSLs can extend into dentin if left untreated.

Diagnosis involves drying and examining lesions for surface texture, carious WSLs are rough and porous; non-carious are smooth and shiny. Differential diagnosis includes fluorosis, enamel hypoplasia, and developmental defects.[19]

Prevalence varies from 2% to 96%. Studies show WSLs increase during treatment, especially in the gingival areas of maxillary lateral incisors. Risk factors include poor oral hygiene, high-sugar diets, lack of fluoride, and previous caries.[20]

The structure of meals and snacks influences the quantity and frequency of exposure to fermentable carbohydrates and to caries risk. An evaluation of sugared beverage intake is a key item that should be included in a patient's dietary assessment. Although orthodontists do not typically assess patients' dietary habits because of resource and time limitations, such evaluations are essential in preventing white spot lesions.[21]

2.5.1.1.. Prevention & management:[22]

1. Reinforce brushing with fluoride toothpaste.
2. Provide diet counseling to reduce fermentable carbs.
3. Use fluoride rinses/varnish, xylitol gum, and professional cleanings.
4. Manage existing lesions with remineralization, microabrasion, or resin infiltration.

2.5.2. Dentin hypersensitivity

Dentin hypersensitivity may result from enamel loss or gingival recession exposing tubules. Stimuli (thermal, tactile) cause fluid movement in dentinal tubules, triggering nerve pain (hydrodynamic theory).[23]

Though data is limited, orthodontic movement near the buccal bone plate may increase susceptibility. Diagnosis is clinical, confirmed by exclusion and patient response to stimuli.

Management includes careful treatment planning, monitoring symptoms, and addressing patient discomfort. Improved diagnostic protocols and more research are needed.

2.6. Effects on pulp

Orthodontic forces can impact pulp vitality, particularly in teeth with pre-existing trauma or restorations. Short-term application of force may reduce pulpal respiration and blood flow; excessive or prolonged force can result in circulatory stasis, inflammation, and, in rare cases, pulp necrosis.[24] Careful force management and regular monitoring are essential when treating pulpal-compromised teeth.

2.7. Biological response to orthodontic forces

Orthodontic tooth movement initiates an acute inflammatory response in the PDL, followed by chronic remodeling. Cytokines, growth factors, and immune cells mediate tissue responses. Pulpal changes may include reduced metabolic activity, odontoblast apoptosis, vacuolization, and tertiary dentin formation. These effects depend on the magnitude, direction, and duration of force, as well as patient factors such as age and trauma history.[25]

2.8. Pulp blood flow (PBF) and vitality monitoring

Transient reductions in PBF occur with orthodontic treatment but typically return to baseline within weeks. Laser Doppler flowmetry (LDF) is a reliable, non-invasive tool for monitoring pulp vitality. Studies have shown PBF decreases

shortly after force application, followed by a rebound post-appliance removal, especially in anterior teeth.[26]

Table 1: Clinical studies summary

	Key Findings
Mousa et al. (2023)[13]	Piezo-orthodontics and conventional retraction had similar effects on pulpal volume; no association with root resorption.
Chen et al. (2008)[14]	PBF temporarily increased post-appliance removal, particularly in adolescents; unrelated to root resorption.
Pomt et al. (2010)[15]	Higher CGRP expression in response to increased orthodontic force indicates pulpal sensitivity.
Binet et al. (2011)[16]	Mild intrusive forces did not result in significant histological pulpal changes.
Howell et al. (1990)[17]	Increased incidence of caries and endodontic treatment post-orthodontics, though not significantly different between fixed and aligner systems.

2.9. Histological and vascular effects

Orthodontic forces stimulate inflammatory mediators (e.g., CGRP) and may cause temporary ischemia. Pulpal volume reduction is often due to tertiary dentin deposition—a defense response to mechanical stress. These changes are generally reversible and do not lead to long-term pulp damage when forces are controlled.[27]

2.10. Clinical considerations for traumatized teeth

When orthodontic treatment involves previously traumatized teeth, specific protocols must be followed. Mild, intermittent forces are recommended, and radiographic monitoring is advised based on injury type.(**Table 2**)

Table 2: Injury types and notes

Injury Type	Orthodontic Delay	Notes
Concussion/ Subluxation	3–5 months	Monitor pulp for 1 year
Extrusion/ Luxation	≥ 6 months	Splint, monitor for 5 years
Intrusion	≥ 6 months post-repositioning	High risk of pulp necrosis
Avulsion	≥ 1 year	Replantation followed by delayed movement

2.11. Effects on periodontal tissues

2.11.1. Gingivitis / Gingival enlargement

Orthodontic appliances increase plaque retention, leading to transient gingival inflammation. In the presence of poor oral hygiene, bands may cause more gingival hyperplasia and pseudo-pockets than bonded brackets.[28] Inflammation typically resolves post-treatment with proper hygiene. Lopez Arrie (2024)[29] identified salivary protein changes as potential biomarkers of gingival enlargement.

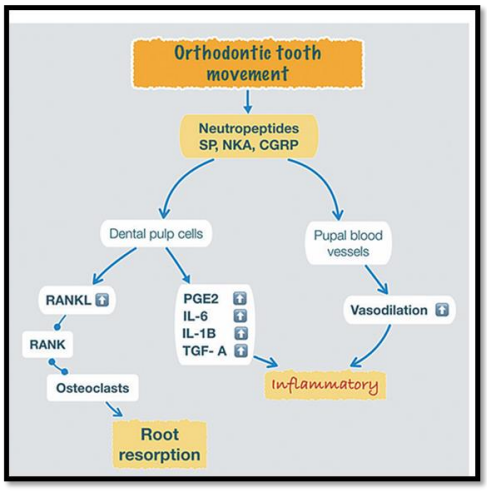

Figure 2: Pulpal inflammatory process to orthodontic force

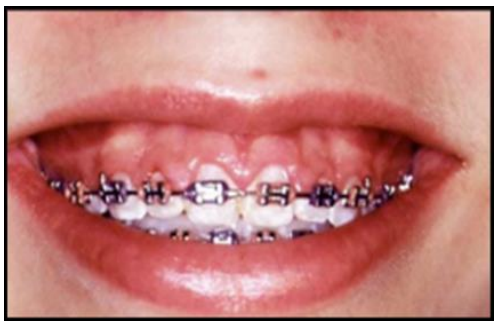

Figure 3: Gingival enlargment

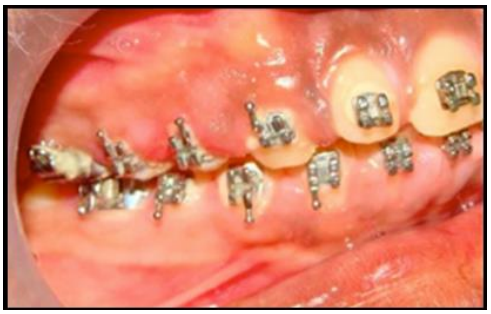

Figure 4: Right lateral view showing inflammatory enlargment.

2.11.2. Gingival recession

Gingival recession is the apical migration of gingival tissue, commonly seen with labial tooth movement or thin biotypes.[30] Miller's classification (Class I–IV) guides prognosis and root coverage potential. While studies differ, buccal movement and tipping increase recession risk, particularly in anterior teeth. Good plaque control and avoiding excessive forces help reduce this risk.[31]

2.11.3. Black triangles (Open Gingival Embrasures)

These occur due to papilla loss between teeth, often after space closure or root divergence. Proper root alignment and

maintenance of interproximal bone help minimize their appearance.

2.12. Alveolar bone changes

Orthodontic forces can alter alveolar bone density. Some studies report temporary reductions post-treatment, while others show minimal changes. CBCT is useful for tracking these changes due to its 3D accuracy and low radiation exposure. Bone recovery during retention is still under investigation.

2.13. Soft tissue injuries from orthodontic appliances

Orthodontic appliances, particularly headgear, may pose a risk of soft tissue injuries such as facial lacerations, mucosal ulcerations, and rare but serious eye trauma. Headgear injuries have been linked to accidental disengagement during play or sleep, incorrect handling, or interference by others. Fuh LJ et al.[33] reported 27% of such injuries due to accidental disengagement during play and sleep, and 19% due to interference by another child. Midfacial injuries were most common, and while eye injuries are rare, they remain a serious concern.

To minimize these risks, the use of safety features such as snap-release straps, rigid neck straps, and safety bows is strongly recommended. Orthodontists should provide patients with both verbal and written safety instructions and discourage wearing headgear during active play. Additionally, dental wax and light-cured materials can be applied to sharp appliance components to prevent mucosal injury. In severe cases, temporary removal of the appliance may be necessary until healing occurs. Mouthguards are advised for patients engaged in contact sports or those playing wind instruments.[33]

2.14. Temporomandibular joint (TMJ) and orthodontic treatment

The link between orthodontic treatment and temporomandibular disorders (TMD) remains a topic of debate. Early studies, such as that by Franks, suggested a possible correlation; however, the author himself acknowledged the variability in treatment types made formal conclusions unreliable. While some believe orthodontics may contribute to TMD, others propose it could alleviate symptoms by correcting occlusion. A review of literature showed no definitive evidence establishing orthodontics as a risk factor for TMD, although a few weak associations were found between TMD and certain occlusal discrepancies like RCP–ICP shifts.[34]

Studies comparing extraction versus non-extraction therapies yielded inconclusive results. Some found non-extraction treatments associated with fewer TMD symptoms, while others found no difference. Additionally, research from Sweden comparing outcomes of treatment by orthodontists versus general dentists found no significant variation in TMD prevalence post-treatment. TMD symptoms may temporarily decline during active orthodontic therapy, but tend to return to pre-treatment levels post-treatment.[35]

Overall, the onset of TMD during or after orthodontic treatment may often be coincidental, especially since TMD signs naturally increase during adolescence—the typical age for orthodontic intervention. Clinicians are advised to monitor occlusion for interferences and adjust forces when TMD symptoms arise during treatment. Ultimately, risks associated with orthodontics depend on both treatment-related factors and individual patient variability. A conceptual framework suggests that adverse outcomes occur when treatment challenges exceed a patient's adaptive capacity. Clear communication of realistic treatment goals is essential to mitigate patient dissatisfaction and medicolegal risks.[36]

2.15. Risks of orthodontic treatment As. A conceptual framework

As the foregoing discussion demonstrates, orthodontic therapy inevitably produces a biological challenge to the stomatognathic system. The outcome of this challenge is dependent upon both the nature of the treatment that is performed and patient-related factors. Whilst some aspects of patient susceptibility to the risks are essentially fixed (e.g. genetics), others are modifiable (e.g. oral hygiene).

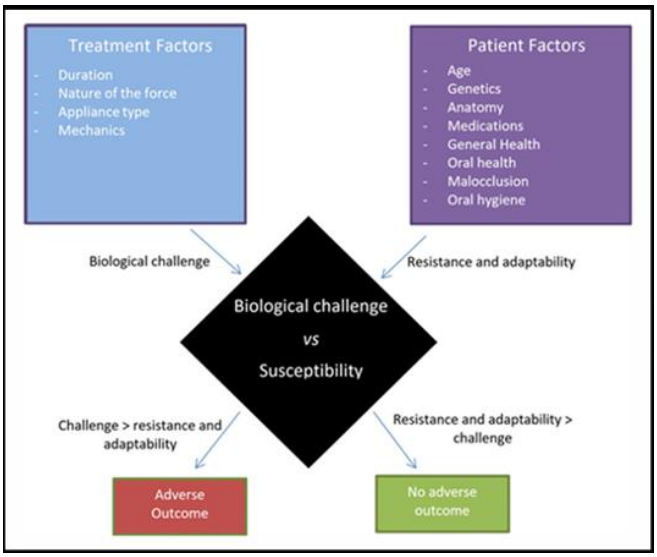

Figure 5: Conceptual frame work to explain the risks of orthodontic therapy

Conceptual framework to illustrate, in general terms, how the risk of adverse outcomes in orthodontic therapy materializes through a synergy between the treatment and the patient. In this framework, an adverse outcome will be the result of the treatment challenge exceeding the patient's resistance and adaptability in some respect Although this framework has natural limitations, it shaped that it will help clinicians better appreciate the importance of having a sound understanding of the orthodontic appliances they use as well

as those patient characteristics that can impact upon treatment.

Finally, clinicians must also carefully manage patients' expectations as part of their overall risk management strategy. From a medicolegal perspective, a very real risk of orthodontic treatment is patient disappointment with an intended or accidental treatment outcome. Treatment goals should represent an agreement between the patient and the clinician, and clinicians must therefore be honest with themselves and patients about whether treatment objectives are realistic.[37]

3. Conclusion

It has been shown that the risks of orthodontic treatment vary between individuals and treatment plans. Clinicians should develop treatment plans in light of an assessment of their patients' susceptibility to these risks and patients should be duly informed of these risks as part of informed consent. Doing so inevitably requires a degree of experience and skill on the part of the clinician. In light of this, a one-size-fits-all treatment philosophy is liable to expose patients to a higher risk of adverse outcomes.

Orthodontic treatment is similar to any other treatment that may be related with unwanted out comes. Recognition of these side effects is critical to the orthodontist and the patient. It is essential to obtain thorough medical, dental and family history before starting treatment. Progress diagnostic records during the treatment such as radiographs and monitoring of the periodontal status is of great importance for success of therapy. Clear communication with the patient regarding the risks and benefits of the planned orthodontic treatment is important to avoid any future misinterpretations.

Clinicians must obtain a signed consent for treatment and the risks involved. The etiology of iatrogenic effects of orthodontic treatment is multifactorial. Patient's genetics, oral hygiene, type of orthodontic treatment and treatment duration are some of the most common causes

4. Conflict of Interest

None.

5. Source of Funding

None.

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