


Gingival Recession Following Orthodontic Retention: Periodontal Improvement Through Orthodontic and Periodontal Treatment. Report of Two Cases of “Wire Syndrome”

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Citation: Michea MA, et al. Gingival Recession Following Orthodontic Retention: Periodontal Improvement Through Orthodontic and Periodontal Treatment. Report of Two Cases of “Wire Syndrome”. J Dental Health Oral Res. 2026;7(1):1-10.

<https://doi.org/10.46889/JDHOR.2026.7129>

Received Date: 19-03-2026

Accepted Date: 08-04-2026

Published Date: 15-04-2026



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Abstract

Wire syndrome is an under-recognised complication associated with post-orthodontic fixed retainers, characterised by unplanned dental movements and gingival recession. This article reports two cases of wire syndrome with gingival recession, highlighting a management strategy that combines compensatory orthodontics and periodontal plastic surgery. The discussion emphasizes the importance of recognizing the risk factors that predispose to Wire Syndrome, its early detection to prevent it and the most appropriate treatment.

Keywords: Tooth Migration; Gingival Recession; Orthodontic Retainers; Orthodontic Wires

Introduction

Orthodontic Treatment (OT) is a widely used procedure aimed at correcting malocclusions, enhancing dental aesthetics and ensuring the proper functioning of mastication, swallowing, breathing and speech. The impact of OT on the periodontium has been a subject of ongoing research, as orthodontic forces induce tooth movement through bone remodelling at pressure and tension sites, which also affect the gingival tissues [1]. OT may be linked to side effects such as Gingival Recession (GR), defined as the apical migration of the gingival margin from the cemento-enamel junction. This condition can result from direct causes or predisposing factors, with post-OT GR prevalence reported between 65% and 86.7% [2]. The aetiology of GR is multifactorial and failure of post-treatment fixed Orthodontic Retention (OR) has been associated with the development of GR, a phenomenon known as ‘Wire Syndrome’ (WS) [3,4].

Wire Syndrome

WS refers to unplanned and unexpected dental movements that are unrelated to pre-treatment dental positions or post-treatment relapse. These movements occur in teeth bonded to an OR and can lead to periodontal sequelae such as GR [5]. The reported prevalence of WS ranges from 1.1% to 43% [4]. OR is used to maintain teeth in their final post-treatment position, without

requiring active movement, thereby ensuring long-term occlusal stability and preventing relapse. Fixed OR systems are less dependent on patient compliance and typically employ adhesive bonding techniques. The retention wire must be passively bonded, constructed from biocompatible materials such as cylindrical or flat stainless steel with high elasticity and low friction [6,7]. While some wires, such as Twistflex, permit micromovements within the alveolus, significant displacement in the presence of OR can result in both aesthetic and functional periodontal consequences, including GR. The onset of GR may occur between 4 and 15 years after completing OT [4,8].

Evidence categorises the risk factors for WS into three groups: professional-related, wire-related and patient-related (Table 1). Among orthodontic interventions, dental proclination has been associated with higher prevalence and severity of GR, particularly in the mandibular anterior region [9]. However, the evidence is inconclusive regarding incisor proclination as a definitive risk factor for GR with only moderate associations found between GR and OT [10-12]. Other identified factors include age, as older patients face a 50% higher risk of post-OT GR [2]. The average time to GR onset due to WS is 4 ± 2.8 years, with a higher prevalence in females and the mandibular region [4].

Orthodontic movements associated with WS include the “X effect” (torque difference between adjacent incisors), the “twist effect” (opposite inclination between contralateral canines), excessive crown and/or root torque (heightened torque changes in incisors or canines), as well as non-specific complications such as diastema opening and discrepancies in incisal height. Combinations of these movements may also occur [4]. A displacement of just 0.2 mm in the retainer wire can generate forces up to 1 Newton, which are enough to cause undesirable movements during the retention period [14].

Most WS cases have been reported with twisted, round stainless-steel wires; however, cases have also been described involving flat, braided wires, single-strand, round wires bonded only to canines and round, coaxial stainless-steel wires [8].

Beyond dental misalignment, WS-related sequelae include periodontal complications such as vestibular and/or lingual clinical attachment loss, GR and bony dehiscences. These may compromise pulpal vitality and are more severe with mandibular fixed retention [5]. Alterations in gingival crevicular fluid composition have also been described [6].

This article aims to report two cases of WS, describe related risk factors and emphasise prevention and treatment strategies.

Professional-Related	Bonding failures: 1. Iatrogenic deformation of the wire 2. Inadequate passivity. 3. Adhesive properties at the adhesive/wire interface under external force 4. Undetected debonded wire
	Orthodontic interventions: dental proclination.
Wire-Related	Multi-strand round wire
	Stainless steel material
	Mechanical property alterations: 1. Fatigue 2. Activation 3. Unwinding of wire strands
	Deformations due to: 1. Masticatory force 2. Hard food
	Wire fracture (remaining attached to one or more teeth).

Patient-Related	Thin periodontal phenotype (gingival and osseous)
	History of gingival recession
	Reduced width and thickness of attached gingiva
	Aberrant frenula
	Age-related physiological changes
	Tooth position before orthodontic treatment
	Deep labiomentental fold
	Traumatic use of dental floss around the retainer
	Poor cooperation: poor oral hygiene, harmful habits or parafunctions (e.g., onychophagia)

Table 1: Risk factors associated with wire syndrome (Adapted from 4,8,14).

Case Reports

Patient 1

A 29-year-old female with no systemic medical history. She began OT at age 13 in 2018, which lasted 1 year and 10 months. Treatment ended with a flat, braided, fixed retainer. In 2023, she reported GR on tooth 4.1, first noticed one month after retainer placement, progressing monthly and associated with mild thermal sensitivity (Fig. 1). Orthodontic examination revealed a skeletal Class II dolichofacial pattern with bilateral molar and left canine neutroclusion, normal step and overjet, retroclined upper and proclined lower incisors. Thin gingival phenotype and RT2 Cairo recession of 4.5 mm length and 2.5 mm width, with complete absence of attached gingiva on tooth 4.1 [15]. The fixed lower retainer was compromised, debonded at 4.1 and showed wire deformation. Pulp vitality tests for 4.1 were normal. CBCT showed a thin symphysis with limited vestibular root coverage (Fig. 2). WS diagnosis was established. Due to stable occlusion, treatment consisted of orthodontic repositioning using the torque cantilever proposed by Laursen and Melsen (Fig. 3) to improve the root inclination of 4.1 degrees, followed by periodontal plastic surgery [16]. Tooth position was corrected within 6 months, showing gains in vestibular bone plate and soft tissue (Fig. 4,5). Passive retainer bonding was performed using a Bond-A-Braid (Reliance Orthodontics) wire, with instructions to monitor any dental changes. Three months later, root coverage surgery was performed on the remaining GR (3.5 mm in length, 1 mm in width apically). A laterally displaced partial-thickness flap with a connective tissue graft from the palate was performed (Fig. 6). Mild post-operative pain was managed with analgesics. Follow-up at weeks 1, 3, 4, 8 and 10 showed favourable outcomes, with the patient satisfied both functionally and aesthetically. No adverse events occurred and informed consent was obtained.

Patient 2

A 27-year-old male with no systemic medical history. He began OT in 2015 at age 14, which lasted 2 years and 6 months and ended with a “hygienic” type retainer (Fig. 7), consisting of a stainless-steel wire contoured to the lingual surfaces of the incisors. GR was noticed by the patient six years after retainer placement, with mild thermal sensitivity and significant aesthetic concerns. Diagnosis revealed a skeletal Class II mesofacial pattern with minor rotations, bilateral molar and left canine neutroclusion, a normal step and overjet and severe linguoversion of both upper and lower incisors. The lower retainer was in poor condition, debonded at tooth 3.1, which presented an RT2 Cairo recession of 9 mm in length and 3 mm in width, with complete loss of attached gingiva (Fig. 8). Pulp testing was normal. CBCT showed total vestibular dehiscence of the root (Fig. 9). WS diagnosis was established. Due to stable occlusion, the same treatment approach was used as in patient 1: root torque correction using Laursen and Melsen’s cantilever (Fig. 10), followed by periodontal plastic surgery [16]. Malposition was corrected within 3 months, resulting in a reduced GR of 7 mm in length and 2 mm in width (Fig. 10,11). Root coverage surgery was then performed with a laterally displaced partial-thickness flap and palatal connective tissue graft (Fig. 12). Analgesics were prescribed for mild post-operative discomfort. Follow-ups at weeks 1, 3, 4, 8 and 10 confirmed successful treatment outcomes, with patient satisfaction and no adverse effects.



Figure 1: Gingival recession on tooth 4.1, Cairo RT2 classification, 4.5 mm length × 2.5 mm width.



Figure 2: CBCT showing a thin symphysis.



Figure 3: Torque cantilever [15].

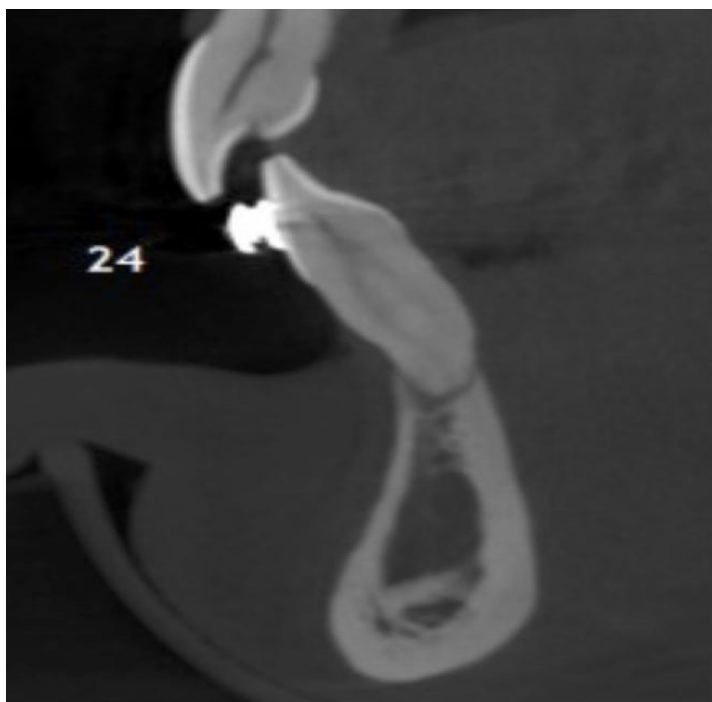


Figure 4: Post-orthodontic CBCT control.



Figure 5: Post-treatment occlusion. GR reduced to 3.5 mm length \times 1 mm apical width (previously 2.5 mm).



Figure 6: Mucogingival surgery on tooth 4.1. Ten-month follow-up.



Figure 7: "Hygienic" type fixed retainer.



Figure 8: Gingival recession on tooth 3.1, Cairo RT2, 9 mm length \times 3 mm width.

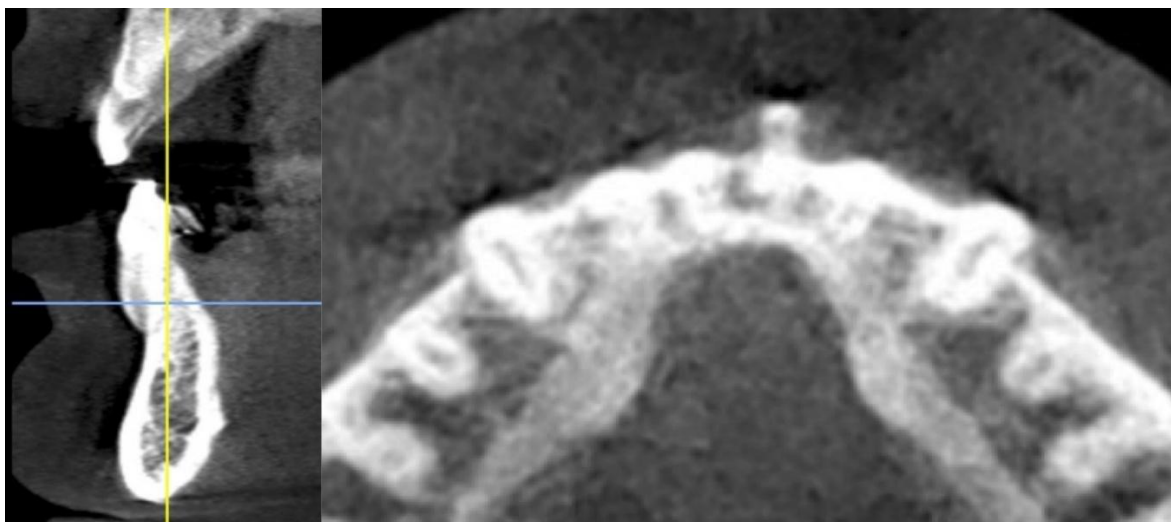


Figure 9: Sagittal and axial CBCT slices before treatment.



Figure 10: Torque cantilever (Laursen and Melsen, 2020). GR reduced to 7 mm length \times 2 mm width (previously 9 mm \times 3 mm).

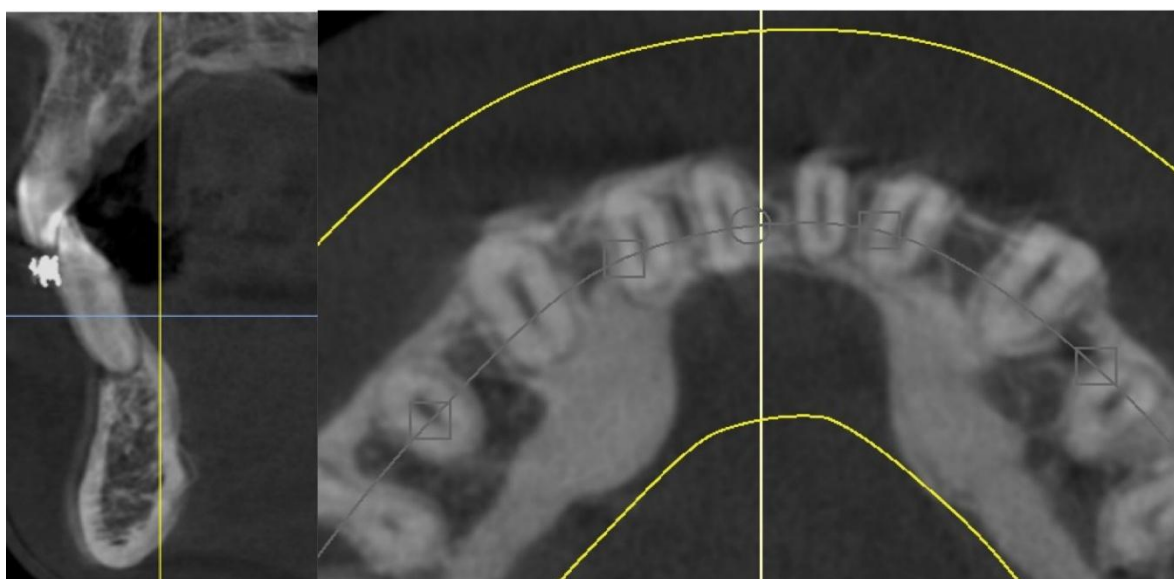


Figure 11: CBCT post-root torque correction.



Figure 12: Mucogingival surgery on tooth 3.1. Ten-month follow-up.

Discussion

WS has recently emerged as a significant concern in orthodontics and periodontics due to its complex aetiology and potential impact on patients. Current evidence identifies risk factors, diagnostic criteria and strategies for treatment and prevention. WS is not considered a relapse phenomenon linked to pre-treatment or final post-treatment tooth positions. It does not affect all patients and understanding remains limited because of the lack of controlled clinical trials. The available literature is diverse; not all studies account for plaque control or patients' periodontal phenotypes before OT [4].

Regarding GR risk factors, multiple causes can act independently or together. In terms of periodontal considerations, in addition to reduced width and thickness of attached gingiva and a thin gingival phenotype, aberrant frenula insertions are also implicated [2,17]. Some risk factors, such as onychophagia, poor oral hygiene adherence or traumatic brushing techniques, can be challenging to control [12,18]. Whether OT alone causes GR remains a matter of debate [3]. The most widely accepted mechanism suggests that dental root displacement towards the alveolar cortex increases the risk of bony dehiscence, especially in gingival phenotypes thinner than 1 mm, where the gingiva migrates apically, leading to GR [1]. Therefore, in WS cases involving unplanned movement of a tooth bonded to a retainer, GR may develop due to the increased risk of dehiscence.

The time between OT and GR onset may offer insights into WS aetiology and the interaction of professional-, wire- and patient-related risk factors (Table 1). For example, early GR following OT may indicate a lack of passive bonding, while delayed GR years after OT could point to wire-related factors [4]. One case series concluded that retainer wire characteristics significantly influence WS: flexible braided retainers can cause unintentional tooth movement even 15 years after treatment [19]. Another study showed that flat braided retainers distribute torsional forces more evenly than multistrand wires [20]. Conversely, some authors argue that wire properties may not trigger WS and that failure rates depend more on bonding protocols than on retainer type [21]. If professional-related factors outweigh wire- or patient-related factors, WS may be better characterised as an iatrogenic result of post-orthodontic retention rather than a true "syndrome," especially given the lack of a single known cause or well-defined pathophysiology [4]. Since professional-related factors are modifiable, clinicians must ensure proper bonding techniques and wire passivity. Routine check-ups are advised for at least six months post-retainer placement aligning with evidence indicating that retainer failure is more common within the first two years after OT [19,22].

WS remains largely unfamiliar to many general dentists [8]. Given its potential to cause root inclination shifts, bony dehiscences and GR, recognising WS and its consequences is essential. As retention periods extend, various dental professionals may detect and manage early signs of WS. Routine periodontal maintenance visits offer an opportunity to identify retainer debonding and WS indicators, enabling early intervention to prevent further progression. WS has been associated with fenestrations, bony dehiscences and hypersensitivity [4]. Early treatment can help prevent severe complications and preserve long-term dental and periodontal health. Despite these risks, fixed retention remains a reliable strategy for stabilising post-OT mandibular and maxillary arches.

Therapeutic Approach

WS treatment varies depending on its severity [4]. In mild cases, removing the retainer may allow the migrated tooth to reposition itself spontaneously, followed by proper rebonding. Moderate to severe cases might require re-treatment with orthodontics, combined with endodontic and periodontal surgery based on clinical needs. In the cases presented, endodontic treatment was not necessary, but orthodontic and periodontal surgical procedures were successfully carried out, resulting in favourable outcomes and no adverse events reported at 10-week follow-up. In WS cases involving orthodontic re-treatment and root coverage procedures, satisfactory functional and aesthetic results can be achieved [16]. Consistent with Laursen's report, root coverage was selected post-OT for mandibular teeth with 3.5 mm and 7 mm recessions, which are the most common locations for WS-related GR [4].

Orthodontically, the torque cantilever described by Melsen proved effective because of its statically determined system design, enabling precise root repositioning and GR reduction [15]. This correction facilitated subsequent periodontal plastic surgery using a laterally displaced partial-thickness flap and submerged connective tissue graft from the palate, enhancing long-term predictability.

Conclusion

WS is a complex phenomenon with multiple associated risk factors. Despite the limited quantity and quality of existing evidence, further investigation is needed to elucidate its aetiology and establish standardised prevention and treatment protocols. Early detection and prompt intervention are essential for preventing serious complications and preserving dental and periodontal health. Future research should include prospective studies and controlled trials to adjust for all relevant variables-patient, wire and clinician-related-and to identify the most effective treatment strategies.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Funding Statement

This research did not receive any specific grant from funding agencies in the public, commercial or non-profit sectors.

Acknowledgement

None.

Data Availability Statement

Not applicable.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore, was exempt.

Informed Consent Statement

Informed consent was taken for this study.

Authors' Contributions

All authors contributed equally to this paper.

<https://doi.org/10.46889/JDHOR.2026.7129>

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