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Original Article

External Root Resorption in Periodontally Compromised Orthodontic Patients: Biological Risk Factors and Prevention

Anna E. Keller[1], Mohamed A. Hassan[1]*, Nur S. Ismail[1]

[1] Department of Periodontics and Orthodontics, Faculty of Medicine, University of Zurich, Zurich, Switzerland.

***E-mail** ✉ mohamed.hassan@outlook.com

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ABSTRACT

External root resorption (ERR) is a common iatrogenic complication of orthodontic treatment, characterized by the irreversible loss of root structure due to odontoclastic activity. In periodontally compromised patients, who often present with reduced alveolar bone support and chronic inflammation, the risk of ERR is significantly heightened, potentially leading to tooth mobility, loss of attachment, and compromised treatment outcomes. This narrative review synthesizes recent literature from 2020 to 2025 to elucidate the biological risk factors contributing to ERR in this vulnerable population and to explore preventive strategies. Key biological risk factors include excessive mechanical forces during tooth movement, which upregulate inflammatory mediators such as receptor activator of nuclear factor kappa-B ligand (RANKL), genetic predispositions, prior dental trauma, abnormal root morphology, and systemic conditions like endocrine disorders. Periodontal compromise exacerbates these risks through microbial dysbiosis, increased cytokine expression (e.g., IL-1β, IL-6, TNF-α), and altered periodontal ligament (PDL) stress distribution, as demonstrated in finite element analyses showing elevated resorption risks during rotations and translations after 4 mm bone loss. Prevention emphasizes interdisciplinary management, commencing orthodontic therapy only after achieving periodontal stability (e.g., no probing pocket depths ≥5 mm with bleeding on probing), utilizing light continuous forces (5-10 g for intrusions), incorporating treatment pauses for healing (2-6 months upon detecting resorption), and regular radiographic monitoring with cone-beam computed tomography (CBCT) for early detection. Additionally, endodontic interventions prior to orthodontics may reduce ERR in vital teeth by minimizing pulp-mediated inflammatory responses. This review underscores the need for tailored protocols to mitigate ERR, ensuring safe orthodontic outcomes in periodontally compromised patients while highlighting gaps in long-term prospective studies.

Keywords: External root resorption, Orthodontic treatment, Periodontal disease, Biological risk factors, Prevention strategies, Inflammatory mediators

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Introduction

Orthodontic treatment is a cornerstone of contemporary dentistry, aiming to achieve optimal dental alignment, functional occlusion, and esthetic outcomes through the controlled application of mechanical forces to teeth [1-5]. While orthodontic tooth movement (OTM) is generally predictable and safe in healthy patients, the process inherently induces biological responses within the periodontal ligament (PDL) and surrounding alveolar bone. Among the potential adverse effects, external root resorption (ERR) represents a significant clinical concern. ERR is a pathological process characterized by the progressive loss of cementum and dentin from the external root surface, mediated by odontoclastic cells that are activated in response to mechanical stress, local

inflammation, and cytokine signaling within the PDL [6].

The severity and clinical impact of ERR vary widely. Mild resorption often remains asymptomatic and self-limiting, detectable only radiographically, whereas severe resorption can compromise tooth structure, functional longevity, and esthetic outcomes, particularly when multiple teeth or incisors are involved [7]. ERR is multifactorial in origin, with risk determined by the interplay between orthodontic mechanics, biological tissue response, and patient-specific factors. Excessive or improperly directed forces, prolonged treatment durations, and certain tooth movements, such as intrusion or torque, have been consistently associated with higher ERR risk. In addition, inflammation and local tissue health modulate the susceptibility of odontoclasts to initiate resorptive activity.

Patients with pre-existing periodontal compromise present unique challenges in this context. Periodontally compromised individuals, classified as stage III or IV according to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, demonstrate reduced alveolar bone height, loss of attachment, increased tooth mobility, and impaired regenerative capacity of the PDL and surrounding alveolar bone [9, 10]. In these patients, the biomechanical environment is altered: the center of resistance of teeth shifts apically, and reduced PDL volume diminishes the tissue's ability to dissipate forces effectively. Consequently, the application of standard orthodontic loads may induce localized overloading, accentuating the inflammatory response and predisposing to accelerated ERR [11-13].

Epidemiological data indicate that ERR is a common occurrence in orthodontic populations, with prevalence ranging from 20% to 100% depending on diagnostic criteria, imaging modality, and treatment parameters [14]. In patients with periodontal involvement, the risk is markedly increased, with studies reporting resorption affecting up to 34.5% of teeth in advanced cases [15, 16]. Contributing factors include mechanical stress from orthodontic forces, occlusal trauma from malpositioned teeth, microbial biofilm accumulation, systemic conditions affecting bone metabolism, and compromised vascular supply [17]. These overlapping risk factors underscore the importance of interdisciplinary management, combining periodontal stabilization, careful force application, and ongoing monitoring to mitigate ERR progression.

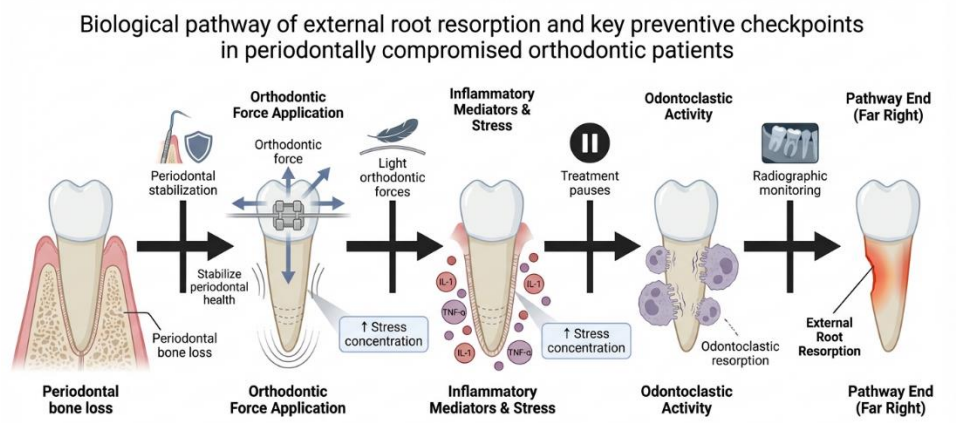

Figure 1. Biological pathway of external root resorption and key preventive checkpoints in periodontally compromised orthodontic patients

The objectives of this narrative review are threefold. First, it aims to provide a detailed overview of the pathophysiology of ERR in the context of orthodontic treatment, emphasizing cellular and molecular mechanisms relevant to periodontally compromised patients. Second, it seeks to identify key biological and mechanical risk factors for ERR, synthesizing evidence from studies published between 2020 and 2025 to highlight current consensus and emerging findings. Third, it explores evidence-based preventive strategies, including force modulation, adjunctive therapies, and treatment sequencing, to minimize the incidence and severity of ERR. By integrating recent clinical and experimental insights, this review intends to update practice guidelines, inform risk assessment protocols, and identify directions for future research in the interdisciplinary management of ERR in adults with compromised periodontal support.

Pathophysiology of External Root Resorption in Orthodontic Treatment

External root resorption (ERR) during orthodontic treatment, commonly referred to as orthodontically

induced external apical root resorption (OIEARR), represents a multifactorial pathological process arising from the interplay of mechanical, biological, and environmental factors [6, 7]. The initiating event occurs when orthodontic forces are applied to teeth, creating zones of compression and tension within the periodontal ligament (PDL). Compressive forces induce localized ischemia and hyalinization of the PDL, disrupting normal cellular architecture and triggering the recruitment and activation of odontoclastic and osteoclastic cells that resorb mineralized root surfaces [18-20]. In a healthy periodontium, this resorptive activity is typically counterbalanced by reparative cementum deposition, maintaining structural integrity. However, in patients with compromised periodontal support, the biomechanical environment is altered: reduced alveolar bone height and attachment loss change the distribution of forces across the root surface, prolonging the presence of hyalinized tissue and favoring sustained resorptive activity [21, 22].

Finite element analysis (FEA) studies have provided critical insights into these biomechanical changes. Progressive alveolar bone loss ranging from 0 to 8 mm significantly increases stress concentration on root surfaces during orthodontic movements. Rotational and translational movements, in particular, impose higher stress on residual PDL regions once bone loss exceeds approximately 4 mm, as uneven stress absorption occurs, concentrating 87.99–97.99% of applied load on specific root segments [21, 23]. These models underscore the importance of adapting force magnitude and direction in periodontally compromised patients to avoid focal overload and excessive ERR. Experimental evidence also demonstrates that heavy orthodontic forces, such as 100 g, upregulate receptor activator of nuclear factor kappa-B ligand (RANKL) in PDL stem cells, promoting osteoclastogenesis and enhancing resorptive activity, whereas lighter, physiologically calibrated forces reduce this response [24, 25]. Interestingly, endodontically treated teeth exhibit lower ERR compared to vital teeth, likely due to the absence of pulp-derived inflammatory mediators, highlighting the role of local cytokine environments in modulating resorptive susceptibility [6,26, 27]. Collectively, these findings emphasize that in periodontally compromised patients, chronic inflammation from periodontitis synergizes with mechanical stress, shifting the balance toward pathological resorption [9,15,28].

Impact of Periodontal Compromise on Root Resorption

Periodontal disease markedly exacerbates ERR during orthodontic treatment by impairing the structural and biological resilience of the supporting tissues [2, 9, 14, 29]. In advanced stage IV periodontitis, teeth are frequently affected by pathologic migration, occlusal trauma, and extensive bone defects, creating a biomechanically unstable environment that predisposes to accelerated resorption [14, 30]. Radiographic surveys indicate a high prevalence of ERR in patients with periodontitis, with up to 34.5% of teeth affected and rates climbing to 72.2% in stage III/IV disease. Localized ERR often correlates with areas of vertical or horizontal bone loss, emphasizing the relationship between alveolar support and resorptive vulnerability [15, 31].

Interdisciplinary case reports illustrate that even teeth deemed "periodontally hopeless" due to severe mobility can be salvaged with combined periodontal regeneration and carefully controlled orthodontic forces. However, untreated inflammation substantially increases ERR risk, underscoring the necessity of achieving periodontal stability before tooth movement [17,32]. Orthodontic appliances themselves may exacerbate the situation by increasing plaque accumulation and shifting the oral microbiome toward pathogenic species, thereby amplifying gingival inflammation and recession, particularly in thin gingival phenotypes [2, 9, 33]. Pilot studies addressing open bite corrections reveal that vertical intrusive forces on anterior teeth can provoke pronounced ERR, mirroring the pattern of accelerated attachment loss observed in compromised periodontium [18,34].

Therefore, a critical prerequisite for orthodontic treatment in periodontally compromised adults is the establishment of periodontal stability, defined by the absence of bleeding on probing (BOP), probing depths consistently below 5 mm, and controlled microbial burden [11,14, 35]. Only when these parameters are met can orthodontic forces be applied safely, minimizing the risk of ERR while promoting controlled tooth movement. Integration of biomechanical planning with periodontal management is thus essential to preserve root integrity, optimize treatment outcomes, and prevent long-term complications in this high-risk patient population.

Biological Risk Factors Associated with External Root Resorption

External root resorption (ERR) in periodontally compromised orthodontic patients is influenced by a complex interplay of biological, mechanical, and systemic factors that collectively determine susceptibility and severity [7, 9, 18]. Among patient-

specific determinants, genetic predisposition plays a substantial role, with heritability estimates as high as 70%, highlighting the influence of polymorphisms in genes regulating osteoclastogenesis, cytokine expression, and extracellular matrix remodeling [1, 7, 18, 36]. Tooth morphology is also critical; teeth with thin, tapered, or conical roots exhibit reduced surface area for stress dissipation, predisposing them to localized overloading and subsequent resorption. Prior dental trauma, whether from sports injuries, accidents, or parafunctional habits such as bruxism and thumb sucking, further increases risk, particularly in anterior teeth where mechanical stress is concentrated [1,7, 18].

In the context of periodontal compromise, chronic inflammation magnifies these intrinsic vulnerabilities. Dysbiotic microbial biofilms trigger persistent immune activation within the PDL, with elevated levels of pro-inflammatory cytokines—including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α)—that upregulate receptor activator of nuclear factor kappa-B ligand (RANKL) expression [6, 24, 37]. This cytokine milieu enhances odontoclastic differentiation and activity, prolonging resorptive phases and reducing the capacity for reparative cementum deposition. Consequently, the threshold for ERR is lower in patients with active periodontal inflammation, necessitating meticulous periodontal control prior to orthodontic intervention [9,15, 38].

Treatment-related factors further modulate risk. Orthodontic force magnitude, duration, and type are paramount; heavy continuous forces exceeding 50 g, prolonged treatment times, and intrusive or rotational movements have been consistently associated with increased ERR, especially in extraction cases where space closure imposes complex vectors on compromised roots [2, 9]. Finite element analysis (FEA) studies demonstrate that progressive alveolar bone loss alters stress distribution along the root surface, concentrating mechanical load in regions where the PDL is thinned or absent. Rotational and translational movements exacerbate localized stress, intensifying the risk of resorption in these structurally vulnerable zones [21,39].

Systemic conditions also influence susceptibility. Endocrine disorders, diabetes, and other metabolic dysfunctions exacerbate periodontal dysbiosis and impair tissue repair, indirectly increasing ERR risk [2, 14]. Additionally, cumulative trauma, whether from contact sports or repetitive micro-injuries, further predisposes anterior teeth to resorption due to chronic microdamage that compounds mechanical and inflammatory stimuli [1]. Collectively, these patient-, treatment-, and system-related factors interact synergistically in periodontally compromised individuals, necessitating individualized risk assessment and treatment planning [11,26, 40].

Cellular and Molecular Mechanisms

At the cellular level, ERR is mediated by odontoclasts—osteoclast-like cells capable of demineralizing and enzymatically degrading cementum and dentin [6, 24, 41]. Orthodontic force-induced PDL compression triggers an inflammatory cascade, characterized by increased RANKL expression from PDL stem cells and decreased secretion of its decoy receptor, osteoprotegerin (OPG), skewing the RANKL/OPG ratio toward resorption [24, 42]. In vital teeth, additional inflammatory mediators released from the pulp amplify this effect, explaining why root-filled teeth typically exhibit lower ERR rates [6, 26].

In periodontally compromised patients, the persistent presence of microbial endotoxins, such as lipopolysaccharides from Porphyromonas gingivalis, sustains cytokine release and prolongs clastic activity, creating a microenvironment conducive to extended root resorption [9,15]. Animal studies corroborate these findings, showing that heavy orthodontic forces activate osteoclasts primarily in the PDL rather than the pulp, leading to external rather than internal resorption [24]. Genetic variations in osteopontin, interleukin-1, and other cytokine-related genes modulate individual susceptibility, linking heritable traits to inflammatory responsiveness and ERR risk [18].

Reparative processes are also critically involved. In vital teeth, tertiary dentin formation serves as a protective barrier against internal resorption, whereas external repair relies on cementoblast activity along the root surface. In inflamed or compromised PDL, cementoblast function is often impaired, limiting the potential for natural repair and reinforcing the need for preventive mechanical strategies [11]. These mechanistic insights highlight potential targets for adjunctive therapies, such as pharmacologic modulation of RANKL/OPG signaling or local anti-inflammatory interventions, though current clinical practice primarily emphasizes mechanical modulation careful force calibration, intermittent loading, and treatment sequencing to minimize ERR in high-risk patients [7, 21].

Diagnostic and Monitoring Strategies

Accurate diagnosis and continuous monitoring are essential for the effective management of external root

resorption (ERR) in periodontally compromised orthodontic patients, particularly given the asymptomatic nature of early resorptive changes [2,9, 18]. Conventional two-dimensional imaging modalities, such as panoramic or periapical radiographs, provide limited sensitivity and may underestimate subtle root defects. Cone-beam computed tomography (CBCT), with its three-dimensional capability, allows precise evaluation of root morphology, bone levels, periodontal ligament (PDL) width, and early signs of resorption, offering superior diagnostic accuracy compared to conventional radiography [9,26]. Baseline CBCT imaging is crucial for documenting initial root anatomy, identifying previous trauma or pre-existing resorption, and assessing alveolar bone support, particularly in patients with advanced periodontal disease where cortical and trabecular bone architecture may already be compromised [2, 21].

Monitoring intervals should be individualized based on risk assessment, with high-risk cases such as stage III/IV periodontitis, thin root morphologies, or teeth subjected to heavy orthodontic forces benefiting from serial CBCT evaluations every 6 to 12 months. This allows for early detection of progressive resorption and facilitates timely interventions [2, 21]. Clinical indicators, although often subtle, can include increased tooth mobility, sensitivity to percussion or thermal stimuli, and changes in occlusion; however, these signs generally manifest only in advanced resorption [15]. Radiographic prevalence studies in periodontitis patients highlight the importance of routine surveillance, revealing ERR in over one-third of individuals undergoing orthodontic therapy [15].

In endodontically treated teeth, monitoring PDL space expansion can serve as a predictive marker for ERR under orthodontic forces, as these teeth typically demonstrate a lower baseline inflammatory response but remain susceptible to mechanical stress [26]. Interdisciplinary protocols recommend pausing orthodontic treatment if radiographic resorption exceeds approximately 3 mm, allowing a period of 2 to 6 months for reparative processes and stabilization before resuming movement [2,11]. Integrating these imaging and clinical monitoring strategies ensures early intervention, minimizing irreversible damage and optimizing outcomes in high-risk groups [14,17].

Preventive Measures and Clinical Recommendations

Prevention of ERR in periodontally compromised patients requires a holistic, multidisciplinary approach that prioritizes periodontal stabilization prior to initiating orthodontic forces [2,9,11,14]. Achieving periodontal endpoints—such as probing pocket depths (PPD) ≤5 mm, minimal bleeding on probing (BOP), and plaque scores below 15–20%—is fundamental. These conditions are typically achieved through a combination of non-surgical therapy (scaling and root planing), surgical interventions for residual pockets, and ongoing supportive periodontal care (SPC) to maintain tissue health [9,14].

Mechanical considerations play a pivotal role in minimizing ERR. Orthodontic forces should be light and continuous, particularly for intrusive movements, with recommended magnitudes of 5–10 g per tooth to reduce PDL ischemia and hyalinization [11,21]. Segmented mechanics and controlled force vectors should be employed to direct stress near the tooth's center of resistance, preventing tipping, uncontrolled rotation, or concentrated pressure on compromised roots [11,21]. Appliance selection must balance biomechanical control and hygiene: fixed labial systems are preferred for complex movements, while aligners may be advantageous in mild compromise due to ease of oral hygiene but are less effective in teeth with severe mobility or root vulnerability [14].

Incorporating strategic treatment breaks—ranging from 4 to 6 months—allows the PDL and surrounding bone to recover when early resorption is detected via CBCT, leveraging the tissue's natural reparative capacity [2,7]. Pre-orthodontic endodontic therapy can reduce ERR risk by eliminating pulp-derived inflammatory mediators that otherwise potentiate odontoclastic activity [6, 26]. Adjunctive measures include meticulous oral hygiene reinforcement, utilizing electric toothbrushes, interdental brushes, and chlorhexidine rinses, alongside structured SPC schedules every 1–6 months depending on risk profile [2, 9].

Long-term retention strategies, including fixed lingual wires or vacuum-formed splints, are critical to maintaining tooth position and preventing further bone loss post-treatment [11, 17]. Patient education is equally important; informing patients about potential risks, ensuring informed consent, and monitoring for trauma—particularly in sports-active individuals—can further mitigate ERR occurrence [1,18]. Collectively, these evidence-based preventive measures and monitoring protocols significantly reduce the incidence and severity of ERR while allowing safe and effective orthodontic treatment in periodontally compromised adults [7,15, 24].

Discussion

The integration of orthodontic treatment in periodontally compromised patients presents a complex interplay of mechanical, biological, and systemic factors that necessitate meticulous planning. One of the most significant challenges in this cohort is the heightened susceptibility to external root resorption (ERR), particularly orthodontically induced external apical root resorption (OIEARR). ERR arises from a convergence of mechanical stress, periodontal inflammation, and host-specific factors that activate odontoclastic cells, ultimately resorbing cementum and dentin [1,18]. While contemporary evidence suggests that orthodontic therapy can, under controlled conditions, stabilize or even enhance periodontal parameters, the presence of pre-existing bone loss, thin gingival phenotypes, or chronic inflammation increases the risk and severity of resorption, making individualized risk assessment imperative [2,6,18].

Synthesis of biological risk factors

Recent literature underscores the multifactorial nature of ERR, emphasizing genetic, systemic, and local contributors. Genetic predisposition plays a pivotal role, with polymorphisms in genes encoding pro-inflammatory cytokines, such as interleukin-1β (IL-1β), and structural proteins like osteopontin, contributing significantly to susceptibility. Heritability estimates for ERR range up to 70%, indicating a strong host determinant [4, 43]. Observational and cohort studies demonstrate that these polymorphisms modulate the inflammatory and clastic response to orthodontic forces, amplifying odontoclastic activity and prolonging resorption phases in susceptible individuals [43, 44]. When combined with chronic periodontal inflammation, these genetic factors act synergistically, as dysbiotic biofilms elevate RANKL, TNF-α, and IL-6 expression, perpetuating osteoclast-mediated root resorption [24,45].

Systemic conditions further exacerbate ERR risk in this population. Endocrine disorders, allergies, and asthma have been consistently linked to higher rates of ERR, with meta-analyses revealing increased odds for orthodontically induced inflammatory root resorption in these patients [46, 47]. The proposed mechanism involves altered immune modulation and an enhanced inflammatory milieu that favors odontoclastic activation. Similarly, metabolic syndromes such as diabetes impair periodontal healing and amplify susceptibility to ERR by promoting chronic low-grade inflammation and vascular dysfunction in the PDL [2,14]. These findings highlight the importance of thorough medical screening and interdisciplinary management in adult orthodontic patients with systemic comorbidities.

Local anatomical factors also significantly influence risk. Teeth with abnormal root morphologies—such as pipette-shaped, dilacerated, or tapered roots—exhibit uneven stress distribution during orthodontic loading, increasing susceptibility to ERR, as demonstrated by finite element analysis (FEA) studies [1,14,48]. Prior dental trauma further predisposes roots to resorption due to microstructural defects and pre-existing inflammatory priming of the PDL. Thin gingival phenotypes and reduced keratinized tissue exacerbate vulnerability, particularly during labial or intrusive movements, where dehiscence and localized resorption are more likely [2,15].

Treatment-related factors are critical, modifiable determinants of ERR. Force magnitude, duration, and type of movement profoundly influence resorption risk. Heavy forces (>50 g) and intrusive or rotational movements concentrate stress at the apical regions, especially in periodontally compromised teeth where alveolar bone loss has shifted the center of resistance apically [6,11,21]. Prolonged treatment durations, particularly those exceeding two years or involving extraction protocols, have been associated with severe ERR (>2 mm), predominantly affecting anterior teeth [49, 50]. In compromised periodontium, these mechanical stressors act in concert with chronic inflammatory stimuli, explaining the high prevalence of ERR observed in 34–72% of teeth in advanced stage III–IV periodontitis [2,15].

Clinical implications and risk management

The synthesis of these risk factors underscores the necessity of a comprehensive, interdisciplinary approach in managing ERR risk. Clinicians must integrate genetic, systemic, and local assessments into treatment planning, employing preventive strategies such as pre-orthodontic periodontal stabilization, force modulation, and meticulous appliance selection. For instance, segmented arch mechanics, TAD-supported intrusion, and light, continuous forces minimize stress concentration on vulnerable roots while allowing controlled tooth movement [11, 21]. Endodontically treated teeth may tolerate slightly higher forces due to reduced pulp-derived inflammatory signaling, whereas teeth with complex root morphology or prior trauma require conservative force application and closer radiographic monitoring [6, 26].

Monitoring strategies, including CBCT imaging, periodic clinical evaluations, and interval reassessments of mobility and periodontal status, enable early detection of ERR and timely intervention

[2,9,26]. Patient-specific factors, such as systemic comorbidities or inflammatory predispositions, should guide individualized force protocols and treatment sequencing, emphasizing minimal risk while optimizing tooth movement efficiency [1,14, 47].

In summary, ERR in periodontally compromised orthodontic patients results from an intricate interaction of host, local, and treatment-related factors. Understanding these mechanisms allows clinicians to anticipate high-risk scenarios, tailor biomechanical strategies, and implement preventive measures, ultimately improving the safety and predictability of orthodontic therapy in this vulnerable population.

Interplay between periodontal compromise and external root resorption

Periodontal disease fundamentally modifies the biomechanical and biological environment in which orthodontic tooth movement occurs, substantially predisposing teeth to external root resorption (ERR) [6, 18, 51]. Reduced alveolar bone height and compromised attachment increase stress concentration within the periodontal ligament (PDL), altering normal force dissipation during orthodontic loading. Finite element analysis (FEA) studies demonstrate that after approximately 4 mm of alveolar bone loss, 87–98% of applied orthodontic forces are transmitted directly to the root surfaces, particularly during rotational or intrusive movements, which amplifies hyalinization and triggers sustained odontoclastic activity [14, 48, 6, 11]. These biomechanical alterations are compounded by the persistent presence of microbial biofilms within periodontal pockets, which sustain a pro-inflammatory microenvironment. Elevated expression of RANKL relative to OPG in these contexts promotes odontoclastogenesis, tipping the balance toward resorption rather than repair [4, 24, 45].

Interdisciplinary case series highlight the transformative potential of coordinated therapy in periodontally compromised patients. Teeth previously considered “hopeless” due to severe bone loss or mobility can become functionally viable when periodontal inflammation is adequately controlled prior to orthodontic intervention [17, 51]. Radiographic evaluations consistently show that the prevalence of ERR is higher in Stage III and IV periodontitis, with resorption frequently localized to regions exhibiting vertical or combined bone defects [2,15]. Endodontic status also modulates risk: meta-analyses reveal reduced ERR in root-filled teeth, likely due to the elimination of pulp-mediated inflammatory signaling [26, 52]. Nevertheless, in the presence of significant periodontal compromise, even endodontically treated teeth remain susceptible to external mechanical stress unless periodontal stability is firmly established [17, 53].

Preventive strategies: Evidence and efficacy

Mitigation of ERR in this patient population relies on a combination of interdisciplinary protocols, force optimization, tissue phenotype management, and vigilant monitoring [7, 18, 54]. Paramount among these strategies is the achievement of periodontal stability before orthodontic intervention. This includes probing depths <5 mm, absence of bleeding on probing (BOP), and plaque indices below 20%, which collectively reduce inflammatory stimuli and provide a more resilient PDL [9]. Randomized controlled trials support the clinical relevance of delaying orthodontic treatment until such regenerative and stabilization endpoints are met, demonstrating lower ERR incidence in patients who initiate orthodontics post-periodontal therapy [9].

Soft and hard tissue phenotype modification further enhances protective capacity. Gingival augmentation in thin biotypes, as shown in systematic reviews, increases keratinized tissue width and reduces the susceptibility to dehiscence and subsequent ERR without compromising orthodontic treatment outcomes [21, 55].

Force application strategies are equally critical. Light, continuous forces within the range of 5–15 g, combined with segmented arch mechanics, minimize PDL ischemia and reduce hyalinized areas that favor resorption [56, 57]. Clear aligners may present an additional advantage in select cases, as umbrella reviews report lower ERR rates compared to conventional fixed appliances, likely due to their gentler force profiles and improved hygiene facilitation [56, 57]. Scheduled treatment pauses of 3–6 months upon early radiographic detection of resorption allow for reparative cementum deposition, as recommended in contemporary clinical guidelines [7,16, 54].

Emerging pharmacologic adjuncts offer potential avenues for ERR prevention. Animal studies demonstrate that sphingosine-1-phosphate modulators and other osteoclast-targeting agents can inhibit excessive root resorption, although human trials are needed to validate safety and efficacy [4, 27].

Diagnostic monitoring remains a cornerstone of prevention. Cone-beam computed tomography (CBCT) provides superior detection of early volumetric changes compared to two-dimensional imaging, enabling timely identification of ERR [2, 10, 26]. Multicenter randomized controlled trials corroborate that routine intermediate CBCT imaging facilitates early intervention and mitigates progression,

though debate persists regarding its cost-effectiveness in non-extraction, low-risk cases [29]. Complementary to imaging, patient education on trauma avoidance—such as the use of protective mouthguards during sports—and meticulous oral hygiene are essential for maintaining periodontal health and reducing additive risk factors for ERR [1, 18].

Overall, the interplay between compromised periodontium and orthodontic forces necessitates a proactive, evidence-based approach that integrates tissue stabilization, individualized biomechanics, phenotype management, and robust monitoring to minimize ERR and optimize long-term outcomes.

Clinical Implications

Clinically, these findings advocate for personalized risk assessments using tools like genetic screening and baseline CBCT to stratify patients [7,43,44]. In periodontally compromised individuals, orthodontic goals should prioritize functional stability over aesthetics, employing biomechanics that respect altered periodontal dynamics [6,11, 14]. Interdisciplinary collaboration between orthodontists and periodontists is essential, with protocols emphasizing pre-treatment stabilization and intra-treatment maintenance [18, 51].

Economically, preventing ERR reduces long-term costs associated with tooth loss or prosthetics, enhancing patient satisfaction [17]. However, in high-risk cases (e.g., trauma history, genetic susceptibility), informed consent must highlight potential outcomes, including treatment abandonment if resorption progresses [7, 54].

Limitations of Current Literature

Despite advances, limitations persist. Most studies are retrospective or observational, with few high-quality RCTs addressing periodontally compromised cohorts specifically [9, 29]. Heterogeneity in ERR measurement (e.g., linear vs. volumetric) and force quantification hampers meta-analyses [49, 50]. Long-term follow-up (>5 years) is scarce, obscuring the permanence of reparative processes [7, 18]. Additionally, while FEA models provide mechanistic insights, they often oversimplify biological variability [14, 48]. Genetic studies are limited by small samples and ethnic biases, warranting larger, diverse cohorts [43, 44]. Finally, emerging therapies like tissue engineering for resorption repair lack clinical validation [3, 25].

Conclusions

In conclusion, ERR in periodontally compromised orthodontic patients stems from intertwined biological risk factors, including genetic predispositions, systemic conditions, and mechanical stressors, amplified by periodontal inflammation. Prevention hinges on achieving periodontal stability, employing light forces, regular monitoring with CBCT, and considering endodontic interventions. Evidence from 2020-2025 underscores the efficacy of interdisciplinary approaches in mitigating risks, ensuring safer outcomes.

Future research should prioritize prospective RCTs evaluating long-term ERR in compromised patients, incorporating genetic and microbiome analyses for personalized protocols. Investigating novel adjuncts, such as anti-resorptive biologics or AI-driven force optimization, could revolutionize prevention. Additionally, standardized ERR grading systems and international registries would enhance comparative studies, ultimately refining guidelines for this high-risk population.

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