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Orthodontic Tooth Movement in Periodontally Compromised Patients: Biological Basis and Clinical Complications

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ABSTRACT

Orthodontic treatment in patients with periodontitis, particularly stages III and IV, presents significant clinical challenges owing to compromised periodontal support, alveolar bone loss, and elevated inflammatory response. This narrative review explores the biological interactions between orthodontic forces and periodontal tissues in periodontitis patients, aiming to clarify mechanisms of inflammation, bone remodelling, and implications for clinical decision-making. A comprehensive literature search was conducted using PubMed, Scopus, ScienceDirect, and Google Scholar, limited to English-language articles published between January 2015 and January 2025. Relevant studies were retrieved using keywords such as "biology" OR "periodontium" OR "tooth supporting structures" OR "inflammation" OR "cell and tissue-based therapy" AND "orthodontic" AND "tooth movement" AND "periodontitis," focusing on biological responses of periodontal tissues during orthodontic therapy in periodontally compromised individuals. Current evidence demonstrates a complex interplay of periodontal ligament adaptation, alveolar bone turnover, and inflammatory mediators, including cytokines and prostaglandins, in response to orthodontic forces. Alterations in the subgingival microbiota and systemic host responses further influence treatment stability. Regenerative periodontal interventions, including guided tissue regeneration and bone grafting, appear promising in enhancing periodontal support during orthodontic tooth movement. Successful management of orthodontic treatment in periodontitis patients demands a multidisciplinary approach, precise control of mechanical forces, and modulation of inflammation. Integrating regenerative therapies and host-modulatory strategies holds potential to improve long-term periodontal stability and treatment outcomes.

Keywords: *Bone remodelling; inflammation; mediators; orthodontic tooth movement; periodontitis; regenerative periodontal therapy*

INTRODUCTION

Periodontitis is a chronic, multifactorial inflammatory disease characterised by progressive destruction of the tooth-supporting structures (Papapanou *et al.*, 2018). According to the current classification system, advanced periodontitis (Stage III and IV) presents as one of the highly prevalent global diseases, where patients often present with complex defects, such as significant bone loss and pathological tooth migration (Tonetti *et al.*, 2018). The management of these complex cases necessitates an interdisciplinary approach for functional and aesthetic rehabilitation (Herrera *et al.*, 2022).

Orthodontic therapy is a crucial component of interdisciplinary management, as it realigns teeth and establishes a stable occlusal relationship. However, a unique biological challenge is present when applying orthodontic forces in a periodontally compromised environment. Orthodontic tooth movement (OTM) may stimulate alveolar bone remodelling, which, depending on a tightly regulated, force-induced inflammatory process (Krishnan & Davidovitch, 2006). In a healthy periodontium, this process is balanced. However, in periodontitis, this balance is disrupted by a pre-existing inflammatory condition caused by microbial dysbiosis (Hajishengallis & Chavakis, 2021). The interplay between the underlying periodontal disease and the orthodontically induced inflammation can act as a "double-edged sword", potentially leading to synergistic tissue repair, or if mismanaged, can exacerbate tissue destruction (Li *et al.*, 2021; Papageorgiou *et al.*, 2024).

Therefore, a better understanding of biological interactions between orthodontic forces and periodontal tissues helps to improve the effectiveness of clinical practice. This narrative review aims to explore these mechanisms, with a specific focus on the dysregulation of the Receptor Activator of Nuclear Factor Kappa-B Ligand (RANKL)/RANK/osteoprotegerin (OPG) pathway, the role of key inflammatory cytokines such as IL-1β, TNF-α, and prostaglandin (PGE2), and the compromised function of periodontal ligament (PDL) cells in the context of advanced periodontitis. By synthesising evidence, this review helps to clarify the biological basis for clinical decision-making in the orthodontic management of periodontitis patients.

MATERIAL AND METHODS

A comprehensive literature search was conducted to identify publications on the biology of OTM in patients with periodontitis. The electronic databases PubMed, Scopus and ScienceDirect were systematically searched. Additionally, Google Scholar was used to ensure coverage of grey literature and cross-verify results. All results from Google Scholar were carefully evaluated to ensure a comprehensive search that prioritises quality and relevant articles, with priority given to peer-reviewed journal articles. The search was limited to the articles published in the English language between January 2015 and January 2025 to capture the most recent evidence.

The search strategy used a combination of keywords and Boolean operators to ensure a focused retrieval of relevant literature. The keywords used were: ("biology" OR "periodontium" OR "tooth supporting structures" OR "inflammation" OR "cell and tissue-based therapy") AND ("orthodontic") AND ("tooth movement") AND ("periodontitis"). Studies were selected based on pre-defined criteria.

The inclusion criteria include narrative reviews, systematic reviews, original research, and clinical trials that specifically investigated the biological mechanisms, cellular responses, or inflammatory mediators involved in OTM in the context of periodontitis. While the search was focused on the last decade (2015–2025) to capture contemporary evidence, several highly relevant older publications were also

included to provide crucial foundational context. Exclusion criteria were case reports, letters to the editor, posters, and studies that were not published in English.

The literature search and selection were performed collaboratively by the authors. To minimise selection bias, the final inclusion of articles was determined by consensus. The process involved an initial keyword database search, followed by a screening of titles and abstracts. The full texts of potentially eligible articles were then assessed. Furthermore, the reference lists of retrieved articles were manually searched to identify additional related studies (a "snowballing" technique). The initial search yielded over 60 articles. After applying the inclusion and exclusion criteria, a final set of 47 articles was selected for in-depth analysis and synthesis in this narrative review.

From the 47 articles included in this narrative review, four key studies were selected for inclusion in the summary table (Table 1). The selection was based on their representatives of core themes (PDL biology, inflammation, regeneration), a high level of evidence (prioritising randomised controlled trials and high-impact reviews), and conceptual significance in explaining clinical concepts. A separate summary of key studies specifically investigating OTM in periodontitis patients is presented in Table 2.

Table 1 Key studies on the biology of normal OTM

Study (author, year)	Study type	Key focus/category	Major findings	Clinical/research implication
Garlet *et al*., 2007	Original research	Inflammatory mediators	In humans, the compression side of OTM shows a distinct pro-inflammatory cytokine profile (e.g., RANKL, TNF-α) compared to the tension side.	Provides direct human evidence of the site-specific inflammatory cascade driving tooth movement.
Yamaguchi & Fukasawa, 2021	Review	Inflammatory mediators in orthodontics	Inflammatory mediators like PGE2 are central to both OTM and pathological root resorption.	Emphasises monitoring inflammation to achieve desired movement while minimising root resorption.
Huang *et al*., 2018	Review	PDL/biology	Periodontal ligament stem cells (PDLSCs) are key mechanosensitive cells that regulate bone remodelling during OTM.	Directs future research towards the use of PDLSC potential for optimising tooth movement.
Naranjo *et al*., 2006	Original research	Microbiome/host	Orthodontic appliance placement leads to significant quantitative and qualitative changes in the subgingival microbiota within 3 months.	Highlights the importance of intensified oral hygiene and monitoring at the start of orthodontic treatment.

Table 2 Key studies on OTM in periodontitis

Study (author, year)	Study type	Key focus/category	Major findings	Clinical/research implication
Li *et al.*, 2021	Review	PDL/biology	Orthodontic forces in a compromised periodontium act as a "double-edged sword", capable of promoting healing or exacerbating tissue destruction.	Highlights the critical need for precise force control and inflammation management.
Jepsen *et al.*, 2023	Randomised controlled trial	Regenerative therapy	The combination of regenerative periodontal surgery and orthodontics in stage IV periodontitis patients significantly improves clinical outcomes and quality of life.	Provides high-level evidence supporting an interdisciplinary approach for complex cases.
Garbo *et al.*, 2022	Retrospective clinical study	Biomechanical	Managing stage IV periodontitis requires interdisciplinary planning, considering the apically displaced centre of resistance and the risk of uncontrolled tipping.	A practical guide for clinicians on force system design in periodontally compromised dentitions.
Rath-Deschner *et al.*, 2021	Original research	Microbiome/host	The pathogen F. nucleatum synergises with orthodontic mechanical strain to amplify pro-inflammatory cytokines (IL-6 but not CXCL2) in PDL fibroblasts.	Suggests that microbial control is crucial to prevent an exaggerated inflammatory response during orthodontics.
Martin & Sanz, 2024	Review	Regenerative therapy	OTM can be successfully conducted after periodontal regeneration of intrabony defects, which improves the overall prognosis.	Confirms the clinical viability and sequence of combining regeneration with orthodontics.
Papageorgiou *et al.*, 2024	Review	Biomechanical	Treating stage IV periodontitis with orthodontics requires a hyper-conservative approach with low-force mechanics and frequent monitoring.	Offers a modern, evidence-based clinical protocol for managing the challenging cases.

PERIODONTAL TISSUE RESPONSE TO ORTHODONTIC FORCES

The primary aim of OTM is to align malposition teeth to an optimal position to improve both dental occlusion and aesthetics. According to the recently published clinical practice guidelines for the treatment of periodontitis, the initiation of OTM in periodontitis patients is only indicated once the endpoints of periodontal therapy (periodontal stability) have been achieved (Sanz *et al.*, 2020; Herrera *et al.*, 2022). This is because a compromised periodontal tissue, commonly found in stages III and IV, is highly susceptible to damage from orthodontic

forces (Herrera *et al*., 2022). When combining periodontal and orthodontic treatments, it is important to understand the impact of the periodontal tissues and how they are likely to influence the outcomes (Martin & Sanz, 2024). The type of orthodontic appliance used commonly influences the amount of force applied, which determines the effectiveness and safety of the treatment (Melsen, 2001). Improper selection of orthodontic devices influenced the magnitudes of forces, which can disrupt the biofilm composition and reactivate periodontitis in susceptible patients (Viglianisi *et al*., 2025). Therefore, careful monitoring of magnitude forces and tooth movement throughout the orthodontic treatment in periodontitis, specifically in stage IV periodontitis, helps in reducing the risk of exacerbating the periodontal disease (Herrera *et al*., 2022).

Cellular Interactions and Remodelling Events in the Periodontal Ligament

The PDL is defined as a dense fibrous connective tissue structure that provides support to the teeth and consists of collagenous fibre bundles (Li *et al*., 2021). At the cellular level, PDL cells are the first to respond to orthodontic forces by preventing the encroachment of bone and cementum into the PDL space, thereby maintaining their normal width (Yong *et al*., 2022). Coordinated action of bone mesenchymal stem cells (BMSCs), periodontal ligament stem cells (PDLSCs), PDL fibroblasts, osteoblasts, osteocytes, and osteoclasts is required in tooth movement (Cho *et al*., 2010). These cells regulate the bone remodelling process by converting orthodontic force into intracellular signals (Qin *et al*., 2020). Orthodontic forces that are applied to teeth will cause movement of the fluid in the PDL space and distort the PDL component, which leads to the release of numerous molecules that initiate the bone remodelling process (Krishnan & Davidovitch, 2006). In periodontally compromised patients, PDL commonly becomes inflamed and compromises the attachment between tooth and alveolar bone. This disorganised ligament structure affects the PDL's ability to adapt to orthodontic forces, making it less responsive to any mechanical changes and thus altering the mechanical properties (Papageorgiou *et al*., 2024). The stress transmission and cellular responses were also affected if the cellular activity of the PDL was reduced. This changes the force required to move teeth, leading to less predictable OTM (Willmot, 2008). The PDLs have been shown to play a critical role in generating biological signals that are transmitted to the alveolar bone to initiate bone remodelling, which is essential for tooth movement.

Orthodontic Force-Induced Bone Remodelling: Cellular and Molecular Perspectives Normal Biology of OTM

In a healthy periodontium, cellular responses to mechanical stimuli regulate bone remodelling, facilitating OTM. The application of orthodontic force triggers a biological cascade within the PDL and alveolar bone, causing changes in fluid flow and creating areas of tension and compression around the tooth (Krishnan & Davidovitch, 2006). This initiates an aseptic inflammatory response, leading to the recruitment and activation of key cells: osteoclasts on the compression side for bone resorption and osteoblasts on the tension side for bone formation (Krishnan & Davidovitch, 2006).

A critical molecular pathway regulating this balance is the RANK-RANKL-OPG system (Fig. 1). Osteoblastic lineage cells release Receptor Activator of Nuclear Factor Kappa-B Ligand (RANKL), which binds to the RANK receptor on pre-osteoclasts, promoting their differentiation and activation. This process is naturally counterbalanced by osteoprotegerin (OPG), a decoy receptor secreted by osteoblasts, which inhibits the RANKL-RANK interaction and thus modulates the rate of bone resorption (Patil *et al*., 2013). The precise equilibrium between RANKL and OPG is essential for controlled tooth movement.

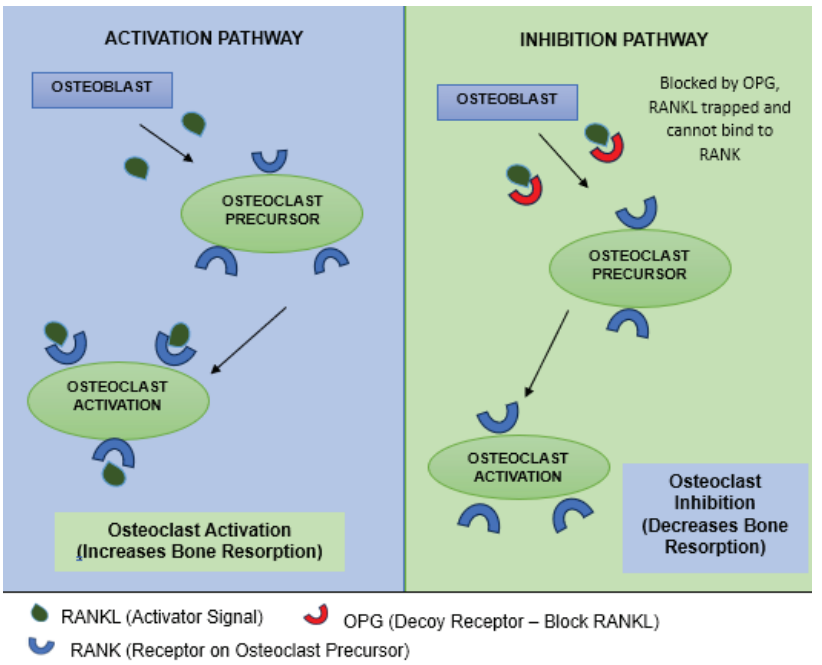

Fig. 1 Pathway of RANKL-RANK-OPG.

Alterations in periodontitis

This homeostatic balance is disrupted in periodontitis patients. The pre-existing, chronic inflammatory environment alters the tissue's response to orthodontic forces. The dysregulated immune response leads to elevated levels of pro-inflammatory cytokines (e.g., IL-1β, TNF-α, PGE2), which further stimulate osteoclastogenesis via the RANKL pathway (Li *et al.*, 2021). Concurrently, the bone formation capacity is often impaired due to the compromised function of osteoblasts and PDL cells within the inflamed tissue. This creates a significant imbalance, where resorption outpaces formation. Furthermore, the hyalinised tissue that forms in response to pressure fails to remodel efficiently, and the overall reduction in bone support dissipates orthodontic forces, leading to unpredictable tooth movement (Papageorgiou *et al.*, 2024).

Clinical implications and safety considerations

The alteration in this biological process has direct clinical consequences. Reduced bony support and hyper-inflammatory response significantly increase the risk of orthodontically induced inflammatory root resorption (OIIRR) and uncontrolled bone loss (Yong *et al.*, 2022). A conservative biomechanical approach is important in preventing these conditions. The application of lighter forces and the use of simple mechanics help avoid excessive strain on the compromised periodontium (Viglianisi *et al.*, 2025). Careful monitoring of inflammation and root integrity throughout treatment is also essential for achieving predictable, safe tooth movement in periodontitis patients.

INFLAMMATION IN ORTHODONTICS: BALANCING BONE REMODELLING AND PERIODONTAL HEALTH

Immediate Molecular Responses

OTM relies on the inflammatory response that is triggered by orthodontic force. This cascade of molecular events can be distinguished based on the timing and function. The rapid release of key mediators occurs within hours of the application

of orthodontic forces, triggeringcute inflammatory responses (Yamaguchi & Fukasawa, 2021). Prostaglandin (PGE2) acts as a key signal in the bone resorption process. Active tooth movement started when the gingival crevicular fluid showed an increase in the number of PGE2 expression in the PDL and alveolar bone (Ngan *et al.*, 1990). At the same time, the cellular recruitment process occurs, during which leukocytes and resident cells secrete pro-inflammatory cytokines such as IL-1β, TNF-α, and IL-6 (Patil *et al.*, 2013; Huang *et al.*, 2014).

Sustained Remodelling and The Role of Cytokines

The bone remodelling process sustained over the subsequent days and weeks. Osteoclastogenesis is stimulated, and osteoblast function is modulated by cytokines to maintain a cycle of tissue turnover. In healthy individuals, this inflammatory process is balanced by anti-inflammatory cytokines, such as IL-10 and TGF-β, which are also involved in tissue healing and the bone regeneration process (Garlet *et al.*, 2007; Krishnan & Davidovitch, 2009).

Exacerbation in the Periodontitis Context

The balance is severely disrupted in periodontitis patients. Before orthodontic forces are applied, the periodontium is already inflamed, with persistently elevated levels of pro-inflammatory cytokines (Garbo *et al.*, 2022). Introducing orthodontic force creates a synergistic effect in which the forces act as a secondary inflammatory stimulus, combined with pre-existing inflammation, leading to an exaggerated and dysregulated response. The bone formation and healing process is impaired due to significantly increased osteoclast activity and bone resorption, which then accelerates periodontal destruction (Xiao *et al.*, 2015; Hajishengallis & Chavakis, 2021).

Clinical Implications for Management

The term "double-edged sword" best describes the use of orthodontic forces in periodontally compromised patients. It can promote a favourable healing response with carefully controlled forces but can also increase the risk of rapid bone loss, orthodontically induced root resorption, and the reactivation of an existing inflammatory response if managed improperly (Papageorgiou *et al.*, 2024; Viglianisi *et al.*, 2025). Thus, the inflammatory timeline is fundamental to be understood, as strict control of inflammation before and during orthodontic therapy was crucial in determining clinical success.

ALVEOLAR BONE INTEGRITY AND ORTHODONTIC FORCES: CONSIDERATIONS FOR PERIODONTALLY COMPROMISED CASES

The hyalinisation process, where forces that are applied to the periodontium in the orthodontic treatment compromise the blood flow in PDL, leading to cell death. This process initiates tooth movement by resorbing the area of dead tissue (Patil *et al.*, 2013). This process occurs more commonly in intact periodontium than ineriodontitis patients, where loss of existing alveolar bone might complicate orthodontic treatment (Tonetti & Sanz, 2019). The alveolar bone becomes less resilient and more susceptible to stress, reducing its ability to withstand orthodontic force. Any changes in the magnitude of orthodontic forces may lead to further alveolar bone loss and root resorption, which can affect the treatment prognosis.

Different types of alveolar bone may also affect the response of orthodontic force. A compact and dense bone, cortical bone responds more slowly compared to trabecular bone, which is more porous and dynamic. Higher surface

area and metabolic activity in trabecular bone make it susceptible to rapid resorption, especially in periodontally compromised areas (Niramitchainon *et al.*, 2020). The stability of the tooth and its surrounding structures may be affected during orthodontic treatment if the resorption rate increases. A shorter time to trabecular bone resorption in periodontal disease patients raised the possibility of complications, such as excessive bone loss and root resorption (Niramitchainon *et al.*, 2020).

The transmission of orthodontic forces may change when alveolar bone loss occurs, due to reduced one support (Choi *et al.*, 2016; Zasciurinskiene *et al.*, 2018). Dehiscence and fenestration are more likely to occur in patients with stage IV periodontitis, especially when they are subjected to high-magnitude orthodontic forces. These defects occur in areas of the bone weakened and thinned by periodontal disease. This exposure complicates OTM and raises the risk of additional bone loss (Sanz *et al.*, 2020).

The strength and direction of orthodontic forces need to be considered in periodontal disease patients. Excessive forces applied can cause gingival recession and various soft tissue complications (Melsen, 2001; Sifakakis *et al.*, 2018). The apically displaced centre of resistance in reduced periodontal support may complicate the biomechanics of tooth movements and affect the effectiveness of applied forces. Thus, it is necessary for an orthodontist to precisely control the magnitude and direction of forces to prevent undesirable results such as uncontrolled tipping and bone loss (Garbo *et al.*, 2022).

Regenerative therapies become other options in treating patients with compromised periodontal health. These therapies help provide a stable foundation for orthodontic force application by restoring lost periodontium. Bone replacement grafts are key approaches that fill the defect and provide a scaffold for new bone growth, while guided tissue regeneration (GTR) selectively guides the growth of periodontal tissues using barrier membranes (Sculean *et al.*, 2015). There is evidence from systematic reviews and randomised controlled trials that demonstrates significantly improved clinical and patient-reported outcomes when combined with orthodontic treatment.

OPTIMISING ORTHODONTIC OUTCOMES THROUGH REGENERATIVE PERIODONTAL THERAPY

Regenerative therapies have demonstrated good prognosis in regenerating periodontal tissue lost (Sculean *et al.*, 2015). However, they are often combined with the placement of different biomaterials that are used as bone replacement grafts due to the lack of scaffold effect (Martin & Sanz, 2024). In healthy patients with reduced periodontium, a combination of regenerative therapy with orthodontic treatment provides better outcomes (Attia *et al.*, 2012). This combined therapy may be particularly effective, particularly in the presence of intrabony defects, based on clinical studies (Stavropoulos *et al.*, 2022).

A favourable anatomical environment for tooth movement was established after the regenerative therapies successfully restored the periodontal support. The primary approach is GTR, a surgical technique that uses a barrier membrane. This membrane prevents the apical migration of the gingival epithelium and excludes the gingival connective tissue fibroblasts from the healing wound. By creating this protected space, it selectively guides the healing process and formation of new cementum, PDL and alveolar bone.

Key components and adjuncts used in regenerative therapy include:

(1) Bone replacement grafts: Materials such as autograft, allograft, xenograft or synthetic grafts are often used with GTR. They provide a scaffold to support bone fill and stabilise the regenerative site, which is important for improving the treatment prognosis.

(2) Biologic agents: Substances like enamel matrix derivatives (EMD) and autologous platelet concentrates (APC) are applied as adjuncts to the root surface or graft material.

Biologics mimic natural developmental signals and enhance the wound-healing environment, thereby improving the quality of regeneration achieved with GTR.

GTR, often combined with bone grafting, has been shown to improve treatment outcomes in patients with periodontal disease, particularly for intra-bony defects. In this combined approach, bone grafts act as a scaffold for bone fill and defect resolution, while GTR membrane facilitates the true regeneration of the periodontal attachment (new cementum, PDL and alveolar bone), which improves tooth stability. Restored periodontal support increases the efficacy of subsequent orthodontic treatment by providing a stable foundation for tooth movement (Jepsen *et al.*, 2023). The regenerative process aims to eliminate periodontal pockets, promote the formation of new functional attachment, and, finally, improve the overall tooth structure and support (Viglianisi *et al.*, 2025).

The timing and control of orthodontic forces are critical after regenerative therapy. Forces applied too early or too aggressively can affect long-term stability by disrupting the healing process and compromising regenerative outcomes (Herrera *et al.*, 2022). Therefore, orthodontic therapy is typically initiated only after complete osseous healing and maturation of the regenerated site. OTM can be successfully integrated, promoting further alveolar bone modelling, and enhancing the final functional and aesthetic result (Martin *et al.*, 2022). Careful monitoring of host inflammatory responses and microbiological control is essential throughout this step to prevent complications.

MICROBIOTA AND HOST RESPONSE ADAPTATION

Orthodontic equipment sets off a series of biological events that can make treating patients with periodontitis more difficult. These events follow a specific order, from the microbial shift to the clinical result. By encouraging bacterial accumulation and changing the subgingival microbiota towards more pathogenic conditions, the appliances function as biofilm-retentive factors (Zasciurinskiene *et al.*, 2018; Polizzi *et al.*, 2024). Gingival hyperplasia, which widens the gingival crevice and creates a favourable environment for pathogenic bacteria to predominate, frequently coexists with these alterations (Patil *et al.*, 2013).

This altered environment then directly initiates the host's inflammatory response. Periodontal pathogens, such as Fusobacterium nucleatum, can increase the inflammatory response under orthodontic mechanical strain. This pathogen increased the signal by producing more IL-6 and CXCL2 in PDL fibroblasts, thereby exacerbating the local inflammatory reaction to orthodontic forces (Rath-Deschner *et al.*, 2021).

This local host-pathogen interaction can be increased by systemic health conditions. In patients with systemic conditions, such as diabetes, baseline inflammation is already elevated. This compromised condition exacerbates periodontal breakdown during orthodontic movement, impairs the body's regenerative capacity, and increases the risk of treatment failure (Fuhrmann, 2002; Herrera *et al.*, 2022).

This signified the more complex and unpredictable treatment environment. Efficacy of orthodontic therapy in periodontitis patients may be impacted by the interactions of appliance-induced microbial changes, underlying systemic vulnerability and dysregulated host inflammatory response (Popova *et al.*, 2013). Therefore, careful consideration of this interaction helps improve treatment planning and outcomes.

FUTURE DIRECTIONS IN ORTHODONTIC MANAGEMENT OF PERIODONTITIS

Periodontal destruction in periodontitis patients offers challenges to the orthodontic team. Each step in orthodontic treatment needs to be carefully assessed in order to prevent any exacerbation of periodontal destruction. Currently, the development

of regenerative therapies and biological approaches provides a better option to improve treatment outcomes. Knowledge of the biological mechanisms underlying tooth movement may help develop new treatment approaches, with the final objective of improving the effectiveness of orthodontic therapy, specifically for periodontitis patients (Li *et al*., 2021). Regenerative therapies such as enamel matrix derivatives, barrier membranes, and bone replacement grafts give new hope in improving periodontal healing and OTM in severe periodontitis (Papageorgiou *et al*., 2024). An effective and predictable treatment outcome could be achieved with this therapy, as it helps in managing the main problems, which are alveolar bone loss and enhancing periodontal support. For future perspective, integration of tissue engineering and stem cell biology shows exciting prospects in overcoming the challenges of compromised periodontal tissues. By utilising the capabilities of stem cells in restoring bone loss and tissues, this will provide a promising future for periodontitis patients in receiving orthodontic treatment (Patil *et al*., 2013).

Establishing healthy oral conditions, improving functional occlusion and realignment of the teeth can be achieved by doing orthodontic treatment (Garbo *et al*., 2022). For better orthodontic outcomes, maintaining good oral hygiene helps prevent plaque accumulation and inflammation, which might negatively affect the microbial flora (Naranjo *et al*., 2006). In the future, periodontitis patients may benefit from specific treatment planning that focuses on their individual bone metabolism and immune responses. Orthodontists can improve the effectiveness of treatment, minimise complications, and increase the overall stability of teeth and periodontium if they tailor treatmento patients' individual biological needs (Chapple *et al*., 2018). Regenerative approaches may become a better option in orthodontic treatment if practitioners understand the signalling molecules involved in tooth eruption and alveolar bone remodelling, as these significantly improve bone support (Viglianisi *et al*., 2025).

SUMMARY AND CLINICAL IMPLICATIONS

This review has examined the biological interactions between orthodontic forces and periodontal tissues in patients with periodontitis. The key finding is that pre-existing inflammation fundamentally alters the RANKL/RANK/OPG pathway and cytokine signalling (IL-1β, TNF-α, PGE2), shifting the balance from controlled bone remodelling towards accelerated resorption and impaired healing. Consequently, orthodontic forces act as a "doubleedged sword", capable of promoting favourable tooth movement when inflammation is controlled, but risking rapid bone loss and root resorption when it is not.

Successful orthodontic management in periodontally compromised patients requires adherence to three core clinical principles. First, achieving documented periodontal stability and inflammation control before force application. Second, using light, controlled forces with continuous monitoring. Third, incorporating regenerative periodontal therapy for intrabony defects. A stepwise clinical framework is summarised in Fig. 2.

For future research and clinical practice, specific treatment protocols based on the patient's inflammatory, and bone metabolic profile are needed. Standardised outcome measures and integration of stem cell biology hold promise for improving longterm stability in periodontitis patients undergoing orthodontic therapy.

Orthodontic management begins only after inflammation is controlled and periodontal stability is achieved. Definition of key criteria including (1) Periodontal stability: Bleeding on probing (BOP) < 10% of sites; probing pocket depths (PPD) ≤ 4 mm at previously diseased sites; and no further clinical attachment loss (CAL) for ≥ 3 months postperiodontal therapy. (2) Inflammation control: Fullmouth bleeding score (FMBS) < 15%; fullmouth plaque score (FMPS) < 20%; and no overt gingival erythema, oedema or suppuration. (3) Stepwise pathway: Antiinfective therapy

(oral hygiene reinforcement, scaling/root debridement) is provided first. For deep intrabony defects, regenerative therapy is introduced. Once stability is achieved, light, controlled orthodontic forces are applied with continuous monitoring. If inflammation recurs (FMBS >15% or BOP >10% of sites), orthodontic forces are reduced or paused, and oral hygiene is reinforced before resuming.

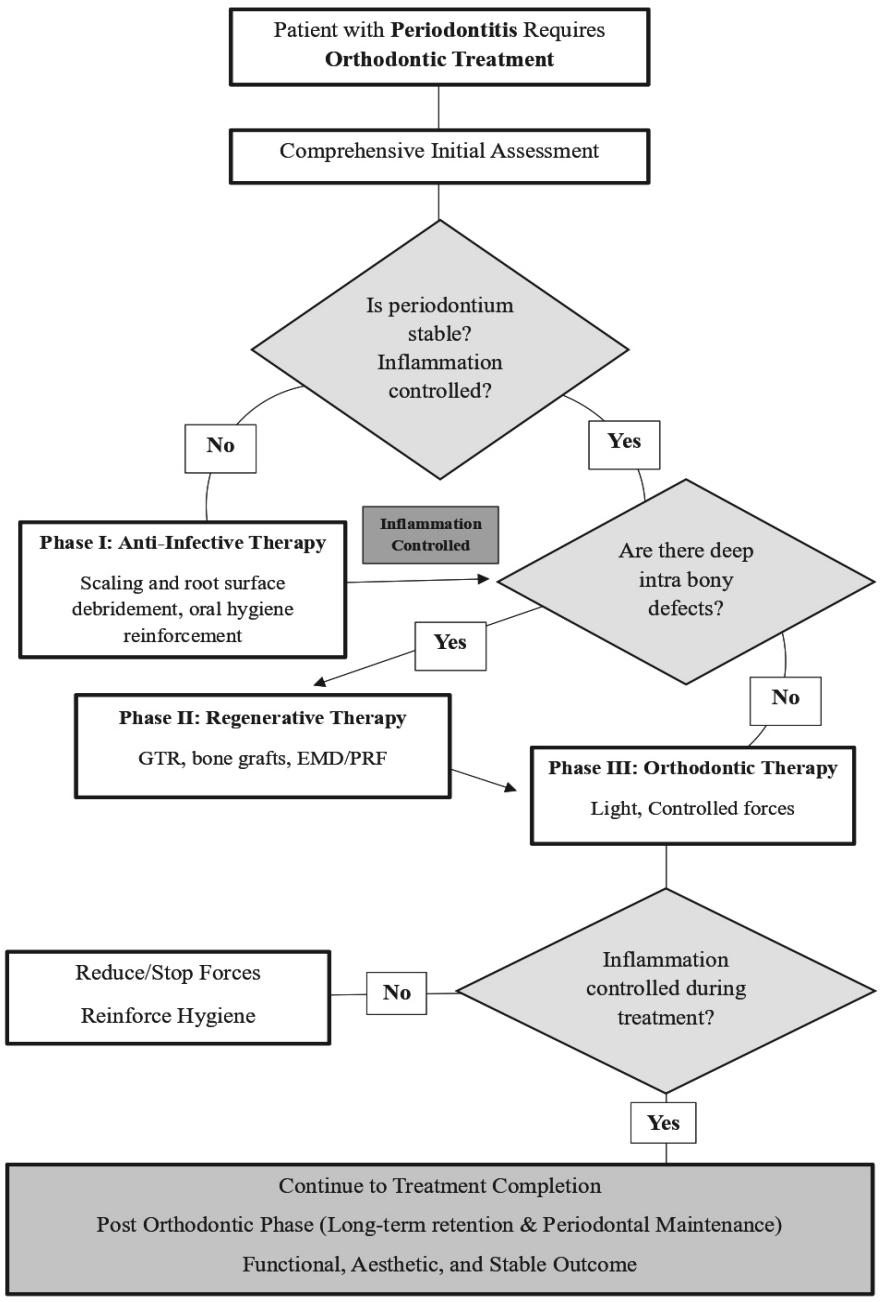

Fig. 2 Clinical decision-making framework for orthodontic therapy in patients with periodontitis.

CONCLUSION

Orthodontic treatment in periodontally compromised patients is biologically feasible but requires strict control of pre-existing inflammation. Successful outcomes depend on three key principles: first, achieving documented periodontal stability before force application; second, applying light, controlled forces with continuous monitoring; and third, integrating regenerative periodontal therapy for intrabony defects when indicated.

When these principles are followed, orthodontic tooth movement can be performed without exacerbating periodontal destruction and may even improve periodontal parameters. Future research should focus on individualised treatment protocols based on patients' inflammatory and bone metabolic profiles, as well as standardised outcome measures to enable stronger evidence synthesis.

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