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Original Article

Plaque Control, Periodontal Inflammation, and Orthodontic Treatment Stability: A Narrative Review

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ABSTRACT

Orthodontic treatment aims to achieve optimal dental alignment and occlusion, yet its success is intricately linked to periodontal health. Plaque accumulation, exacerbated by fixed appliances, can lead to gingival inflammation, periodontal attachment loss, and potential compromise of treatment stability. This narrative review synthesizes recent literature on the interplay between plaque control, periodontal inflammation, and the long-term stability of orthodontic outcomes. Drawing from peer-reviewed studies published between 2020 and 2025, we explore how orthodontic appliances influence microbial biofilms, inflammatory responses, and periodontal tissues. Key findings indicate that inadequate plaque control during treatment heightens the risk of gingivitis and periodontitis, which may contribute to post-treatment relapse through altered bone remodeling and soft tissue changes. Effective interventions, including powered toothbrushes, antimicrobial mouthrinses like chlorhexidine, and adjunctive tools such as water irrigators, have demonstrated superior plaque reduction and improved gingival health compared to conventional methods. Comparative analyses reveal that clear aligners may offer better periodontal outcomes than fixed appliances due to easier hygiene maintenance. Furthermore, periodontal inflammation during active orthodontics can impair tooth movement efficiency and increase susceptibility to root resorption, ultimately affecting retention phase stability. The review underscores the necessity of integrated orthodontic-periodontal management, emphasizing patient education and regular monitoring to mitigate inflammation and enhance stability. Future directions include personalized microbiome-targeted therapies and advanced biomaterials for plaque-resistant appliances. By prioritizing plaque control, clinicians can optimize orthodontic stability while preserving periodontal integrity, leading to more predictable and sustainable results.

Keywords: Plaque control, Periodontal inflammation, Orthodontic treatment, Treatment stability, Gingival health, Microbial biofilm

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Introduction

Orthodontic treatment has undergone remarkable advancements in recent decades, with innovations in appliance design, materials, and biomechanical techniques allowing more precise and efficient correction of malocclusions. However, the use of orthodontic devices—particularly fixed appliances such as brackets, bands, and wires—introduces unique challenges to maintaining oral hygiene, often resulting in increased plaque accumulation and subsequent periodontal inflammation [1,2]. The integrity of periodontal tissues is essential for successful orthodontic therapy, as inflamed gingiva and compromised supporting structures can alter the

biomechanical responses to applied forces, potentially leading to adverse outcomes such as gingival hyperplasia, attachment loss, alveolar bone resorption, and external root resorption (ERR) [3,4]. Moreover, the long-term stability of orthodontic results—defined as the maintenance of corrected tooth positions without relapse—is heavily dependent on the health of the periodontal ligament (PDL) and alveolar bone, which can be adversely affected by unresolved inflammation [5,6].

The interplay between plaque control, periodontal health, and orthodontic outcomes has been a central focus of contemporary dental research [7, 8]. Dental plaque, a complex microbial biofilm, thrives in the retention niches created by brackets, wires, and other orthodontic components. Pathogenic species such as *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* proliferate in these microenvironments, fostering a pro-inflammatory milieu conducive to gingivitis and periodontitis [2,9]. Studies published between 2020 and 2025 have further elucidated the biochemical consequences of plaque accumulation, demonstrating elevated levels of inflammatory mediators in the gingival crevicular fluid (GCF), including interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), and prostaglandin E2 (PGE2) [4, 10]. These cytokines not only exacerbate local tissue breakdown but can also influence alveolar bone remodeling by promoting osteoclastogenesis, potentially affecting the rate and safety of orthodontic tooth movement [4, 10]. Inadequate plaque control has also been correlated with prolonged treatment duration, increased incidence of enamel demineralization, and the development of white spot lesions, which collectively compromise both periodontal and orthodontic outcomes [11, 12].

Conversely, appropriately managed orthodontic treatment may confer periodontal benefits. Correction of crowding and malalignment can improve access for oral hygiene, reducing plaque retention and fostering a healthier periodontal environment in the long term [5,13]. However, in patients with pre-existing periodontal compromise—such as reduced attachment levels, thin gingival phenotypes, or alveolar bone defects—orthodontic forces must be meticulously calibrated to prevent exacerbation of bone loss and gingival recession [3, 5]. Evidence from recent interdisciplinary studies indicates that combining periodontal therapy with orthodontic interventions, such as scaling, root planing, and regenerative procedures prior to tooth movement, enhances both treatment efficacy and long-term stability [5, 14]. This approach not only mitigates inflammatory sequelae but also optimizes biomechanical responses and minimizes complications such as ERR and relapse.

The objectives of this narrative review are threefold: first, to examine the mechanisms by which plaque accumulation and periodontal inflammation arise during orthodontic therapy, including microbial, immunologic, and biomechanical factors; second, to evaluate the impact of these factors on treatment stability, particularly in relation to relapse and post-treatment retention; and third, to discuss evidence-based strategies for plaque control and inflammation management, encompassing both behavioral interventions and clinical adjuncts, to optimize long-term orthodontic outcomes [15-20]. By synthesizing findings from 32 peer-reviewed articles published between 2020 and 2025, this review aims to provide clinicians with a comprehensive, evidence-based framework for integrating periodontal care into orthodontic protocols, thereby enhancing both patient safety and treatment success.

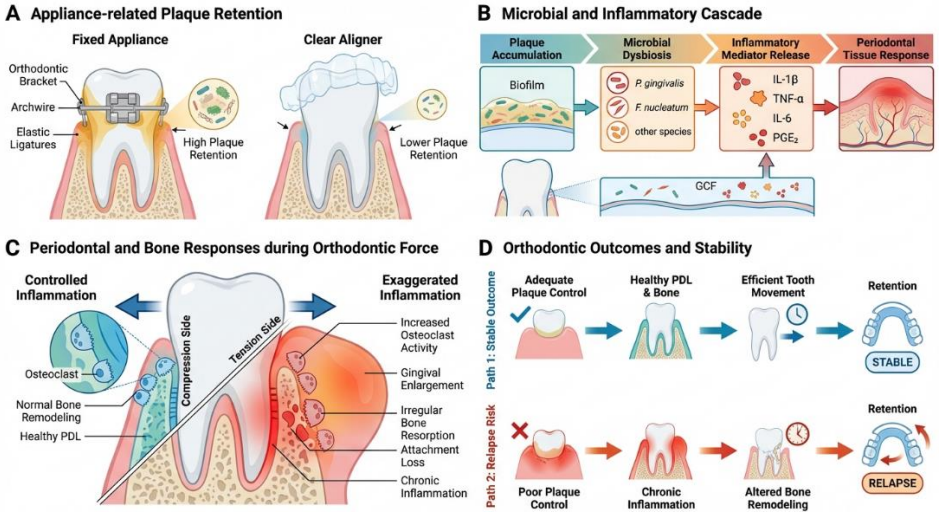

Figure 1. Conceptual relationship between orthodontic appliances, plaque accumulation, periodontal inflammation, and treatment stability

Plaque Accumulation and Microbial Changes in Orthodontic Patients

Orthodontic appliances profoundly influence the oral microbial ecosystem by creating complex surfaces and niches that hinder effective plaque removal. Fixed appliances, including brackets, archwires, and ligatures, generate retentive areas where biofilm can readily accumulate, resulting in a shift from a predominantly commensal microbiota to a more pathogenic profile [1, 2, 9]. This transition has been documented in longitudinal studies showing that plaque indices (PI) and gingival bleeding scores increase within weeks of appliance placement, reflecting early inflammatory responses [12, 21]. For instance, a randomized controlled trial demonstrated that adolescents fitted with fixed appliances exhibited significantly higher levels of *Streptococcus mutans* and *Lactobacillus* species—microorganisms closely associated with caries and gingivitis—compared to pre-treatment baselines [21].

Beyond cariogenic species, orthodontic treatment is linked to an enrichment of periodontal pathogens [22-29]. Metagenomic and 16S rRNA sequencing analyses reveal that subgingival plaque in patients with fixed appliances shows elevated levels of *Fusobacterium nucleatum*, *Prevotella intermedia*, and *Porphyromonas gingivalis*, which stimulate host inflammatory responses through cytokine release and matrix-degrading enzymes [9, 30]. A prospective cohort study correlated orthodontic appliance placement with increased pro-inflammatory cytokines, including interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α) in gingival crevicular fluid (GCF), establishing a mechanistic link between microbial dysbiosis and periodontal inflammation [4]. This inflammatory environment not only compromises gingival health but may also influence alveolar bone remodeling and tooth stability, highlighting the clinical significance of microbial management during orthodontic therapy [31-34].

Appliance type plays a crucial role in the magnitude of microbial changes. Removable clear aligners are associated with lower plaque accumulation and reduced periodontal pathogen loads compared to fixed appliances, likely due to their ease of removal and facilitation of thorough oral hygiene practices [35, 36]. Among fixed systems, self-ligating brackets have been proposed to minimize plaque retention compared to conventional ligature-based systems, though evidence is mixed and dependent on patient compliance [13]. Adjunctive measures, such as chlorhexidine rinses, have been shown to mitigate differences between metal and non-metal appliances by reducing microbial endotoxin activity and suppressing pathogenic colonization [37].

Individual patient factors further modulate plaque accumulation and microbial shifts [38-43]. Oral hygiene practices, salivary flow, diet, and systemic conditions such as diabetes or immunosuppression can exacerbate biofilm formation and inflammatory responses [1]. Despite these variations, untreated plaque accumulation inevitably progresses to gingivitis and, in susceptible individuals, periodontitis, emphasizing the need for proactive monitoring and targeted preventive strategies. Regular professional prophylaxis, reinforcement of oral hygiene education, and adjunctive chemical plaque control are essential components of orthodontic care to minimize microbial dysbiosis and its downstream effects on periodontal and orthodontic outcomes [1,12].

Mechanisms of Periodontal Inflammation During Orthodontic Therapy

Periodontal inflammation during orthodontic treatment arises from the complex interplay between mechanical forces applied to teeth and microbial challenges within the oral cavity. Orthodontic tooth movement relies on carefully controlled pressure and tension within the periodontal ligament (PDL) to stimulate alveolar bone remodeling, enabling teeth to achieve desired alignment [3, 4]. In healthy periodontium, this remodeling is balanced and largely aseptic; however, the presence of dental plaque and pathogenic biofilms shifts the response toward a pro-inflammatory state, exacerbating tissue breakdown and compromising periodontal homeostasis [1,9].

At the molecular level, active orthodontic therapy is associated with elevated levels of inflammatory mediators in the gingival crevicular fluid (GCF). Cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), along with matrix metalloproteinases (MMPs), are upregulated in response to both mechanical stress and microbial antigens [10, 44]. These molecules coordinate recruitment and activation of immune cells, including neutrophils, macrophages, and lymphocytes, which in turn release further proteolytic enzymes and reactive oxygen species that degrade connective tissue and alveolar bone. Clinically, this manifests as gingival enlargement, increased probing depths, and transient or sustained loss of attachment [3,4].

Recent studies have elucidated specific pathways linking mechanical load, microbial presence, and inflammatory amplification. For example, in patients undergoing orthodontic intrusion with reduced

periodontal support, higher baseline levels of IL-6 and TNF-α in GCF were predictive of greater attachment loss during treatment, highlighting their role as both biomarkers and mediators of tissue breakdown [3]. In periodontally compromised individuals, initiation of orthodontic forces without prior periodontal stabilization can exacerbate inflammation, resulting in accelerated probing depth increase and clinical attachment loss (CAL) compared to healthy controls [5]. Meta-analyses indicate that fixed appliances are particularly associated with elevated gingival index (GI) scores, with adolescents demonstrating more pronounced effects due to challenges in maintaining optimal oral hygiene [30].

Appliance-specific factors also influence inflammatory responses. Temporary anchorage devices (mini-screws) may induce localized gingival inflammation at insertion sites; however, longitudinal clinical assessments demonstrate minimal long-term impact on overall periodontal health when patients adhere to rigorous hygiene protocols [45]. Elastomeric ligatures have been shown to harbor greater bacterial loads than self-ligating systems, potentially amplifying local inflammatory responses and increasing risk of gingival overgrowth or bleeding [11, 46].

Adjunctive therapeutic approaches have been investigated to modulate inflammation. Topical agents such as 1% curcumin gel have been shown to reduce myeloperoxidase levels in GCF during initial orthodontic tooth movement, reflecting dampened neutrophilic activity [10]. Similarly, probiotics have been proposed to restore microbial balance, attenuate pro-inflammatory cytokine production, and support periodontal stability during orthodontic therapy [9].

Collectively, these findings underscore that periodontal inflammation during orthodontics is not merely a consequence of mechanical forces but is amplified by microbial dysbiosis and patient-specific factors. Uncontrolled inflammation can slow tooth movement, increase susceptibility to complications such as gingival recession, and jeopardize long-term treatment stability [4, 9]. Therefore, integrating rigorous plaque control, periodontal assessment, and targeted anti-inflammatory interventions is essential for optimizing outcomes, particularly in patients with pre-existing periodontal compromise.

Impact of Periodontal Inflammation on Orthodontic Treatment Outcomes and Stability

Periodontal inflammation exerts a profound influence on both the efficiency of orthodontic tooth movement and the long-term stability of treatment outcomes. Inflamed periodontal tissues exhibit altered biomechanical and biological properties, including disrupted collagen turnover, impaired fibroblast function, and diminished PDL viscoelasticity, which collectively impede the uniform transmission of orthodontic forces [3,6]. Such alterations may result in uneven tooth movement, requiring prolonged treatment durations or adjustments to force magnitude and direction. Recent longitudinal studies have demonstrated that gingival phenotype modulates these effects: patients with thicker biotypes exhibit greater resilience against inflammatory-mediated tissue breakdown and gingival recession, whereas thin phenotypes are more susceptible to compromised remodeling and subsequent attachment loss [6].

Beyond immediate movement, periodontal inflammation has significant implications for post-treatment stability. Active inflammation can induce asymmetric alveolar bone remodeling, generating areas of differential bone density and thickness that predispose to relapse once orthodontic forces are removed [5, 14]. This effect is particularly pronounced in patients with advanced periodontitis (stage III/IV), where baseline attachment loss and bone defects create a mechanically and biologically vulnerable environment. A narrative review focusing on supportive periodontal care (SPC) demonstrated that unresolved inflammation in these patients increased post-treatment relapse rates by as much as 30%, highlighting the critical importance of achieving periodontal stability prior to initiating orthodontic interventions [14].

Persistent microbial colonization further contributes to instability. Salivary and subgingival analyses indicate that residual pathogenic bacteria, including Fusobacterium nucleatum and Prevotella intermedia, can maintain a low-grade inflammatory state even after active treatment, subtly undermining bone and PDL integrity and promoting minor relapse [2, 9]. This underscores the necessity of ongoing maintenance therapy, including regular professional cleaning, plaque control reinforcement, and monitoring of cytokine markers, to sustain a healthy periodontal milieu post-orthodontics.

Appliance selection also modulates the impact of inflammation on stability. Clear aligner therapy, due to its removability and reduced plaque-retentive surfaces, has been associated with superior periodontal parameters, such as lower bleeding on probing and reduced probing depths, which in turn support more predictable long-term retention of tooth positions [35, 36]. Conversely, fixed appliances placed in sites with existing inflammation may exacerbate attachment loss and gingival recession, compromising both immediate

and post-treatment outcomes [5, 45]. Clinical studies have further identified specific inflammatory biomarkers predictive of stability: elevated interleukin-2 (IL-2) and interleukin-8 (IL-8) levels during treatment correlate with poorer retention, suggesting that monitoring cytokine profiles could inform individualized risk stratification and management strategies [4,10].

Overall, the evidence underscores that periodontal inflammation is not merely a transient concern during orthodontics but a critical determinant of treatment efficacy and longevity. Optimal outcomes require a multifaceted approach, integrating thorough periodontal assessment, phenotype evaluation, microbial control, judicious appliance selection, and targeted anti-inflammatory interventions. This strategy ensures that orthodontic forces are delivered to biologically stable tissues, minimizing relapse risk and maximizing the longevity of both alignment and occlusal harmony.

Strategies for Plaque Control and Inflammation Management in Orthodontics

Effective plaque control represents a cornerstone in minimizing periodontal inflammation and ensuring long-term stability during orthodontic treatment. Mechanical methods remain the foundation of oral hygiene, with a substantial body of evidence demonstrating the superiority of powered toothbrushes over manual alternatives. Meta-analyses indicate that in patients with fixed appliances, powered brushes reduce plaque indices (PI) by 20–30% and gingival inflammation markers, such as bleeding on probing (BOP), more effectively than manual brushing [30, 47]. Orthodontic-specific brushes, designed to access beneath brackets and around archwires, further enhance cleaning efficacy, contributing to measurable reductions in gingival index (GI) scores and probing depths (PD) over the course of treatment [12, 30]. Correct brushing technique, including angulation and duration, is critical, as improper use diminishes mechanical effectiveness despite high brush quality.

Chemical adjuncts serve as valuable complements to mechanical plaque control. Chlorhexidine gluconate mouthrinses have been extensively studied, demonstrating significant reductions in plaque accumulation, gingival inflammation, and microbial loads [11, 48, 49]. While long-term use may result in extrinsic tooth staining or altered taste perception, these effects are typically reversible and outweighed by the benefits in high-risk orthodontic patients. Emerging alternatives, such as probiotic toothpastes containing Lactobacillus or Bifidobacterium species, modulate oral microbiota by enhancing beneficial commensals and suppressing pathogenic species, thereby reducing gingival inflammation without contributing to antimicrobial resistance [44, 50]. Similarly, xylitol-impregnated brushes disrupt biofilm formation and acid production, offering additional preventive effects against plaque-related inflammation.

Adjunctive technologies, particularly water irrigators, are especially effective in areas inaccessible to conventional brushing or flossing. Systematic reviews demonstrate that pulsating water devices reduce plaque and bleeding indices more effectively than flossing alone in patients with fixed orthodontic appliances, facilitating biofilm disruption around brackets, under wires, and along the gingival margin [51, 52]. Integration of these devices into daily oral hygiene routines is recommended for patients with thin biotypes, crowded dentition, or pre-existing periodontal compromise.

Patient education and behavioral interventions are crucial for achieving compliance and long-term success. Motivational strategies, including the use of plaque-disclosing agents, personalized feedback, and digital reminders, significantly improve oral hygiene adherence, translating into lower PI and GI scores throughout treatment [53]. Clinicians should tailor educational approaches to patient age, motivation, and dexterity, ensuring that instructions are practical, comprehensible, and reinforced regularly.

In periodontally compromised patients, a comprehensive, integrated approach is essential. Combining routine scaling and root planing with mechanical and chemical plaque control not only mitigates inflammation but also enhances the periodontal environment for safe orthodontic movement [5, 14]. Regular periodontal assessments—every 1–3 months depending on severity—allow early detection of inflammation, enabling timely intervention to prevent attachment loss and support treatment stability [6, 14]. Evidence suggests that structured maintenance programs, when combined with patient adherence, reduce the incidence of gingival overgrowth, recession, and even external root resorption, underscoring the interdependence of periodontal health and orthodontic outcomes.

Effective management of plaque and inflammation during orthodontic therapy requires a multifaceted strategy integrating mechanical cleaning, chemical adjuncts, advanced irrigation tools, patient education, and rigorous professional monitoring. This integrated approach not only preserves periodontal health but also contributes to optimal treatment efficiency and long-

term stability, particularly in patients with pre-existing periodontal compromise.

Emerging Approaches and Considerations for Future Practice

Recent innovations focus on biofilm-resistant materials and microbiome modulation. Antimicrobial coatings on appliances reduce pathogen adhesion, preserving periodontal health [2, 46]. Personalized strategies, informed by genetic and microbial profiling, may tailor interventions [9,10].

In summary, proactive plaque control minimizes inflammation, safeguarding orthodontic stability.

Discussion

The intricate relationship between plaque control, periodontal inflammation, and orthodontic treatment stability represents a critical area of concern in contemporary orthodontics. As elucidated in the main text, orthodontic appliances, particularly fixed ones, predispose patients to increased plaque accumulation due to their architectural complexity, which impedes effective oral hygiene [1, 35, 47]. This predisposition fosters microbial dysbiosis, elevating the prevalence of periodontal pathogens and triggering inflammatory responses that can compromise both the efficiency of tooth movement and long-term stability [3, 4, 9]. The synthesis of recent evidence from 2020 to 2025 underscores that while orthodontic therapy can enhance periodontal health through improved tooth alignment, inadequate plaque management often exacerbates gingivitis and periodontitis, leading to potential relapse [5, 6, 11, 54].

One key insight from the reviewed literature is the differential impact of appliance types on periodontal outcomes. Fixed appliances consistently demonstrate higher plaque indices and gingival inflammation compared to removable clear aligners, as the latter allow for unhindered brushing and flossing [35, 36]. For instance, studies comparing microbial profiles reveal reduced pathogen loads and cytokine levels with aligners, suggesting a lower inflammatory burden that may contribute to greater post-treatment stability [4, 35, 10]. However, even with aligners, patient compliance remains pivotal; non-adherence can still result in biofilm buildup, albeit to a lesser extent than with brackets [30, 35]. This highlights the need for tailored appliance selection based on individual periodontal risk profiles, such as in patients with pre-existing attachment loss, where fixed appliances might accelerate bone resorption if inflammation is not controlled [4, 11].

The mechanisms linking inflammation to treatment instability are multifaceted, involving altered bone remodeling and soft tissue adaptations. Periodontal inflammation elevates pro-inflammatory cytokines like IL-1β and TNF-α, which disrupt osteoclast-osteoblast balance, potentially leading to uneven alveolar bone density and increased relapse propensity [3, 4, 10]. Longitudinal data indicate that unresolved gingivitis during active treatment correlates with higher relapse rates, particularly in the lower anterior region, where crowding relapse is common [5, 6, 50]. Moreover, inflammation may exacerbate external apical root resorption, further compromising periodontal support and stability [3, 52]. These findings align with biomechanical models suggesting that inflamed periodontium exhibits reduced resistance to orthodontic forces, prolonging treatment and heightening post-retention shifts [3, 4].

Strategies for plaque control emerge as a cornerstone for mitigating these risks. Mechanical interventions, such as powered toothbrushes and orthodontic-specific brushes, have proven superior in reducing plaque and gingival indices, with meta-analyses reporting 20-30% greater efficacy over manual methods [30, 45, 47, 51]. Chemical adjuncts like chlorhexidine mouthrinses offer additional benefits, significantly lowering bacterial counts and inflammation, though concerns about staining and taste alterations necessitate judicious use [3, 11, 48, 49, 55]. Emerging tools, including water irrigators and probiotic lozenges, provide promising alternatives, demonstrating comparable or enhanced plaque removal in randomized trials [1, 21, 49, 52]. Patient education and motivational techniques, such as plaque disclosure, further amplify compliance, leading to sustained improvements in periodontal health [37, 48, 53].

However, the literature reveals several limitations that warrant cautious interpretation. Many studies are short-term, focusing on active treatment phases rather than long-term stability, with follow-up periods rarely exceeding 12-24 months [5, 6, 11]. This temporal constraint limits insights into chronic inflammatory effects on relapse. Additionally, heterogeneity in methodologies—ranging from plaque indices to microbiome analyses—complicates direct comparisons, as seen in mixed results for self-ligating versus conventional brackets [13, 46]. Sample sizes are often modest, and few account for confounding factors like smoking or systemic conditions, which could influence inflammatory responses [4, 5]. Moreover, while adolescents predominate in trials due to higher orthodontic prevalence, evidence for adults with

compromised periodontium is sparse, despite their elevated risk [11, 54].

Clinically, these findings advocate for an interdisciplinary approach, integrating periodontal therapy with orthodontics [56-59]. Pre-treatment periodontal stabilization, including scaling and root planing, is essential for high-risk patients to minimize inflammation and enhance stability [5, 11, 54]. Regular monitoring via gingival crevicular fluid analysis or digital plaque imaging could enable early intervention, potentially reducing relapse [12, 46]. The role of supportive periodontal care during retention phases is particularly emphasized, with evidence suggesting that ongoing hygiene protocols preserve treatment outcomes [21, 54]. Economically, effective plaque control may reduce treatment costs by shortening duration and averting complications like white spot lesions [12, 47].

From a broader perspective, the reviewed evidence challenges traditional orthodontic paradigms by prioritizing periodontal health as a predictor of success. In an era of personalized medicine, genetic and microbial profiling could refine risk assessments, guiding customized interventions [9, 10, 60-67]. However, ethical considerations arise in balancing aesthetic demands with periodontal integrity, especially in aggressive treatments for periodontally compromised individuals [4, 11]. Future research should address these gaps through large-scale, long-term randomized controlled trials incorporating advanced biomarkers and digital tools for objective monitoring.

In summary, the discourse on plaque control and inflammation in orthodontics reveals a dynamic interplay that profoundly influences treatment stability. By adopting evidence-based hygiene strategies and fostering periodontal-orthodontic synergy, clinicians can mitigate risks, optimizing outcomes in an increasingly diverse patient population.

Conclusions

In conclusion, this narrative review highlights the pivotal role of effective plaque control in managing periodontal inflammation and ensuring orthodontic treatment stability. Orthodontic appliances inherently promote plaque accumulation, leading to microbial shifts and inflammatory responses that can impair tooth movement and precipitate relapse [1, 3, 4, 35]. Key evidence from 2020-2025 demonstrates that interventions like powered toothbrushes, chlorhexidine rinses, and water irrigators significantly reduce plaque and inflammation, with clear aligners offering advantages over fixed appliances in maintaining periodontal health [1, 35, 47, 49]. Ultimately, integrated management strategies, emphasizing patient education and interdisciplinary care, are essential for achieving sustainable orthodontic results while preserving periodontal integrity [5, 11, 37, 48, 54].

Looking ahead, future research should prioritize longitudinal studies to elucidate long-term impacts of inflammation on stability, incorporating diverse populations and advanced technologies like AI-driven plaque detection [12, 46]. Personalized therapies targeting the oral microbiome, such as probiotic formulations or biofilm-resistant biomaterials, hold promise for revolutionizing plaque control [9, 49, 50, 52]. Additionally, exploring the efficacy of telehealth for hygiene monitoring could enhance compliance, particularly in underserved areas [48, 53]. By addressing these avenues, orthodontics can evolve toward more predictable, patient-centered approaches that minimize relapse and maximize periodontal health.

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