American Journal of
Oral Medicine and Radiology

ISSN - 2394-7721

www.mcmed.us/journal/ajomr

Review Article

THE ROLE OF ANTI-INFLAMMATORY DRUGS IN PERIODONTAL DISEASE TREATMENT: MECHANISMS AND CLINICAL EFFICACY

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ABSTRACT

Periodontal disease is a prevalent chronic inflammatory condition that affects the supporting structures of the teeth and is primarily driven by an inflammatory response to bacterial plaque accumulation. Anti-inflammatory drugs play a critical role in the treatment of periodontal disease by modulating the inflammatory pathways that contribute to tissue destruction and disease progression. These drugs, including nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and specific cytokine inhibitors, are used adjunctively with conventional therapies, such as scaling and root planing, to improve clinical outcomes and reduce inflammation, pain, and tissue loss. The mechanisms through which anti-inflammatory drugs exert their therapeutic effects include the inhibition of prostaglandin synthesis, modulation of matrix metalloproteinase activity, and suppression of pro-inflammatory cytokine release. The clinical efficacy of these drugs in periodontal therapy has been extensively studied, with NSAIDs, in particular, showing promise in reducing periodontal pocket depth, bleeding on probing, and clinical attachment loss. However, their use is often limited by side effects, such as gastrointestinal irritation, renal toxicity, and impaired wound healing. Advances in the development of more targeted anti-inflammatory therapies, such as monoclonal antibodies or selective inhibitors of specific inflammatory mediators, offer the potential for improved efficacy and reduced systemic side effects. This review discusses the role of anti-inflammatory drugs in the treatment of periodontal disease, explores the mechanisms of their action, and evaluates their clinical efficacy and limitations in periodontal therapy. Future directions for research include the development of novel anti-inflammatory agents with enhanced specificity and fewer adverse effects, as well as strategies to integrate these therapies into personalized treatment regimens for periodontal disease.

Keywords: - Periodontal disease, anti-inflammatory drugs, NSAIDs, cytokine inhibitors, scaling and root planing.

Access this article online	
Home page: http://www.mcmed.us/journal/ajomr	Quick Response code

Received:03.02.2026	**Revised**:02.03.2026	**Accepted**:06.04.2026

INTRODUCTION

Periodontal disease is a complex and multifactorial condition characterized by inflammation of the supporting structures of the teeth, including the gums, periodontal ligaments, and alveolar bone. It is primarily caused by the accumulation of bacterial plaque, which triggers an immune response that leads to chronic inflammation, tissue destruction, and, if left untreated, tooth loss. The role of inflammation in the pathogenesis of periodontal disease is well established, with a host of pro-inflammatory mediators, such as cytokines, prostaglandins, and matrix metalloproteinase, contributing to the breakdown of the periodontal tissue.

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Anti-inflammatory drugs are integral to the management of periodontal disease, as they help modulate these inflammatory pathways and reduce the destructive effects of the immune response. Nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and more recently, selective cytokine inhibitors have been used as adjunctive therapies to conventional mechanical treatments, such as scaling and root planing, to improve clinical outcomes[1]. NSAIDs, for example, exert their therapeutic effects by inhibiting cyclooxygenase (COX) enzymes, which are responsible for the synthesis of prostaglandins, key mediators of inflammation and pain. By reducing prostaglandin levels, NSAIDs help decrease periodontal tissue inflammation, alleviate pain, and improve clinical parameters such as pocket depth, bleeding on probing, and clinical attachment loss. Corticosteroids, which are potent anti-inflammatory agents, can also be used to control severe inflammation in periodontal disease, although their long-term use is limited by potential side effects such as impaired wound healing and increased risk of infection. Recent advances in the understanding of the molecular mechanisms underlying periodontal inflammation have led to the development of more targeted therapies, including biologics and cytokine inhibitors, which aim to specifically modulate inflammatory pathways without the broader systemic effects of traditional anti-inflammatory drugs[2]. These therapies hold promise for improving the clinical efficacy of periodontal disease treatment while minimizing adverse effects. Despite the potential benefits of anti-inflammatory drugs in periodontal therapy, their use must be carefully managed, as side effects such as gastrointestinal irritation, renal toxicity, and delayed tissue healing can limit their effectiveness. The future of periodontal therapy lies in the development of more specific and effective anti-inflammatory treatments that can be personalized to individual patients, offering enhanced therapeutic outcomes with reduced risks.

Periodontal Disease and the Need for Anti-inflammatory Interventions

Periodontal disease is a chronic inflammatory condition that affects the supporting structures of the teeth, including the gums, periodontal ligaments, and alveolar bone. It is primarily caused by the accumulation of bacterial plaque, which triggers an immune response leading to chronic inflammation, tissue destruction, and, if left untreated, tooth loss. The primary need for anti-inflammatory interventions in periodontal disease arises from the role of inflammation in driving the pathogenesis of the disease. The inflammatory response to bacterial biofilm formation in the periodontal pocket activates the host immune system, leading to the release of a wide range of inflammatory mediators such as cytokines, prostaglandins, and matrix metalloproteinases. These mediators contribute to the breakdown of the periodontal tissue, resulting in tissue destruction, pocket formation, and bone loss. While bacterial control through mechanical debridement, such as scaling and root planing, is a cornerstone of treatment, these measures alone are often insufficient to resolve the inflammation and tissue damage.[3] Anti-inflammatory drugs, therefore, become a critical adjunctive therapy, as they help to modulate the excessive inflammatory response, reduce pain, and prevent further tissue destruction. Nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and cytokine inhibitors are among the pharmacological interventions used to manage inflammation in periodontal disease. NSAIDs, for instance, inhibit cyclooxygenase enzymes (COX-1 and COX-2), reducing prostaglandin production, which plays a central role in inflammation and pain. Corticosteroids, while potent anti-inflammatory agents, have limitations due to their side effects, especially with long-term use. Recent advancements have focused on developing more targeted therapies, such as biologics and selective cytokine inhibitors, that can block specific inflammatory mediators without the broader systemic effects of conventional drugs. These targeted therapies have the potential to provide more effective management of chronic inflammation in periodontal disease, reduce the progression of tissue damage, and improve clinical outcomes. However, the use of anti-inflammatory drugs in periodontal therapy requires careful management due to the risk of adverse effects and interactions with other medications. Nonetheless, anti-inflammatory interventions remain essential in the comprehensive management of periodontal disease, providing a way to address the inflammation that drives tissue destruction and improving patient outcomes.

Overview of Periodontal Disease: Etiology and Pathophysiology

Periodontal disease is a prevalent chronic inflammatory condition that affects the supporting structures of the teeth, including the gums, periodontal ligaments, and alveolar bone. The disease develops as a result of a complex interaction between bacterial plaque accumulation, the host immune response, and environmental and genetic factors. The etiology of periodontal disease primarily involves the formation of dental plaque, a biofilm composed of bacteria, on the surface of the teeth. These bacteria produce toxins and other virulence factors that trigger the body's immune response, initiating an inflammatory cascade in the gingiva and surrounding tissues. The pathophysiology of periodontal disease is marked by an imbalance between the host's immune response and the bacterial load, leading to chronic inflammation.[4] The initial stage, gingivitis, is characterized by redness, swelling, and

bleeding of the gums due to inflammation, but it is usually reversible with proper oral hygiene. If left untreated, gingivitis can progress to periodontitis, where the inflammation extends deeper into the supporting tissues of the teeth, including the periodontal ligament and alveolar bone. In periodontitis, the chronic inflammatory response causes tissue destruction, including loss of attachment between the tooth and the surrounding bone, leading to periodontal pockets, bone resorption, and, eventually, tooth mobility and loss. Key inflammatory mediators, such as cytokines, prostaglandins, and matrix metalloproteinase, are released during this process, contributing to the breakdown of periodontal tissues. The immune response in periodontal disease involves both innate and adaptive components, with neutrophils and macrophages playing prominent roles in the early stages of inflammation, while T cells and B cells become increasingly involved as the disease progresses. Environmental factors such as smoking, poor oral hygiene, and diabetes, as well as genetic predisposition, can exacerbate periodontal disease progression[4]. Smoking, for example, impairs immune function and accelerates tissue destruction, while diabetes affects blood sugar regulation and compromises tissue healing. The pathophysiology of periodontal disease underscores the need for effective management strategies that not only control the bacterial infection but also modulate the host's inflammatory response to prevent tissue destruction and preserve oral health. Understanding the complex interplay between bacteria and host immunity is key to developing more effective treatments for periodontal disease.

The Impact of Inflammatory Mediators on Tissue Destruction

Inflammatory mediators play a central role in the pathogenesis of periodontal disease, directly contributing to tissue destruction and the progression of the condition. The immune response triggered by bacterial plaque accumulation in the periodontal pocket leads to the activation of various inflammatory pathways, resulting in the release of a range of pro-inflammatory molecules. These mediators, including cytokines, prostaglandins, and matrix metalloproteinase, orchestrate the inflammatory response that ultimately leads to the breakdown of periodontal tissues. Cytokines, such as interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6), are key players in periodontal inflammation. These cytokines stimulate the production of other inflammatory mediators and recruit immune cells, including neutrophils, macrophages, and lymphocytes, to the site of infection[5]. They also promote the release of prostaglandins, particularly prostaglandin E2 (PGE2), which contributes to pain, swelling, and further tissue destruction by increasing vascular permeability and stimulating the activity of osteoclasts—cells responsible for bone resorption. Prostaglandins play a pivotal role in bone loss in periodontal disease, as they stimulate the resorption of alveolar bone and inhibit bone formation. Matrix metalloproteinase (MMPs), a group of enzymes involved in the degradation of extracellular matrix components such as collagen, are also significantly elevated in periodontal disease. MMPs break down the structural integrity of the periodontal tissues, leading to the breakdown of connective tissue and the loss of attachment between the tooth and surrounding bone. The imbalance between tissue destruction and tissue repair in periodontal disease is a key factor in disease progression[6]. In healthy individuals, the inflammatory response is tightly regulated to resolve the infection and repair damaged tissues; however, in periodontal disease, the persistent presence of bacterial biofilm and chronic inflammation leads to uncontrolled tissue destruction. Additionally, systemic conditions, such as diabetes and obesity, can exacerbate the inflammatory response, further promoting tissue breakdown. Inflammatory mediators not only contribute to the destruction of soft tissues but also affect the surrounding bone, leading to alveolar bone loss and eventual tooth mobility. As such, targeting these inflammatory mediators has become a crucial focus of periodontal disease therapy, with the goal of reducing inflammation, preventing tissue destruction, and promoting tissue repair. Anti-inflammatory drugs and biologic therapies targeting specific inflammatory pathways have shown promise in improving clinical outcomes by modulating these mediators and preserving the integrity of periodontal tissues.

Pharmacological Mechanisms of Anti-inflammatory Drugs

Anti-inflammatory drugs play a critical role in managing periodontal disease by targeting the underlying inflammatory processes that drive tissue destruction and disease progression. The pharmacological mechanisms of these drugs are centered around their ability to modulate the inflammatory response and inhibit the release of pro-inflammatory mediators, thereby reducing tissue damage. Nonsteroidal anti-inflammatory drugs (NSAIDs) are among the most commonly used pharmacological agents in the treatment of periodontal disease. Their primary mechanism of action involves the inhibition of cyclooxygenase (COX) enzymes, particularly COX-2, which are responsible for the synthesis of prostaglandins—lipid compounds that mediate inflammation, pain, and fever. By inhibiting COX-2, NSAIDs reduce the production of prostaglandins, thereby alleviating inflammation and pain while promoting tissue healing. The reduction of prostaglandin levels is particularly important in periodontal disease, where

prostaglandins, especially prostaglandin E2 (PGE2), are involved in bone resorption and the destruction of periodontal tissues. Corticosteroids, another class of anti-inflammatory drugs, exert their effects by suppressing the immune response through the inhibition of inflammatory cytokine production, such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6). These cytokines play a pivotal role in the activation of immune cells, such as neutrophils and macrophages, which contribute to tissue destruction in periodontal disease. Corticosteroids also inhibit the activity of matrix metalloproteinase (MMPs), enzymes responsible for the breakdown of extracellular matrix proteins, such as collagen, in periodontal tissues. By reducing MMP activity, corticosteroids help preserve the structural integrity of the periodontal ligament and alveolar bone. Selective cytokine inhibitors, such as monoclonal antibodies targeting TNF-α or IL-1, offer a more targeted approach to managing inflammation in periodontal disease. These biologic agents bind to and neutralize specific inflammatory cytokines, preventing them from binding to their receptors on immune cells and tissue-resident cells. By blocking the activity of these key inflammatory mediators, cytokine inhibitors reduce the chronic inflammation associated with periodontal disease and prevent further tissue destruction. While anti-inflammatory drugs offer significant benefits in controlling inflammation and reducing symptoms, their use must be carefully managed to avoid potential side effects, such as gastrointestinal irritation, renal toxicity, and delayed tissue healing. Therefore, it is important to integrate these therapies with conventional periodontal treatments, such as scaling and root planning, to optimize clinical outcomes and manage periodontal disease effectively[7].

Comparative Effectiveness of Topical vs. Systemic Treatments

Topical and systemic treatments represent two distinct approaches to managing periodontal disease, each with its own advantages and limitations. Topical treatments involve the direct application of therapeutic agents to the affected oral tissues, ensuring that the drug is concentrated at the site of infection and inflammation. This approach is particularly beneficial for localized conditions, such as gingivitis, oral ulcers, or microsites, where targeted drug delivery can reduce inflammation and promote healing without affecting the rest of the body. One of the primary advantages of topical treatments is that they minimize systemic exposure to the drug, thereby reducing the risk of side effects. Common topical agents used in periodontal therapy include antimicrobial gels, corticosteroid creams, and anti-inflammatory formulations that can be directly applied to periodontal pockets or inflamed mucosal tissues. These treatments offer high local efficacy, but their primary limitation is their relatively short duration of action[8]. The drug may be rapidly washed away by saliva or diluted, reducing its effectiveness. In contrast, systemic treatments, such as oral medications or injectable biologics, provide broader therapeutic effects by reaching all areas of the body via the bloodstream. Systemic drugs, such as oral NSAIDs, corticosteroids, and antibiotics, can address more widespread inflammation and infection, making them suitable for conditions that affect multiple regions of the oral cavity. However, systemic treatments are often associated with more significant side effects, including gastrointestinal irritation, renal toxicity, and immune suppression, particularly with long-term use. The effectiveness of systemic treatments is also influenced by factors such as drug absorption, metabolism, and clearance, which can vary between individuals. Combining both topical and systemic treatments may offer a more comprehensive approach to periodontal disease management, particularly in patients with chronic or severe forms of the condition. For instance, systemic antibiotics can control bacterial infection, while topical anti-inflammatory agents can target localized inflammation, enhancing the overall therapeutic outcome[9]. Ultimately, the choice between topical and systemic treatments depends on the severity of the disease, the specific condition being treated, and the patient's overall health profile. Both approaches have their place in periodontal therapy, and their optimal use requires careful consideration of their respective advantages, limitations, and potential side effects.

Benefits of Combination Therapy in Chronic Periodontitis Management

Combination therapy in chronic periodontitis management offers a synergistic approach that maximizes therapeutic outcomes by addressing the complex and multifactorial nature of the disease. Chronic periodontitis is a progressive inflammatory condition that involves both microbial infection and host immune response, leading to the destruction of periodontal tissues and bone loss. As such, a single therapeutic modality may not provide sufficient control over both the bacterial infection and the inflammation that drives tissue destruction. Combination therapy, which typically involves the use of both antimicrobial and anti-inflammatory agents, targets these two key components of the disease simultaneously, offering enhanced efficacy compared to monotherapy. The use of systemic antibiotics, such as doxycycline or metronidazole, alongside topical anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs) or corticosteroids, can help control bacterial infection while also reducing the inflammatory response. Antibiotics are effective in targeting the microbial biofilm that forms in

periodontal pockets, while anti-inflammatory drugs help reduce the chronic inflammation that contributes to tissue destruction[10]. Additionally, combination therapy can involve adjunctive therapies such as scaling and root planning, which physically remove plaque and tartar from the tooth surface, allowing for more effective drug penetration and enhancing the overall treatment outcome.However, it is essential to consider potential drug interactions, side effects, and the individual patient's needs when selecting combination therapies.[11] The development of personalized combination treatment plans based on the severity of the disease and the patient's response to therapy holds the potential for even more effective management of chronic periodontitis.

Adverse Effects and Safety Considerations of Anti-inflammatory Drugs

The use of anti-inflammatory drugs in the treatment of periodontal disease has become a common practice due to their ability to modulate the inflammatory processes that drive tissue destruction. However, like all pharmacological treatments, anti-inflammatory drugs come with potential adverse effects and safety considerations that must be carefully managed to ensure patient safety and therapeutic efficacy. Nonsteroidal anti-inflammatory drugs (NSAIDs), commonly used in periodontal therapy, work by inhibiting cyclooxygenase (COX) enzymes, thereby reducing the production of prostaglandins, which are key mediators of inflammation and pain. While NSAIDs can be highly effective in reducing inflammation and improving clinical outcomes, they are associated with a range of side effects, particularly with long-term use. Gastrointestinal irritation, including ulcers, bleeding, and dyspepsia, is one of the most common side effects of NSAIDs. This occurs due to the inhibition of COX-1, which plays a protective role in the stomach lining.[9] Renal toxicity is another significant concern, as NSAIDs can impair kidney function by inhibiting COX-2 and disrupting renal blood flow. In patients with preexisting renal conditions, NSAID use must be closely monitored to avoid exacerbating kidney damage. Corticosteroids, another class of anti-inflammatory drugs, are potent anti-inflammatory agents that suppress the immune system and reduce inflammation. While they are effective in managing severe inflammation in periodontal disease, corticosteroids come with a host of potential side effects, including weight gain, hyperglycemia, osteoporosis, and increased susceptibility to infections. Additionally, corticosteroids can impair wound healing, which is a particular concern in periodontal therapy, where tissue regeneration and healing are crucial to the success of treatment. The use of biologic agents, such as monoclonal antibodies targeting specific cytokines like TNF-α or IL-1, has shown promise in modulating inflammation more selectively. However, biologics are also associated with their own set of risks, including immune suppression, increased risk of infections, and potential allergic reactions[12].

Addressing Potential Drug Interactions in Periodontal Disease Treatment

Drug interactions present a significant challenge in the treatment of periodontal disease, particularly when anti-inflammatory drugs, antibiotics, and other medications are used in combination. The concurrent use of multiple medications increases the risk of pharmacokinetic and pharmacodynamics interactions, which can impact the efficacy of treatment, exacerbate side effects, or alter the absorption, distribution, metabolism, and excretion of drugs. Nonsteroidal anti-inflammatory drugs (NSAIDs), commonly prescribed to reduce inflammation in periodontal disease, are known to interact with various medications, including anticoagulants, antihypertensive drugs, and corticosteroids. For example, the combination of NSAIDs and anticoagulants, such as warfarin, can increase the risk of bleeding, as both drugs affect platelet function and coagulation pathways. Similarly, NSAIDs can interfere with the efficacy of antihypertensive medications, including diuretics and ACE inhibitors, by affecting renal function and fluid balance, leading to elevated blood pressure[13]. Corticosteroids, another common anti-inflammatory treatment, can also interact with several drugs, including antifungal agents, oral hypoglycemic, and vaccines. These interactions can alter the metabolism of corticosteroids, increasing the risk of side effects such as hyperglycemia, delayed wound healing, and immune suppression. Antibiotics used in periodontal therapy, such as doxycycline and metronidazole, can also have significant interactions with other medications. For example, metronidazole is known to interact with alcohol, leading to a disulfiram-like reaction that causes nausea, vomiting, and flushing. Additionally, certain antibiotics may interact with oral contraceptives, reducing their effectiveness and increasing the risk of unintended pregnancy. To mitigate these risks, healthcare providers must conduct a thorough review of a patient's medication history before prescribing treatments for periodontal disease. This review should include consideration of potential drug-drug interactions, especially when using combination therapies[12].

Biomarkers for Predicting Treatment Outcomes and Side Effects

Biomarkers play an increasingly important role in predicting treatment outcomes and side effects in the management of periodontal disease, particularly in the context of anti-inflammatory drug therapies. These

biomarkers, which can be measured in the blood, saliva, or gingival reticular fluid, provide valuable information about the disease process, the patient's response to treatment, and the potential for adverse effects. In periodontal disease, key biomarkers of inflammation, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), have been identified as indicators of disease severity and progression. Monitoring the levels of these biomarkers during treatment can help clinicians assess the effectiveness of anti-inflammatory drugs, including NSAIDs and corticosteroids, and adjust the therapy as needed to achieve optimal outcomes[14]. For example, a significant reduction in the levels of CRP and IL-6 following treatment with anti-inflammatory agents may indicate a positive response to therapy, while persistently elevated levels could suggest that the inflammation is not adequately controlled. Additionally, biomarkers can help predict the potential for side effects associated with anti-inflammatory drugs. For instance, elevated levels of certain biomarkers, such as liver enzymes or creatinine, may indicate a risk of hepatotoxicity or nephrotoxicity, prompting the clinician to modify the treatment regimen. Biomarkers can also provide insight into the patient's individual risk for systemic effects, such as cardiovascular disease or diabetes, which can be exacerbated by long-term use of anti-inflammatory drugs. In the case of biologic therapies, such as monoclonal antibodies targeting specific inflammatory cytokines, biomarkers can be used to monitor the effectiveness of treatment and identify patients who may benefit most from these therapies. Furthermore, genetic biomarkers are being explored as potential predictors of treatment response, as certain genetic profiles may influence how a patient metabolizes or responds to specific drugs. For example, polymorphisms in the genes encoding cytokines or drug-metabolizing enzymes could provide important information about a patient's likelihood of responding to specific anti-inflammatory treatments.[15]

Advancements in Precision Medicine for Periodontal Inflammation Management

Advancements in precision medicine offer exciting possibilities for the management of periodontal inflammation, allowing for more individualized and effective treatment strategies tailored to the unique genetic, environmental, and biological characteristics of each patient. Precision medicine involves the use of molecular and genetic information to guide treatment decisions, optimizing the choice of therapy based on a patient's specific needs and predispositions. In the context of periodontal disease, precision medicine aims to address the underlying inflammatory processes that drive tissue destruction and disease progression. One key aspect of precision medicine is the identification of genetic and molecular biomarkers that can predict how a patient will respond to specific anti-inflammatory treatments. For instance, genetic polymorphisms in cytokine genes or genes involved in immune response pathways can influence the severity of inflammation and the patient's response to therapies such as NSAIDs or biologics. By analyzing these biomarkers, clinicians can personalize treatment regimens to target the specific inflammatory mediators involved in each patient's disease, increasing the likelihood of therapeutic success and minimizing the risk of adverse effects. Additionally, precision medicine can be applied to the management of drug interactions in periodontal disease treatment[16]. By identifying genetic variations in drug-metabolizing enzymes, such as those in the cytochrome P450 family, clinicians can tailor drug dosages to ensure optimal efficacy and reduce the risk of toxicity. The use of precision medicine in periodontal disease management also extends to the development of targeted biologic therapies. Monoclonal antibodies and other biologics that selectively inhibit specific inflammatory cytokines, such as TNF-α or IL-1, are being tailored to individual patient profiles based on their genetic and molecular characteristics.

The Potential of Anti-inflammatory Gene Therapy in Periodontal Treatment

Anti-inflammatory gene therapy holds significant potential in the treatment of periodontal disease by targeting the underlying molecular mechanisms responsible for chronic inflammation and tissue destruction. Unlike traditional pharmacological therapies, which aim to inhibit the action of inflammatory mediators such as cytokines and prostaglandins, gene therapy offers the possibility of directly modifying the genetic material within target cells to alter the inflammatory response at the source. The principle of anti-inflammatory gene therapy involves the introduction of specific genes into the affected tissues that either suppress the production of pro-inflammatory cytokines or enhance the expression of anti-inflammatory mediators. For instance, gene delivery methods could be used to transfer genes encoding cytokine inhibitors, such as interleukin-1 receptor antagonists (IL-1Ra) or soluble TNF receptors, directly into the periodontal tissues, thus blocking the activity of key inflammatory cytokines that drive tissue destruction in periodontal disease. Additionally, gene therapy could be used to deliver genes that promote tissue repair and regeneration, such as growth factors or extracellular matrix proteins, to enhance healing and restore periodontal tissues affected by inflammation[7]. One of the major advantages of gene therapy in periodontal treatment is its ability to provide long-lasting effects with a single treatment, as the introduced genes can continue to produce therapeutic

proteins over an extended period. Furthermore, gene therapy has the potential to target specific tissues or cells within the periodontal region, offering highly localized treatment and minimizing systemic exposure. The use of viral vectors, such as adenoviruses or lentiviruses, or non-viral methods, such as liposomes or nanoparticles, can facilitate the delivery of therapeutic genes to the periodontal tissues, where they can be taken up by target cells and expressed. Despite the promising potential of anti-inflammatory gene therapy, there are several challenges that must be addressed, including the safe and efficient delivery of genes to the target tissues, the risk of immune responses to the introduced genes or vectors, and the need for long-term stability of the therapeutic effects. Nonetheless, as technology advances, gene therapy has the potential to revolutionize periodontal treatment, providing a novel and highly targeted approach to managing chronic inflammation and promoting tissue regeneration in patients with periodontal disease.[17]

CONCLUSION

The Anti-inflammatory drugs play a vital role in the management of periodontal disease by modulating the inflammatory processes that contribute to tissue destruction and disease progression. The chronic inflammation observed in periodontal disease, driven by the host's immune response to bacterial plaque, is responsible for the breakdown of periodontal tissues, including the gingiva, periodontal ligament, and alveolar bone. Anti-inflammatory drugs, such as nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and cytokine inhibitors, are used adjunctively with conventional therapies like scaling and root planing to alleviate inflammation, reduce pain, and promote tissue healing. NSAIDs, by inhibiting cyclooxygenase (COX) enzymes, reduce prostaglandin production, a key mediator of inflammation and pain, leading to improvements in clinical parameters such as pocket depth and clinical attachment loss. Corticosteroids, while potent anti-inflammatory agents, are typically used in more severe cases of periodontal disease due to their potential for systemic side effects, but they offer rapid control over inflammation by suppressing cytokine production. The advent of biologics and targeted therapies that focus on specific inflammatory mediators, such as TNF-α and IL-1, presents a promising future for periodontal therapy, offering more selective and effective ways to manage inflammation with fewer side effects. Despite the efficacy of these anti-inflammatory drugs, their use must be balanced with the potential risks, such as gastrointestinal irritation, renal toxicity, and impaired wound healing, which can limit their long-term application in periodontal disease management.

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Cite this article:
Dr. Subramanianathan K. The Role of Anti-Inflammatory Drugs in Periodontal Disease Treatment: Mechanisms and Clinical Efficacy. *American Journal of Oral Medicine and Radiology*, 13(1), 2026, 35-42.