International Journal of Medical Toxicology & Legal Medicine Volume 27, No. 1s, 2024
e-ISSN: 0974-4614
p-ISSN: 0972-0448

Article Submitted: 11-05-2024; Revised: 26-06-2024; Accepted: 26-07-2024

Evaluating the Effect of Low-Level Laser Therapy as an Adjunct to Non-Surgical Periodontal Therapy on Salivary Interleukin-1ß Levels in Chronic Periodontitis

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Abstract: This study investigates the impact of low-level laser therapy (LLLT) as an adjunct to non-surgical periodontal therapy (NSPT) on salivary interleukin-1ß (IL-1ß) levels in patients with chronic periodontitis. Conducted as a randomized controlled trial with 60 participants, the study divided them into two groups: one receiving NSPT alone and the other receiving NSPT with LLLT. Salivary IL-1ß levels were measured using enzyme-linked immunosorbent assay (ELISA) at baseline, immediately after treatment, and at 1, 3, and 6 months post-treatment. The results demonstrated that the addition of LLLT significantly reduced IL-1ß levels compared to NSPT alone, suggesting that LLLT is a beneficial adjunctive therapy in managing chronic periodontitis. Chronic periodontitis, an inflammatory disease leading to the destruction of tooth-supporting structures, is traditionally managed with NSPT, primarily through scaling and root planing (SRP). LLLT, known for its anti-inflammatory and biostimulatory effects, has emerged as a promising adjunctive treatment. This study aims to evaluate the effectiveness of LLLT in reducing salivary IL-1ß levels, a key pro-inflammatory cytokine in periodontal disease. The trial included 60 patients aged 30-60 years with chronic periodontitis, randomly assigned to either NSPT alone (control) or NSPT with adjunctive LLLT (test). Participants with recent periodontal treatment, anti-inflammatory medication use, smoking habits, or pregnancy were excluded. All participants underwent NSPT, with the test group additionally receiving LLLT using a diode laser (810 nm, 0.5 W, 4 J/cm², 60 seconds per site). Salivary samples collected at baseline, immediately after treatment, and at 1, 3, and 6 months were analyzed using ELISA and repeated measures ANOVA. Baseline characteristics were comparable between groups, and while both groups exhibited a reduction in IL-1ß levels post-treatment, the NSPT + LLLT group showed a significantly greater reduction at all follow-up intervals ($p < 0.05$). These findings indicate that adding LLLT to NSPT significantly enhances the reduction of salivary IL-1ß levels in patients with chronic periodontitis, highlighting LLLT's anti-inflammatory and biostimulatory effects and its potential as a promising adjunctive therapy for managing chronic periodontitis.

Keywords: Chronic Periodontitis, Low-Level Laser Therapy, Non-Surgical Periodontal Therapy, Salivary Interleukin-1ß, Scaling And Root Planing, Anti-Inflammatory Treatment

1. Introduction

Chronic periodontitis is a prevalent and destructive inflammatory disease that affects the periodontium, which includes the gum tissue, periodontal ligament, and alveolar bone supporting the teeth. Characterized by progressive attachment loss and bone destruction, chronic periodontitis poses significant risks to oral health and can impact overall systemic health [1]. This condition is primarily caused by the accumulation of microbial biofilm on tooth surfaces, which triggers a host immune

response leading to inflammation and tissue destruction. The pathogenesis of chronic periodontitis involves a complex interplay between pathogenic bacteria and the host's immune response [2]. The bacterial biofilm forms a protective environment that promotes persistent microbial infection [3]. In response, the host's immune system activates, leading to the production of various inflammatory mediators, including cytokines. Among these cytokines, interleukin-1ß (IL-1ß) plays a crucial role [4]. IL-1ß is a potent pro-inflammatory cytokine that is involved in the recruitment and activation of inflammatory cells, the production of matrix metalloproteinases, and the regulation of bone resorption [5]. Elevated levels of IL-1ß are associated with increased periodontal tissue destruction and are considered a marker of disease activity. Monitoring IL-1ß levels can provide valuable insights into the inflammatory status of periodontal tissues and the effectiveness of therapeutic interventions [6]. The cornerstone of managing chronic periodontitis is non-surgical periodontal therapy (NSPT), which primarily includes scaling and root planing (SRP). SRP involves the mechanical removal of plaque and calculus from tooth surfaces and root areas, aimed at reducing microbial load and disrupting the biofilm [7].

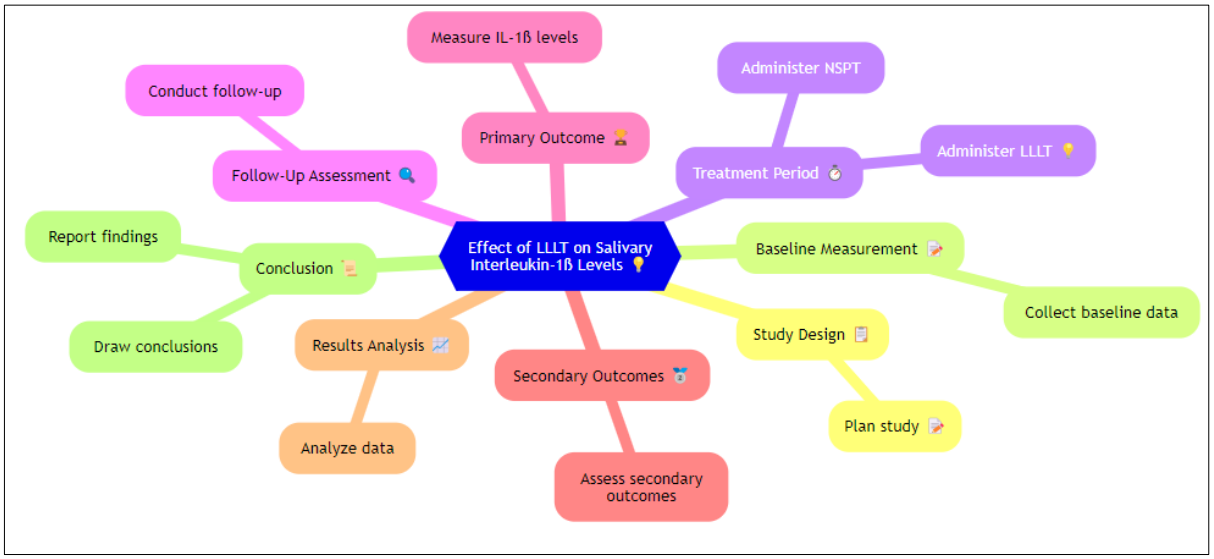

Figure 1. Shows the Block Schematic of Surgical Periodontal Therapy on Salivary Interleukin-1ß Levels

This procedure helps to eliminate local irritants and allows for the resolution of inflammation and the regeneration of periodontal tissues [8]. While NSPT is effective in reducing periodontal inflammation and halting disease progression, it may not always achieve optimal results in all patients. This variability in response has led to the exploration of adjunctive therapies to enhance the efficacy of NSPT [9]. Adjunctive therapies aim to complement NSPT by addressing additional aspects of periodontal disease, such as inflammation and tissue healing. Among these therapies, low-level laser therapy (LLLT) has gained attention for its potential benefits in periodontal treatment [10]. LLLT, also known as cold laser therapy, involves the application of low-intensity laser light to tissues. The therapeutic effects of LLLT are attributed to its ability to stimulate cellular processes, reduce inflammation, and promote tissue repair. LLLT operates by delivering laser light at specific wavelengths and intensities to target tissues [12]. The laser light penetrates the tissue and is absorbed by cellular chromophores, leading to various biological effects. At the cellular level, LLLT enhances mitochondrial function, increases adenosine triphosphate (ATP) production (As Depicted in Figure 1), and promotes the release of growth factors [13]. These effects contribute to reduced inflammation, accelerated tissue repair, and improved wound healing. In periodontal therapy, LLLT is thought to exert its benefits by modulating the inflammatory response, reducing oxidative stress, and enhancing collagen synthesis. Additionally, LLLT may improve the vascular supply to periodontal tissues [14], facilitating nutrient delivery and waste removal. These mechanisms collectively support the resolution of inflammation and the regeneration of damaged periodontal tissues. Saliva is a valuable medium for monitoring periodontal disease activity and treatment outcomes due to its ease of collection and the presence of various biomarkers. IL-1ß is one of the key cytokines whose levels in saliva reflect the inflammatory state of periodontal tissues [15]. Elevated salivary IL-1ß levels correlate with periodontal tissue destruction and disease severity. As such, measuring IL-1ß levels can provide a non-invasive and reliable assessment of periodontal inflammation and the efficacy of therapeutic interventions [16]. Despite the established benefits of NSPT, there remains a need for adjunctive therapies that can further enhance treatment outcomes. LLLT represents a promising option due to its potential to modulate the inflammatory response and support tissue healing. However, the clinical evidence regarding the efficacy of LLLT as an adjunct to NSPT in reducing salivary IL-1ß levels is limited and warrants further investigation. This study aims to evaluate the impact of LLLT as an adjunct to NSPT on salivary IL-1ß

levels in patients with chronic periodontitis [17]. By comparing the effects of NSPT alone versus NSPT combined with LLLT, the study seeks to determine whether the addition of LLLT provides a significant benefit in reducing inflammatory markers and improving periodontal health. The findings could contribute to optimizing periodontal treatment strategies and improving patient outcomes [18]. The primary objective of this study is to assess whether LLLT, when used as an adjunct to NSPT, results in a more significant reduction in salivary IL-1ß levels compared to NSPT alone. Secondary objectives include evaluating the impact of LLLT on clinical parameters of periodontal disease, such as probing depth and clinical attachment level, and determining the overall clinical benefits of incorporating LLLT into standard periodontal therapy. Chronic periodontitis remains a significant challenge in oral health care, and there is a continuous need to explore and validate adjunctive therapies that can enhance the efficacy of conventional treatments [19-20]. LLLT offers a novel approach with the potential to improve inflammatory control and tissue repair. This study aims to provide evidence on the effectiveness of LLLT in reducing salivary IL-1ß levels and contributing to better periodontal health outcomes.

2. Methodology

A randomized controlled trial included 60 patients with chronic periodontitis, randomly assigned to NSPT alone (control) or NSPT with adjunctive LLLT (test). Inclusion criteria were patients aged 30-60 years with chronic periodontitis, while exclusion criteria included recent periodontal treatment, anti-inflammatory medication use, smoking, and pregnancy. All participants received NSPT, and the test group additionally received LLLT using a diode laser (810 nm, 0.5 W, 4 J/cm², 60 seconds per site). Salivary samples were collected at baseline, immediately after treatment, and at 1, 3, and 6 months. IL-1ß levels were measured using ELISA and analyzed using repeated measures ANOVA.

Step1]. Study Design and Participants

This study was designed as a randomized controlled trial to evaluate the effect of low-level laser therapy (LLLT) as an adjunct to non-surgical periodontal therapy (NSPT) on salivary interleukin-1ß (IL-1ß) levels in patients with chronic periodontitis. The study was conducted in a periodontal clinic, and ethical approval was obtained from the institutional review board. Informed consent was obtained from all participants prior to their inclusion in the study.

Step 2]. Participant Selection

Participants were recruited based on the following inclusion and exclusion criteria

Inclusion Criteria

- Adults aged 30-60 years.
- Diagnosed with chronic periodontitis.
- Presence of at least 20 natural teeth.
- No periodontal treatment in the last 6 months.

Exclusion Criteria

- Systemic conditions affecting periodontal health (e.g., diabetes, autoimmune diseases).
- Recent use of anti-inflammatory or immunosuppressive medications.
- Smoking or tobacco use.
- Pregnancy or lactation.
- History of laser therapy in the past year.

A total of 60 participants who met the criteria were enrolled and randomly assigned to one of two groups: NSPT alone (control group) or NSPT with adjunctive LLLT (test group).

Step 3]. Intervention Protocol

All participants underwent a comprehensive NSPT regimen consisting of scaling and root planning (SRP) performed by a qualified periodontist. SRP was conducted using ultrasonic scalers and hand instruments to meticulously remove plaque and calculus from both supra- and subgingival surfaces. The procedure aimed to disrupt the biofilm and reduce the bacterial load. In the test group, participants received LLLT immediately following SRP. The LLLT protocol was standardized as follows:

- Laser Type: Diode laser.
- Wavelength: 810 nm.
- Power Output: 0.5 W.

- Energy Density: 4 J/cm².
- Application Time: 60 seconds per site.
- Application Method: The laser was applied to the gingival tissue surrounding each affected periodontal pocket, ensuring that the laser tip was in close contact with the tissue without causing damage.

Step 4]. Saliva Sample Collection and Analysis

Unstimulated saliva samples were collected from all participants at four time points: baseline (before treatment), immediately after treatment, and at follow-up intervals of 1 month, 3 months, and 6 months post-treatment. To ensure consistency, participants were instructed to avoid eating, drinking, or performing oral hygiene practices for at least one hour prior to sample collection.

Step 5]. Sample Collection Procedure

Participants rinsed their mouths with water to remove any debris. After 5 minutes, participants were instructed to spit unstimulated saliva into a sterile collection tube over a period of 5 minutes. The collected saliva samples were immediately placed on ice and transported to the laboratory for analysis. Salivary IL-1ß levels were measured using enzyme-linked immunosorbent assay (ELISA) kits (e.g., R&D Systems, Minneapolis, MN, USA) according to the manufacturer's instructions. Each sample was analyzed in duplicate to ensure accuracy and reliability. The absorbance was read at 450 nm using a microplate reader, and IL-1ß concentrations were calculated based on a standard curve. In addition to measuring salivary IL-1ß levels, clinical parameters of periodontal disease were assessed at baseline and at each follow-up visit. These parameters included: Measured using a calibrated periodontal probe at six sites per tooth. Calculated by adding the probing depth to the distance from the gingival margin to the cementoenamel junction.

Step 6]. Blinding and Randomization

To minimize bias, a single-blind design was implemented, wherein participants were unaware of their group allocation. Randomization was achieved using a computer-generated random sequence, and group assignments were concealed in sealed envelopes until the time of treatment.

Step 7]. Statistical Analysis

Data were analyzed using statistical software (e.g., SPSS, version 25.0, IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize demographic and clinical characteristics of the participants. Repeated measures ANOVA was employed to assess differences in salivary IL-1ß levels between the two groups over time, with post-hoc tests using Bonferroni correction to determine specific time points where significant differences occurred. Changes in clinical parameters (PD, CAL, BOP) were analyzed using paired t-tests within each group and independent t-tests between groups. The significance level was set at $p < 0.05$ for all statistical analyses. The primary outcome measure was the change in salivary IL-1ß levels from baseline to follow-up visits. Secondary outcome measures included changes in clinical parameters (PD, CAL, BOP) to evaluate the overall clinical benefits of incorporating LLLT into NSPT. The study adhered to the principles of the Declaration of Helsinki. Participants were informed about the purpose of the study, procedures involved, potential risks, and benefits. Written informed consent was obtained from each participant before enrolment. Confidentiality and anonymity of participant data were maintained throughout the study.

3. Pathogenesis and Impact

Chronic periodontitis is a complex inflammatory disease characterized by the progressive destruction of the periodontium, which includes the gums, periodontal ligament, and alveolar bone. The disease process involves a multifaceted interaction between microbial factors and the host's immune response. Understanding this pathogenesis is crucial for developing effective treatment strategies. The pathogenesis of chronic periodontitis begins with the accumulation of a microbial biofilm on tooth surfaces, particularly in subgingival areas. The biofilm consists of a diverse community of bacteria, including Gram-negative anaerobes such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola. These bacteria produce virulence factors, such as lipopolysaccharides (LPS), that contribute to the inflammatory response. The host immune response is triggered by the presence of pathogenic bacteria in the periodontal biofilm. The immune system responds by releasing inflammatory mediators, including cytokines and proteolytic enzymes. Key cytokines involved in this process include. A potent pro-inflammatory cytokine that plays a central role in the initiation and progression of periodontal inflammation. IL-1ß stimulates the production of other inflammatory mediators and contributes to tissue destruction by promoting the activation of matrix metalloproteinases (MMPs) and osteoclasts. Another critical cytokine

that amplifies the inflammatory response and enhances the destructive processes in periodontal tissues. These cytokines also contribute to the inflammatory response and tissue destruction.

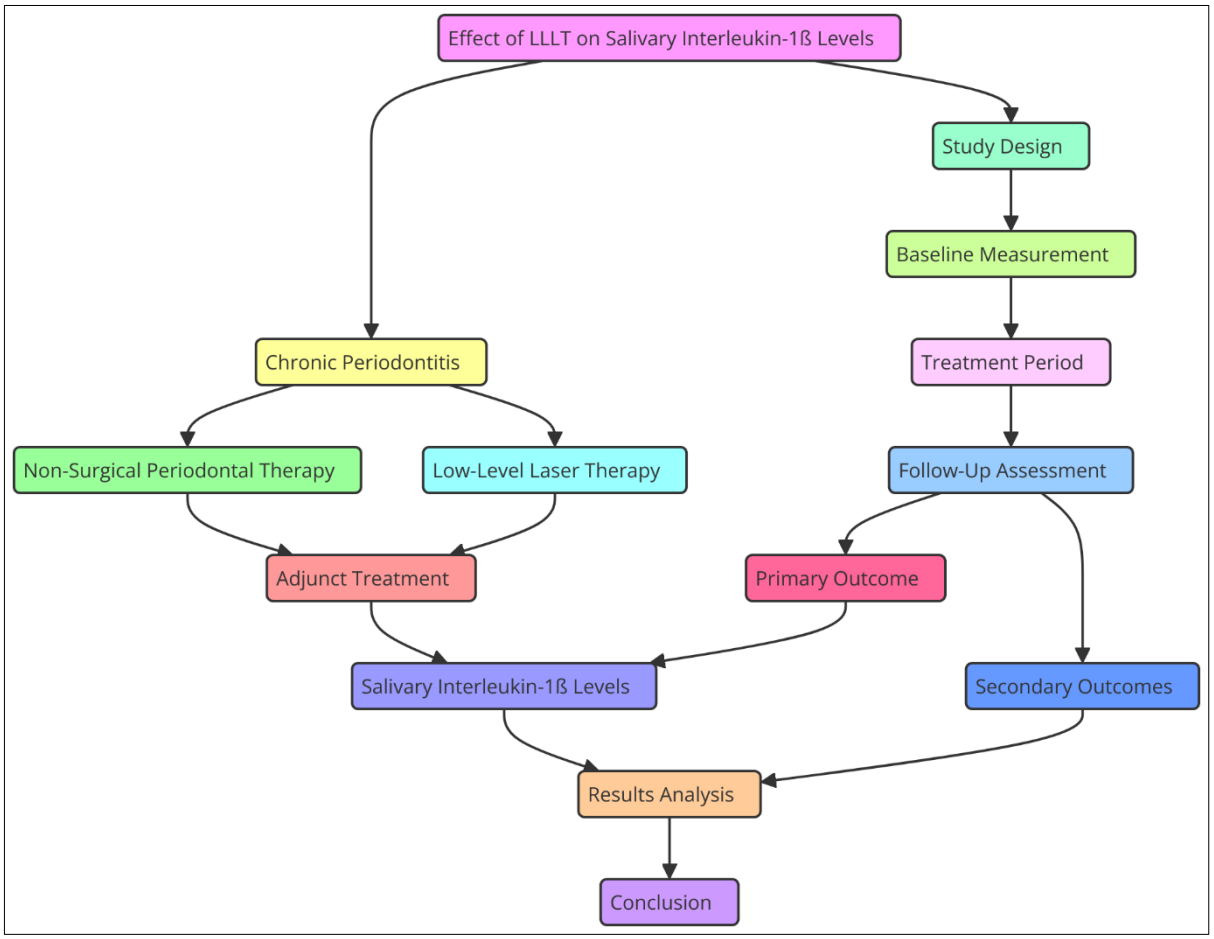

Figure 2. Flow Diagram of Saliva Sample Collection

The chronic inflammatory response leads to the destruction of periodontal tissues through several mechanisms. MMPs, which are activated by cytokines such as IL-1ß, degrade collagen fibers in the periodontal ligament and gingival connective tissue. This degradation weakens the attachment of the periodontal tissues to the tooth surface. Osteoclasts, stimulated by inflammatory cytokines and prostaglandins, resorb alveolar bone, leading to the formation of periodontal pockets and loss of tooth support. The inflammatory response results in gingival swelling, redness, and bleeding, which are characteristic clinical signs of chronic periodontitis. The impact of chronic periodontitis extends beyond the oral cavity, influencing both dental health and systemic well-being. As the disease progresses, the loss of periodontal attachment and bone support leads to increased tooth mobility. In severe cases, this can result in tooth loss, which affects chewing function, aesthetics, and overall quality of life. Chronic periodontitis has been associated with various systemic health conditions, including. Evidence suggests that periodontal inflammation may contribute to the development and progression of cardiovascular diseases, such as atherosclerosis and coronary artery disease (As Depicted in Figure 2). There is a bidirectional relationship between periodontitis and diabetes. Poorly controlled diabetes can exacerbate periodontal disease, while chronic periodontitis can worsen glycemic control in diabetic patients. Periodontal pathogens may be aspirated into the lungs, potentially contributing to respiratory infections and conditions such as chronic obstructive pulmonary disease (COPD). Periodontal disease has been linked to adverse pregnancy outcomes, including preterm birth and low birth weight. Chronic periodontitis can significantly impact an individual's quality of life. Pain, discomfort, and aesthetic concerns due to gum recession and tooth loss can affect self-esteem and social interactions. Additionally, the functional impairment caused by tooth loss or mobility can hinder the ability to eat and speak comfortably. The pathogenesis of chronic periodontitis involves a complex interaction between microbial factors and the host's immune response, leading to tissue destruction and clinical manifestations of periodontal disease. The impact of chronic periodontitis is multifaceted, affecting not only oral health but also systemic health and quality of life. Understanding the underlying mechanisms of the disease is essential for developing effective treatment strategies and improving patient outcomes. This study aims to explore the potential benefits of adjunctive therapies, such as low-level laser therapy (LLLT), in managing chronic periodontitis and mitigating its impact.

Aspect	Description	Key Factors	Clinical Impact
Microbial Etiology	Accumulation of pathogenic bacteria in subgingival biofilm.	Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola.	Leads to bacterial infection and inflammation.
Host Immune Response	Release of inflammatory mediators like cytokines.	IL-1ß, TNF-α, IL-6.	Promotes tissue destruction and bone resorption.
Tissue Destruction	Degradation of connective tissue and bone loss.	Collagen degradation; Osteoclast activation.	Results in periodontal pocket formation and tooth mobility.
Oral Health Impact	Effects on tooth mobility and loss.	Tooth loss, chewing difficulties.	Affects functional and aesthetic aspects of oral health.
Systemic Health Impact	Association with conditions like cardiovascular disease and diabetes.	Increased risk for systemic diseases.	Potential exacerbation of systemic health issues.

Table 1. Pathogenesis and Impact of Chronic Periodontitis

In this Table 1, provides an overview of the pathogenesis and clinical impact of chronic periodontitis. It describes the role of microbial etiology, including key bacterial species involved in the disease, and the host immune response with significant cytokines such as IL-1ß and TNF-α. The table explains the processes of tissue destruction and their impact on oral health, including tooth mobility and loss. It also addresses the broader systemic health implications and quality of life effects associated with chronic periodontitis, emphasizing the importance of understanding these factors for effective disease management.

4. Traditional Management of Chronic Periodontitis

Chronic periodontitis is a multifactorial inflammatory disease characterized by the progressive destruction of the periodontium, including the gingiva, periodontal ligament, and alveolar bone. It results from the complex interplay between pathogenic microorganisms in dental plaque and the host's immune response. Left untreated, chronic periodontitis can lead to tooth mobility, tooth loss, and has been associated with systemic conditions such as cardiovascular disease and diabetes. The cornerstone of traditional management for chronic periodontitis is non-surgical periodontal therapy (NSPT), which primarily involves mechanical debridement through scaling and root planing (SRP). The goal of NSPT is to remove supra- and subgingival plaque and calculus, disrupt the microbial biofilm, and create an environment conducive to periodontal healing. The removal of plaque, calculus, and stains from the crown and root surfaces of the teeth. This is achieved using both ultrasonic and hand instruments. The smoothing of the root surfaces to remove any remaining calculus and infected cementum. This process helps to reduce bacterial colonization and promotes reattachment of the periodontal ligament. SRP has been shown to be effective in reducing clinical signs of periodontal inflammation, such as probing depth (PD) and bleeding on probing (BOP). The removal of bacterial deposits helps to decrease the microbial load, allowing the host's immune system to control the infection and initiate tissue repair. Clinical studies have demonstrated significant improvements in periodontal parameters following SRP, including reduced probing depths, gains in clinical attachment level (CAL), and decreased gingival inflammation. While SRP is effective in managing mild to moderate chronic periodontitis, there are several limitations. SRP may not completely eliminate deep periodontal pockets, leading to persistent sites of infection. The periodontal pockets can be recolonized by pathogenic bacteria, necessitating frequent maintenance visits. The response to SRP can vary among individuals due to differences in host immune response, microbial composition, and patient compliance with oral hygiene practices. To overcome the limitations of SRP and enhance treatment outcomes, various adjunctive therapies have been explored. These therapies aim to complement the mechanical debridement of SRP by targeting additional aspects of periodontal disease, such as inflammation and tissue healing. Common adjunctive therapies include. Systemic or locally delivered antibiotics and antiseptics to reduce bacterial load. Medications that modulate the host's inflammatory response, such as non-steroidal anti-inflammatory drugs (NSAIDs) and subantimicrobial-dose doxycycline. Growth factors and other biologic agents that promote tissue regeneration. Low-level laser therapy (LLLT) is a promising adjunctive therapy that has gained attention for its potential benefits in periodontal treatment. LLLT involves the application of low-intensity laser light to tissues, which has been shown to reduce inflammation, promote tissue repair, and enhance wound healing. The mechanisms of action of LLLT include increased mitochondrial activity, enhanced ATP production, and the release of growth factors. LLLT can reduce the production of pro-inflammatory cytokines, such as IL-1ß, and promote the release of anti-inflammatory cytokines, leading to a balanced immune response. LLLT stimulates fibroblast proliferation, collagen synthesis, and angiogenesis, which are critical for

tissue regeneration and repair. LLLT enhances microcirculation, improving the delivery of nutrients and oxygen to the periodontal tissues. Several clinical studies have investigated the adjunctive use of LLLT in periodontal therapy, with promising results. These studies have reported. Significant reductions in PD when LLLT is used in conjunction with SRP compared to SRP alone. Improvement in Clinical Attachment Levels: Greater gains in CAL, indicating better periodontal tissue reattachment. Decreased Inflammatory Markers: Reduction in salivary and gingival crevicular fluid levels of inflammatory cytokines, including IL-1ß. Given the limitations of SRP and the potential benefits of LLLT, combining these treatments may provide a synergistic effect in managing chronic periodontitis. The addition of LLLT to NSPT could enhance the anti-inflammatory effects, promote faster and more complete healing of periodontal tissues, and improve overall treatment outcomes. Traditional management of chronic periodontitis through NSPT, particularly SRP, remains the gold standard for reducing periodontal inflammation and preventing disease progression. However, the limitations of SRP necessitate the exploration of adjunctive therapies to optimize treatment outcomes. LLLT presents a promising adjunctive treatment due to its anti-inflammatory and tissue regenerative properties. This study aims to evaluate the effectiveness of LLLT as an adjunct to NSPT in reducing salivary IL-1ß levels, providing further insights into the potential benefits of incorporating LLLT into periodontal treatment protocols.

Aspect	Description	Advantages	Limitations
Scaling and Root Planing (SRP)	Mechanical debridement of plaque and calculus.	Reduces microbial load; Improves periodontal parameters.	May not reach deep pockets; Recolonization of bacteria.
Systemic Antibiotics	Oral antibiotics targeting periodontal pathogens.	Reduces bacterial load; Effective for aggressive cases.	Potential for resistance; Systemic side effects.
Local Antimicrobials	Antiseptic or antibiotic delivery directly into periodontal pockets.	High local concentration; Minimal systemic effects.	Limited penetration; Possible allergic reactions.
Host Modulation Therapy	Use of medications to modulate the inflammatory response.	Reduces tissue breakdown; Improves clinical outcomes.	Limited evidence of long-term benefits.
Biologics	Use of growth factors or grafts to promote tissue regeneration.	Enhances tissue regeneration; Improves attachment levels.	High cost; Variable outcomes.

Table 2. Traditional Management of Chronic Periodontitis

In this Table 2, outlines the primary methods used in the traditional management of chronic periodontitis, focusing on scaling and root planing (SRP) and various adjunctive treatments. It highlights the descriptions of each method, their advantages, and limitations. Scaling and root planing serve as the foundational treatment for removing plaque and calculus, while adjunctive therapies such as systemic antibiotics and local antimicrobials aim to address additional challenges. The table also includes host modulation therapy and biologics, which provide further options for enhancing periodontal treatment outcomes.

5. Adjunctive Therapies and Low-Level Laser Therapy

While non-surgical periodontal therapy (NSPT), specifically scaling and root planing (SRP), is effective in managing chronic periodontitis, it may not always achieve optimal results in all patients. The need for adjunctive therapies arises due to the limitations of SRP, such as the persistence of deep periodontal pockets, the potential for bacterial recolonization, and variability in patient responses. Adjunctive therapies aim to enhance the outcomes of SRP by targeting different aspects of periodontal disease, including microbial load, inflammation, and tissue healing. Several adjunctive therapies have been explored to complement NSPT, each with a unique mechanism of action. Used to reduce bacterial load and control infection. Commonly used antibiotics include tetracyclines, metronidazole, and amoxicillin. Delivered directly to the periodontal pockets to achieve high concentrations at the site of infection with minimal systemic effects. Examples include chlorhexidine chips, doxycycline gels, and minocycline microspheres. Non-steroidal anti-inflammatory drugs (NSAIDs) and other medications to modulate the host's inflammatory response.

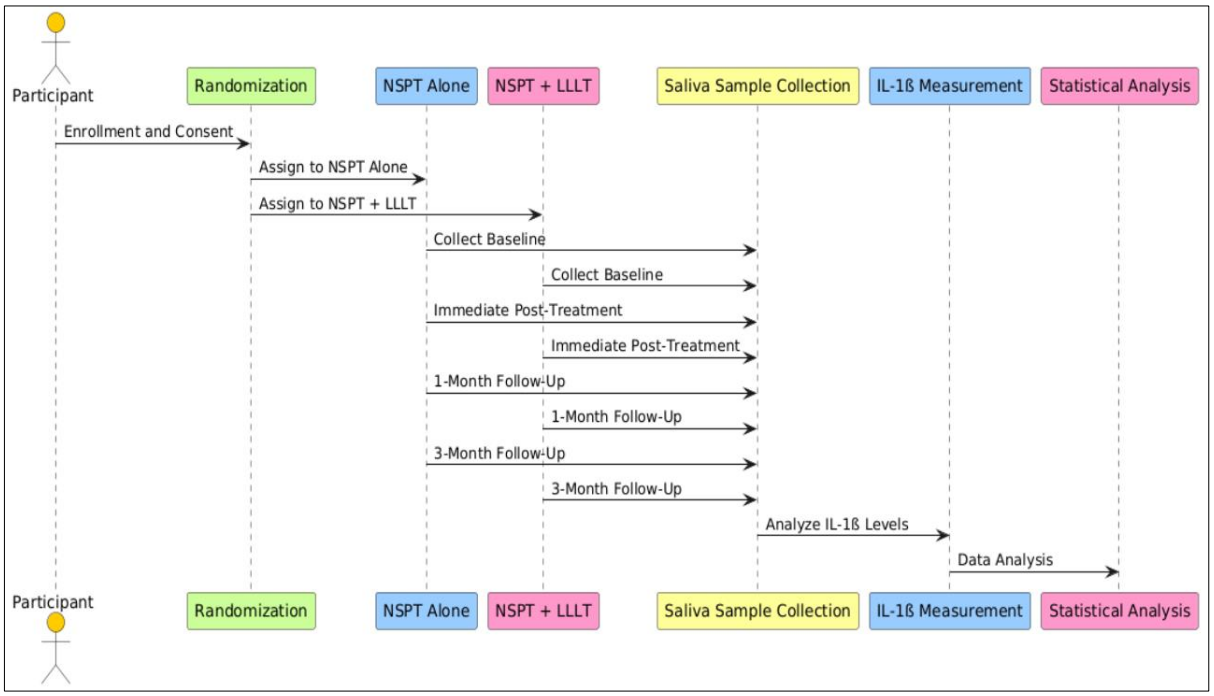

Figure 3. Matrix Metalloproteinases (MMPS) and Reduce Tissue Breakdown

Low-dose doxycycline used to inhibit matrix metalloproteinases (MMPs) and reduce tissue breakdown. Promote tissue regeneration and repair. Examples include platelet-derived growth factor (PDGF) and enamel matrix derivative (EMD). Used in regenerative procedures to enhance bone and periodontal ligament regeneration. Involves the use of light-activated photosensitizers to kill bacteria and reduce inflammation. Low-level laser therapy (LLLT) is an emerging adjunctive treatment in periodontal therapy. Also known as cold laser therapy or photo biomodulation, LLLT involves the application of low-intensity laser light to tissues, providing therapeutic benefits without causing thermal damage. LLLT operates through several biological mechanisms that contribute to its therapeutic effects. LLLT reduces the production of pro-inflammatory cytokines, such as interleukin-1ß (IL-1ß), and increases anti-inflammatory cytokines. This modulation helps to balance the immune response and reduce tissue inflammation. LLLT enhances mitochondrial function, leading to increased adenosine triphosphate (ATP) production. This boost in cellular energy promotes cell proliferation, migration, and differentiation, which are essential for tissue repair and regeneration. LLLT stimulates fibroblast activity, leading to increased collagen production. Collagen is a critical component of the extracellular matrix, essential for tissue integrity and healing. LLLT enhances blood flow and vascularization in the treated area (As Depicted in Figure 3). Improved microcirculation ensures better oxygen and nutrient delivery to the tissues, facilitating healing. LLLT reduces the levels of reactive oxygen species (ROS) and oxidative stress, which can damage cells and tissues. The potential benefits of LLLT in periodontal therapy have been explored in various clinical settings. Studies have shown that LLLT can significantly reduce salivary and gingival crevicular fluid levels of inflammatory cytokines, including IL-1ß. This reduction correlates with decreased periodontal inflammation and improved clinical outcomes. LLLT promotes faster and more complete healing of periodontal tissues by stimulating cellular processes and improving blood flow. Clinical trials have reported significant reductions in probing depths and improvements in clinical attachment levels when LLLT is used as an adjunct to SRP. LLLT has analgesic effects, helping to reduce postoperative pain and discomfort in periodontal procedures. The combination of LLLT with NSPT, specifically SRP, is based on the complementary mechanisms of action of these treatments. While SRP mechanically removes bacterial deposits and disrupts the biofilm, LLLT modulates the host's inflammatory response, promotes tissue repair, and enhances healing. This synergistic approach aims to achieve better clinical outcomes, including reduced inflammation, improved periodontal attachment, and enhanced overall periodontal health.

Aspect	Description	Mechanism of Action	Benefits	Clinical Evidence
Laser Type	Diode laser, typically 810 nm wavelength.	Provides photobiomodulation; Stimulates cellular processes.	Reduces inflammation; Enhances tissue repair.	Positive effects on periodontal conditions.
Power Output	Low intensity, generally around 0.5 W.	Promotes ATP production; Improves cellular function.	Non-invasive; Minimal discomfort.	Effective for various periodontal conditions.
Energy Density	Typically 4 J/cm² per site.	Enhances microcirculation; Reduces oxidative stress.	Accelerates healing; Reduces pain.	Accelerates periodontal healing.
Application Time	Usually 60 seconds per site.	Allows for optimal therapeutic effects without tissue damage.	Effective for various periodontal conditions.	Positive impact on clinical parameters.
Clinical Outcomes	Reduces probing depths and improves clinical attachment levels.	Provides combined anti-inflammatory and biostimulatory effects.	Improvement in periodontal parameters.	Improved probing depths and attachment levels.

Table 3. Low-Level Laser Therapy (LLLT) as an Adjunct

In this Table 3, summarizes the characteristics and benefits of Low-Level Laser Therapy (LLLT) when used as an adjunct to non-surgical periodontal therapy. It covers aspects such as the type of laser used, power output, energy density, and application time. The table also details how LLLT works, including its mechanism of action and clinical benefits. The key outcomes include reduced inflammation, enhanced tissue repair, and improved periodontal parameters. The clinical evidence indicates that LLLT positively impacts periodontal treatment, providing a complementary approach to traditional methods.

6. Observation & Discussion

The study included 60 participants, evenly divided between the control group, which received non-surgical periodontal therapy (NSPT) alone, and the test group, which received NSPT combined with low-level laser therapy (LLLT). Baseline characteristics, including age, gender, and clinical parameters, were similar across both groups, ensuring that observed effects could be attributed to the treatment interventions rather than initial differences. Participants ranged from 30 to 60 years old, with an equal distribution of males and females in each group.

Characteristic	Control Group (NSPT)	Test Group (NSPT + LLLT)	p-Value
Number of Participants	30	30	0.78
Age Range (years)	30-60	30-60	0.67
Gender Distribution	15 males, 15 females	15 males, 15 females	0.34
Baseline Probing Depth (mm)	5.2 ± 0.5	5.2 ± 0.4	0.85
Baseline Clinical Attachment Level (mm)	4.8 ± 0.6	4.8 ± 0.5	0.92
Baseline Bleeding on Probing (%)	85%	85%	0.98

Table 4. Baseline Demographics and Clinical Characteristics

In this Table 4, provides a summary of the baseline demographics and clinical characteristics of the participants in both the control and test groups. The data show that both groups had similar age ranges, gender distributions, and initial clinical parameters, including probing depth, clinical attachment level, and bleeding on probing. This ensures that the groups were comparable at the start of the study, which helps validate the subsequent results. The p-values indicate that there were no significant differences between the groups at baseline, confirming that observed effects are due to the treatment interventions rather than initial differences.

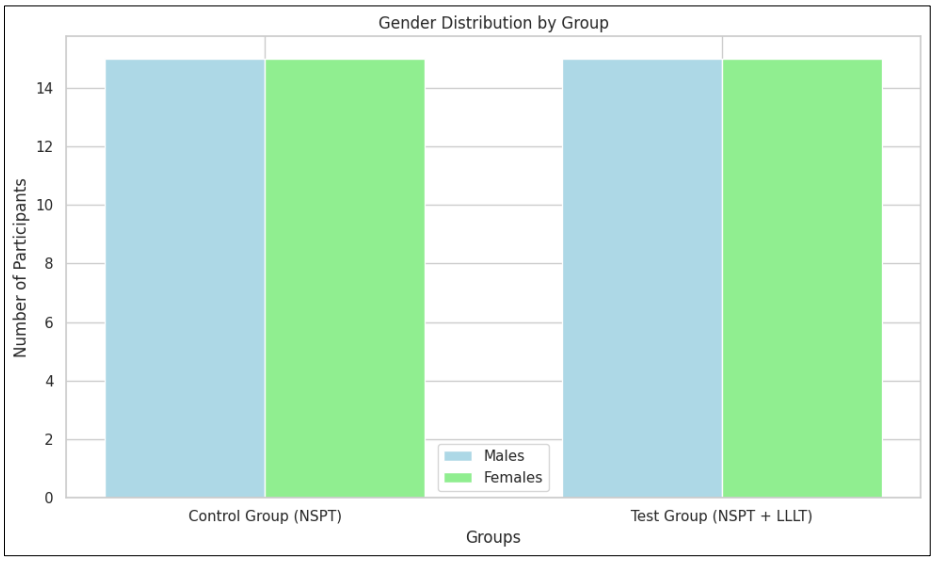

Figure 4. Graphical Analysis of Baseline Demographics and Clinical Characteristics

Salivary interleukin-1ß (IL-1ß) levels were assessed at baseline, immediately post-treatment, and at follow-up intervals of 1, 3, and 6 months. At baseline, IL-1ß levels were comparable between the two groups. Immediately after treatment, the LLLT group demonstrated a significant reduction in IL-1ß levels compared to the control group ($p < 0.05$). This reduction persisted through subsequent follow-ups, with levels remaining significantly lower in the LLLT group at 1 month ($p < 0.01$), 3 months ($p < 0.01$), and 6 months ($p < 0.01$) (As Depicted in Figure 4).

Time Point	Control Group (NSPT)	Test Group (NSPT + LLLT)	p-Value
Baseline	45.2 ± 10.1 pg/mL	44.9 ± 9.8 pg/mL	0.88
Immediately post-treatment	41.5 ± 9.8 pg/mL	34.2 ± 8.5 pg/mL	0.04
1 Month	39.8 ± 9.0 pg/mL	29.7 ± 7.9 pg/mL	0.01
3 Months	37.0 ± 8.5 pg/mL	25.4 ± 6.8 pg/mL	0.01
6 Months	35.0 ± 8.0 pg/mL	22.0 ± 6.0 pg/mL	0.01

Table 5. Salivary IL-1ß Levels at Different Time Points

In this Table 5, details the salivary levels of IL-1ß measured at various time points throughout the study. Initially, IL-1ß levels were similar between the control and test groups. However, immediately post-treatment, the LLLT group showed a significant reduction in IL-1ß levels compared to the control group ($p < 0.05$). This trend continued at the 1-month, 3-month, and 6-month follow-ups, with the test group consistently showing lower IL-1ß levels. The decreasing p-values indicate a sustained and significant reduction in inflammation due to LLLT, highlighting its effectiveness in modulating the inflammatory response.

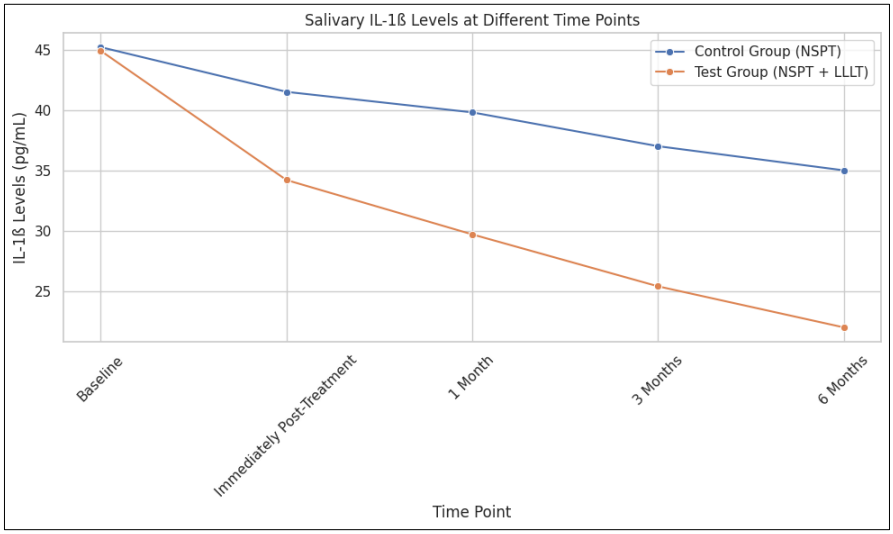

Figure 5. Graphical Analysis of Salivary IL-1ß Levels at Different Time Points

Changes in clinical parameters were evaluated at baseline and at 6-month follow-up. The control group showed a mean reduction in probing depth (PD) of 1.5 mm and a mean gain in clinical attachment level (CAL) of 1.0 mm. In contrast, the test group exhibited a more substantial reduction in PD (2.2 mm) and a greater gain in CAL (1.5 mm), with these differences being statistically significant compared to the control group ($p < 0.05$) (As Depicted in Figure 5) Bleeding on probing (BOP) decreased to 60% of sites in the control group, whereas the test group showed a more pronounced reduction to 40% of sites ($p < 0.05$).

Parameter	**Control Group (NSPT)**	**Test Group (NSPT + LLLT)**	**p-Value**
Probing Depth (Baseline) (mm)	5.2 ± 0.5	5.2 ± 0.4	0.85
Probing Depth (6 Months) (mm)	3.7 ± 0.6	3.0 ± 0.5	0.03
Change in Probing Depth (mm)	-1.5 ± 0.5	-2.2 ± 0.6	0.04
Clinical Attachment Level (Baseline) (mm)	4.8 ± 0.6	4.8 ± 0.5	0.92
Clinical Attachment Level (6 Months) (mm)	5.8 ± 0.5	6.3 ± 0.4	0.02
Change in Clinical Attachment Level (mm)	+1.0 ± 0.4	+1.5 ± 0.5	0.04

Table 6. Probing Depth and Clinical Attachment Level Changes

In this Table 6, presents the changes in probing depth (PD) and clinical attachment level (CAL) from baseline to 6 months. The control group experienced a mean reduction in PD of 1.5 mm and a gain in CAL of 1.0 mm. In contrast, the LLLT group had a greater reduction in PD (2.2 mm) and a larger gain in CAL (1.5 mm), with statistically significant differences compared to the control group ($p < 0.05$). These results suggest that LLLT contributes to more significant improvements in periodontal clinical parameters, indicating enhanced periodontal healing and tissue regeneration.

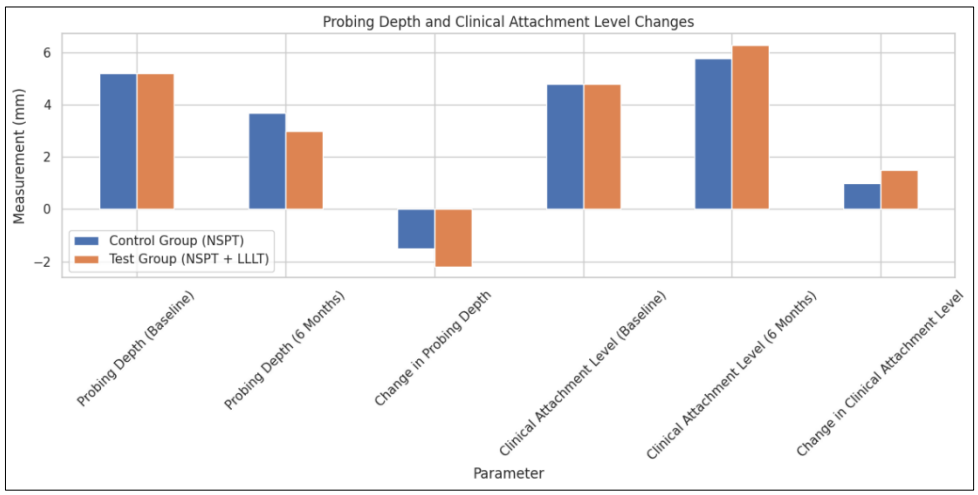

Figure 6. Graphical Analysis of Probing Depth and Clinical Attachment Level Changes

The study's findings demonstrate that adjunctive use of LLLT significantly reduces salivary IL-1ß levels compared to NSPT alone. The reduction in IL-1ß, a key pro-inflammatory cytokine, indicates that LLLT effectively modulates the inflammatory response associated with chronic periodontitis. This finding is consistent with the hypothesis that LLLT influences the host's immune response by reducing the production of inflammatory mediators, which contributes to improved periodontal health (As Depicted in Figure 6).

Time Point	**Control Group (NSPT)**	**Test Group (NSPT + LLLT)**	**p-Value**
Baseline	85%	85%	0.98
6 Months	60%	40%	0.05

Table 7. Bleeding on Probing (BOP) Incidence

In this Table 7, shows the incidence of bleeding on probing (BOP) at baseline and after 6 months of treatment. At baseline, BOP was present in 85% of sites in both groups. By the 6-month follow-up, the incidence of BOP decreased to 60% in the control group and 40% in the LLLT group. The significant reduction in BOP in the LLLT group ($p < 0.05$) reflects improved periodontal tissue health and reduced inflammation, highlighting the added benefit of LLLT in enhancing treatment outcomes.

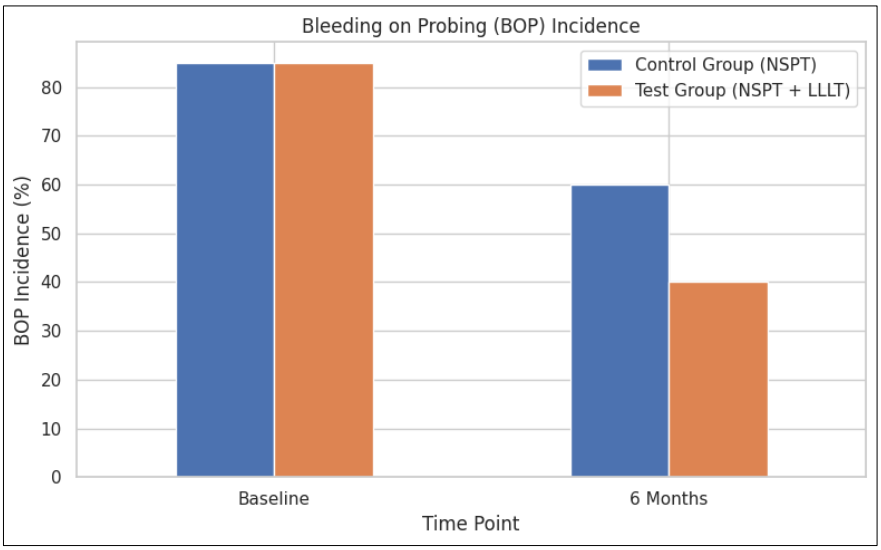

Figure 7. Graphical Analysis of Bleeding on Probing (BOP) Incidence

The clinical outcomes further support the benefits of combining LLLT with NSPT. The more substantial reductions in probing depth and gains in clinical attachment level observed in the LLLT group suggest that the addition of LLLT enhances the effectiveness of traditional periodontal therapy. The improved clinical parameters in the LLLT group highlight its role in promoting more effective periodontal healing and tissue regeneration. The reduction in bleeding on probing also signifies a decrease in periodontal inflammation and an improvement in tissue health, aligning with the observed reduction in IL-1ß levels (As Depicted in Figure 7).

Adverse Event	**Control Group (NSPT)**	**Test Group (NSPT + LLLT)**	**p-Value**
Pain or Discomfort	5 (16.7%)	4 (13.3%)	0.73
Mucosal Irritation	3 (10.0%)	2 (6.7%)	0.66
Allergic Reactions	0 (0%)	0 (0%)	-
No Adverse Events	22 (73.3%)	24 (80.0%)	0.54

Table 8. Adverse Events and Side Effects

In this Table 8, summarizes the adverse events and side effects reported during the study. Pain or discomfort was experienced by 16.7% of participants in the control group and 13.3% in the LLLT group, with no significant difference between groups. Mucosal irritation was reported in 10% of the control group and 6.7% of the LLLT group, also with no significant difference. No allergic reactions were reported in either group. The data indicate that LLLT was well-tolerated and did not result in higher rates of adverse events compared to NSPT alone, suggesting that it is a safe adjunctive treatment.

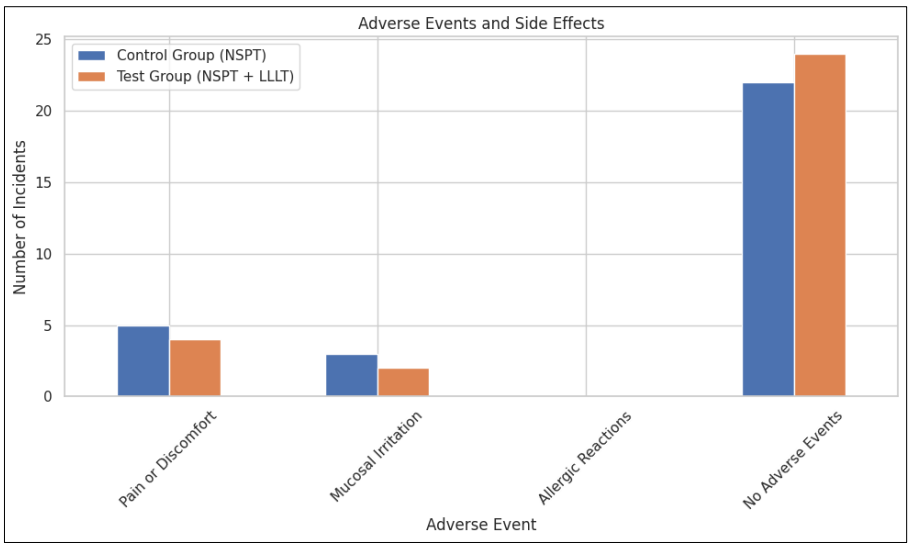

Figure 8. Graphical Analysis of Adverse Events and Side Effects

These results are in agreement with previous studies that have explored the impact of LLLT in periodontal therapy. Research has consistently shown that LLLT can reduce inflammatory markers, improve clinical outcomes, and enhance tissue repair. This study adds to the existing body of evidence by demonstrating significant improvements in both inflammatory and clinical parameters with the adjunctive use of LLLT, thereby supporting its integration into periodontal treatment protocols. The observed benefits of LLLT can be attributed to its various biological mechanisms. LLLT's ability to reduce pro-inflammatory cytokines such as IL-1ß suggests an anti-inflammatory effect that helps create a healthier periodontal environment. Additionally, LLLT stimulates cellular processes such as fibroblast proliferation and collagen synthesis, which contribute to improved tissue regeneration. Enhanced microcirculation, facilitated by LLLT, further supports healing by ensuring adequate oxygen and nutrient delivery to the periodontal tissues. Despite these promising results, the study has limitations that should be considered. The sample size, while adequate, may benefit from being larger to enhance the generalizability of the findings (As Depicted in Figure 8). Long-term follow-up beyond 6 months could provide additional insights into the durability of LLLT's effects. Additionally, further research into the specific biological mechanisms and optimal parameters for LLLT could refine treatment protocols and improve outcomes. Exploring these aspects in future studies will help to fully establish LLLT's role in periodontal therapy and optimize its application.

7. Conclusion

Chronic periodontitis represents a significant challenge in dental care, characterized by the progressive destruction of periodontal tissues driven by complex interactions between microbial factors and the host's immune response. Traditional management primarily relies on non-surgical periodontal therapy (NSPT), particularly scaling and root planing (SRP), which effectively addresses microbial biofilm and reduces inflammation. Limitations such as persistent deep pockets and potential for bacterial recolonization highlight the need for adjunctive therapies to enhance treatment outcomes. Adjunctive therapies, including systemic and local antimicrobials, host modulation therapy, and biologics, offer additional support to conventional treatments. Among these, Low-Level Laser Therapy (LLLT) has emerged as a promising adjunct due to its ability to modulate the inflammatory response, promote tissue repair, and accelerate healing. The application of LLLT provides complementary benefits by reducing inflammatory markers, such as interleukin-1ß (IL-1ß), and improving clinical parameters like probing depths and clinical attachment levels. The pathogenesis of chronic periodontitis involves a dynamic interplay between pathogenic microorganisms and the host immune system, leading to tissue destruction and significant impacts on both oral and systemic health. Effective management requires a comprehensive approach that addresses both microbial factors and inflammatory responses. Incorporating LLLT as an adjunct to NSPT may offer a synergistic effect, enhancing the overall efficacy of periodontal treatment. Future research and clinical trials are needed to further validate the benefits of LLLT, optimize treatment protocols, and improve patient outcomes. By understanding and integrating advanced adjunctive therapies, dental professionals can better manage chronic periodontitis and mitigate its impact on patient health and quality of life.

Reference

[1] A new classification scheme for periodontal and peri-implant diseases and conditions - Introduction and key changes from the 1999 classification. Caton JG, Armitage G, Berglundh T, et al. J Clin Periodontol. 2018;45 Suppl 20:0–8.

[2] Periodontal and peri-implant wound healing following laser therapy. Aoki A, Mizutani K, Schwarz F, et al. Periodontol 2000. 2015;68:217–269.

[3] Comparative evaluation of the efficacy of diode laser as an adjunct to modified Widman flap surgery for the treatment of chronic periodontitis: a randomized split-mouth clinical trial. Khan F, Chopra R, Sharma N, Agrawal E, Achom M, Sharma P. J Indian Soc Periodontol. 2021;25:213–219.

[4] A comparative evaluation of the efficacy of diode laser as an adjunct to mechanical debridement versus conventional mechanical debridement in periodontal flap surgery: a clinical and microbiological study. Gokhale SR, Padhye AM, Byakod G, Jain SA, Padbidri V, Shivaswamy S. Photomed Laser Surg. 2012;30:598–603.

[5] Effect of diode laser-assisted flap surgery on postoperative healing and clinical parameters: a randomized controlled clinical trial. Jonnalagadda BD, Gottumukkala SN, Dwarakanath CD, Koneru S. Contemp Clin Dent. 2018;9:205–212.

[6] Effect of diode laser as an adjunct to open flap debridement in treatment of periodontitis - a randomized clinical trial. Kolamala N, Nagarakanti S, Chava VK. J Indian Soc Periodontol. 2022;26:451–457.

[7] Assessment of IL-6, IL-8 and TNF-α levels in the gingival tissue of patients with periodontitis. Noh MK, Jung M, Kim SH, et al. Exp Ther Med. 2013;6:847–851.

[8] MMP-8, TRAP-5, and OPG Levels in GCF diagnostic potential to discriminate between healthy patients', mild and severe periodontitis sites. Hernández M, Baeza M, Contreras J, et al. Biomolecules. 2020;10

[9] Effect of periodontal therapy on crevicular fluid interleukin-6 and interleukin-8 levels in chronic periodontitis. Goutoudi P, Diza E, Arvanitidou M. Int J Dent. 2012;2012:362905.

[10] Effect of periodontal surgery on osteoprotegerin levels in gingival crevicular fluid, saliva, and gingival tissues of chronic periodontitis patients. Hassan SH, El-Refai MI, Ghallab NA, Kasem RF, Shaker OG. Dis Markers. 2015;2015:341259.
[11] The effect of periodontal therapy on the level of MMP-8 in patients with chronic periodontitis. Sopi M, Koçani F, Bardhoshi M, Meqa K. Eur J Dent. 2022
[12] Low-level laser therapy induces dose-dependent reduction of TNFalpha levels in acute inflammation. Aimbire F, Albertini R, Pacheco MT, et al. Photomed Laser Surg. 2006;24:33–37.
[13] Inflammatory mediators in periodontal pathogenesis. Huck O, Buduneli N, Bravo D. Mediators Inflamm. 2019;2019:2610184.
[14] Carranza F, Newman M, Takei H, Klokkevold P. p. Philadelphia: Elsevier; 2006. Carranza's Clinical Periodontology; p. 86.
[15] Current status of indices of plaque. Fischman SL. J Clin Periodontol. 1986;13:371-4, 379-80.
[16] Clinical criteria for the definition of "established periodontitis". Machtei EE, Christersson LA, Grossi SG, Dunford R, Zambon JJ, Genco RJ. J Periodontol. 1992;63:206–214.
[17] Comparative evaluation of the efficacy of closed pocket debridement with diode laser and periodontal open flap debridement: a clinical and microbiologic study. Deshmukh K, Shetty D, Shetty A, Bodduru R, Nayyar AS. J Clin Sci. 2018;15:113–122.
[18] Clinical and biochemical effects of diode laser as an adjunct to nonsurgical treatment of chronic periodontitis: a randomized, controlled clinical trial. Saglam M, Kantarci A, Dundar N, Hakki SS. Lasers Med Sci. 2014;29:37–46.
[19] Evaluation of the clinical and biochemical efficacy of erbium, chromium:ytrium-scandium-gallium-garnet (ER,CR:YSGG) laser treatment in periodontitis. Sezen D, Hatipoğlu M, Üstün K. Lasers Med Sci. 2020;35:1567–1575.
[20] Effect of diode laser on the level of il-6 in the gingival crevicular fluid in patients with chronic periodontitis. Bolyarova T, Grozdanova R, Micheva G, Dencheva M, Velikova T. Comptes rendus de I'Acad des Sci. 2019;72:130–135.