Synergistic Effects of Erbium Laser and Non-Surgical Mechanical Debridement on Clinical Metrics and MMP-9 Modulation in Peri-Implant Disease: A Prospective Clinical Study

Abstract

Background
Peri-implantitis is a significant concern in implant dentistry, characterized by inflammation and progressive bone loss around dental implants, with implications for oral health and overall wellbeing. Conventional mechanical debridement often falls short in effectively eliminating biofilms and controlling the associated infectious disease burden. Er,Cr:YSGG laser therapy has been proposed as an adjunctive approach to enhance clinical outcomes and modulate inflammatory mediators, supporting improved disease management and patient health.

Aim
This study aimed to evaluate the synergistic effects of Er,Cr:YSGG laser therapy combined with mechanical debridement on clinical parameters and MMP-9 modulation in peri-implantitis patients.

Methods
A prospective clinical study was conducted on 30 patients with peri-implantitis, randomly assigned to either mechanical debridement alone (control) or mechanical debridement along with Er,Cr:YSGG laser therapy (test). Clinical parameters including PI, GI, PPD, and CBL were assessed at baseline and three months. MMP-9 levels in peri-implant crevicular fluid were analyzed using ELISA. Statistical analysis was performed using SPSS software.

Results
Both groups showed significant improvements in PI, GI, PPD, and MMP-9 levels ($p < 0.05$), with greater reductions in the test group. However, no significant difference in CBL was observed between groups ($p > 0.05$).

Conclusion
Er,Cr:YSGG laser therapy, as an adjunct to mechanical debridement, enhances peri-implant health by improving clinical parameters and reducing inflammatory mediators. Further studies with longer follow-up are warranted to validate these findings.

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INTRODUCTION

Dental implants are a groundbreaking innovation in dentistry, celebrated for their high success rates and ability to restore both the functionality and aesthetics of missing teeth. However, despite their reliability, dental implants are not without risks, as peri-implant diseases can occasionally arise (1). These conditions primarily include peri-implant mucositis and peri-implantitis. Peri-implant mucositis manifests as reversible inflammation restricted to the soft tissues around the implant, while peri-implantitis involves a more severe inflammatory response, leading to bone loss around the implant. If left untreated, peri-implantitis can compromise the implant's stability and potentially result in its failure (2). The host-microbial interaction in peri-implantitis plays a pivotal role in the progression of the disease, as the host immune response is triggered by the pathogenic biofilm on the implant surface (3). Pathogenic microorganisms, such as Porphyromonas gingivalis, Tannerella forsythia, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, and Treponema denticola produce virulence factors that stimulate an inflammatory response (4-6). This response, while aimed at controlling the infection, can result in the release of pro-inflammatory mediators, such as interleukin-1β (IL-1β), tumor necrosis factor-alpha (TNF-α) and matrix metalloproteinases (MMPs) (7). These mediators, in turn, activate osteoclasts and promote bone resorption, contributing to the characteristic bone loss seen in peri-implantitis. Additionally, the implant surface characteristics, such as its roughness, may exacerbate microbial adhesion and immune activation (8). The interplay between microbial virulence and host inflammatory mechanisms is central to the pathogenesis of peri-implantitis, emphasizing the need for therapies targeting both bacterial biofilms and host immune modulation.

MMPs play a significant role in the pathogenesis of peri-implantitis, primarily due to their involvement in the degradation of extracellular matrix components and tissue remodeling. These zinc-dependent endopeptidases are produced by various host cells, including fibroblasts, macrophages, and osteoclasts, in response to inflammatory stimuli and microbial challenges (9). Among the MMPs, MMP-9, also known as gelatinase B, has been particularly implicated in peri-implantitis. MMP-9 is actively involved in the breakdown of type IV collagen, a major component of the basement membrane, facilitating the progression of inflammation and tissue destruction (10). Elevated levels of MMP-9 are frequently detected in peri-implant crevicular fluid (PICF) of affected sites, correlating with the severity of the disease (11,12) This enzyme contributes to the inflammatory cascade by modulating the activity of cytokines and chemokines, further amplifying tissue degradation and bone resorption. Additionally, MMP-9 plays a role in osteoclastic activity, exacerbating bone loss, a hallmark of peri-implantitis (13).

The conventional non-surgical management of peri-implantitis primarily involves mechanical debridement to disrupt and remove the pathogenic biofilm from the implant surface. While this approach can reduce microbial load and inflammation, it often has limitations in achieving complete biofilm removal, particularly on rough implant surfaces and within peri-implant pockets (14). These shortcomings have driven interest in adjunctive therapies, with lasers gaining prominence for their antimicrobial and biomodulatory effects. Among these, the erbium lasers, especially the erbium-chromium-yttrium-scandium-gallium-garnet (Er,Cr:YSGG) laser, have demonstrated significant potential. Er,Cr:YSGG lasers offer precise ablation, effective decontamination, and the ability to modulate the inflammatory response, making them a valuable adjunct to mechanical debridement (15). In this context, the present study aims to explore the synergistic effects of Er,Cr:YSGG laser therapy combined with mechanical debridement, focusing on clinical outcomes and the modulation of MMP-9 levels.

MATERIALS AND METHODS

Study Setting

This prospective clinical study was conducted at the Department of Periodontology, Saveetha Dental College and Hospitals, Chennai, India, and included 30 participants aged 25 to 50 years diagnosed with peri-implantitis (16). Participants were recruited based on the following inclusion criteria: the presence of at least one dental implant diagnosed with peri-implantitis, systemic health, no history of immunocompromised conditions or uncontrolled systemic diseases, and no periodontal or antibiotic therapy within the preceding six months. All implants used in the study were commercially available, placed at the bone level, and featured a platform-switched connection. After a 3-month healing period following implant placement, the implants were restored with cement-retained, implant-supported porcelain-fused-to-metal prosthetic restorations. Exclusion criteria included smoking, pregnancy or lactation, recent antibiotic use (within the last six months), active or previous periodontitis, and other systemic conditions that could interfere with wound healing or implant outcomes. All participants provided written informed consent after the study procedures were explained. The study protocol adhered to the Helsinki Declaration guidelines and was approved by the Saveetha University Ethical Committee. Participants were randomly assigned into two groups: Group 1 (control group): received mechanical debridement alone (n=15); group 2 (test group): received mechanical debridement combined with Er,Cr:YSGG laser therapy (n=15). Sample size calculation was performed using G*Power Software (Version 3.0), based on the mean and standard deviation of a previous study (17), with an alpha value of 0.05 and a statistical power of 80%, yielding a required sample size of 30 participants.

Clinical Procedures

Mechanical debridement was performed using a sterile titanium curette, followed by irrigation of peri-implant pockets with 0.12% chlorhexidine gluconate solution in both groups. After debridement, the test group received Er,Cr:YSGG laser therapy on the same day. The laser, set to 2780 nm, 1.5 W power, 20 Hz frequency, 50% water, and 40% air, was applied with a radial firing tip in sweeping motions along the implant surface and surrounding tissues to ensure thorough decontamination. All procedures were conducted by the same operator (AJ) to ensure consistency.

Clinical and Radiographic Assessments

Clinical measurements, including plaque index (PI), gingival index (GI), and peri-implant probing depth (PPD), were assessed using standard methods. PI and GI were measured using the Silness and Loe index, averaging scores from four implant surfaces. PPD was recorded at six sites, and the average depth was calculated. For radiographic measurements, digital periapical radiographs were taken using the paralleling angle technique. Crestal bone loss (CBL) was assessed by measuring the distance from the implant platform to the highest coronal bone level at mesial and distal sites, with the average distance recorded.

Assessment of MMP-9 levels

Sterile cotton was used to isolate the implant sites, and supragingival plaque was removed with curettes. Peri-implant crevicular fluid (PICF) was collected using microcapillary pipettes and stored at -20°C for analysis. MMP-9 levels were measured using the Human MMP-9 ELISA Kit (Elabscience®, US). The ELISA kit employed the Sandwich-ELISA principle. The micro ELISA plate provided in the kit had been pre-coated with an antibody specific to Human MMP-9. Standards or samples were added to the wells of the micro ELISA plate, where they were incubated with the specific antibody. Subsequently, a biotinylated detection antibody specific to Human MMP-9 and Avidin-Horseradish Peroxidase (HRP) conjugate were added to each well and incubated. Free components were washed away, and substrate solution was added to the wells. Only the wells containing Human MMP-9, biotinylated detection antibody, and Avidin-HRP conjugate turned blue. The enzyme-substrate reaction was terminated by the addition of a stop solution, causing the color to change to yellow. The optical density (OD) was measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The OD value was proportional to the concentration of Human MMP-9. Results were expressed in ng/mL, with a detection range of 0.16-10 ng/mL.

Clinical, radiographic evaluations, and PICF collection were conducted at baseline and again at the 3-month follow-up by the same examiner (AR).

Statistical Analysis

The data were processed using the Statistical Package for the Social Sciences (SPSS Software, Version 23.0; IBM Corp., Armonk, NY, USA). The Shapiro-Wilk and Kolmogorov-Smirnov tests confirmed a parametric distribution. Comparisons of age, PI, GI, PPD, CBL, and MMP-9 levels between the two groups were performed using an independent t-test. Gender distribution was evaluated with the Chi-square test. Intragroup comparisons were carried out using a paired t-test. A p-value of less than 0.05 was considered to be statistically significant.

RESULTS

Group 1 included 7 males and 8 females, with an average age of 46.5 ± 3.72 years, while Group 2 comprised 8 males and 7 females, with an average age of 45.1 ± 3.91 years. No significant differences were found between the two groups regarding age and gender, with p-values of 0.79 and 0.82, respectively.

On intergroup comparison, at baseline, there were no significant differences between the control and test groups across all parameters, indicating comparability. However, after three months, the test group showed significantly greater reductions in PI, GI, PPD, and MMP-9 levels ($p = 0.00$), suggesting superior improvements compared to the control group. CBL remained statistically unchanged between groups ($p > 0.05$). These findings indicate that the test intervention effectively enhances peri-implant health over time (Table 1).

Variable	Baseline Group 1 (Control)	Baseline Group 2 (Test)	p value (Baseline)	3-month Group 1 (Control)	3-month Group 2 (Test)	p value (3 months)
Plaque Index (PI)	2.56 ± 0.21	2.54 ± 0.26	0.82	1.71 ± 0.06	0.53 ± 0.12	0.00*
Gingival Index (GI)	2.44 ± 0.29	2.42 ± 0.32	0.86	1.23 ± 0.09	0.46 ± 0.12	0.00*
Peri-implant Pocket Depth (PPD)	4.98 ± 0.26	4.89 ± 0.32	0.41	2.79 ± 0.19	1.61 ± 0.24	0.00*
Crestal Bone Loss (CBL)	0.41 ± 0.05	0.42 ± 0.04	0.55	0.42 ± 0.03	0.43 ± 0.04	0.45
MMP-9 Levels (ng/mL)	7.15 ± 0.45	6.97 ± 0.66	0.39	3.76 ± 0.28	1.81 ± 0.14	0.00*
*Statistically significant						

Tab. 1 Intergroup comparison between both groups at baseline and 3 months

Variable	Baseline Group 1 (Control)	3-month Group 1 (Control)	p value (Group 1)	Baseline Group 2 (Test)	3-month Group 2 (Test)	p value (Group 2)
Plaque Index (PI)	2.56 ± 0.21	1.71 ± 0.06	0.00*	2.54 ± 0.26	0.53 ± 0.12	0.00*
Gingival Index (GI)	2.44 ± 0.29	1.23 ± 0.09	0.00*	2.42 ± 0.32	0.46 ± 0.12	0.00*
Peri-implant Pocket Depth (PPD)	4.98 ± 0.26	2.79 ± 0.19	0.00*	4.89 ± 0.32	1.61 ± 0.24	0.00*
Crestal Bone Loss (CBL)	0.41 ± 0.05	0.42 ± 0.03	0.52	0.42 ± 0.04	0.43 ± 0.04	0.51
MMP-9 Levels (ng/mL)	7.15 ± 0.45	3.76 ± 0.28	0.00*	6.97 ± 0.66	1.81 ± 0.14	0.00*
*Statistically significant						

Tab. 2 Intragroup comparison of both groups from baseline to 3 months

On intragroup comparison, both groups showed significant improvements in PI, GI, PPD, and MMP-9 levels from baseline to three months ($p = 0.00$), indicating that both interventions contributed to improved clinical and biochemical outcomes. However, the magnitude of reduction was more pronounced in the test group, suggesting greater efficacy. CBL remained statistically unchanged in both groups ($p > 0.05$). These results reinforce the superior clinical benefits of the test intervention compared to the control (Table 2).

DISCUSSION

The present study aimed to assess the combined effects of Er,Cr:YSGG laser therapy and non-surgical mechanical debridement in the management of peri-implantitis by evaluating clinical parameters and MMP-9 modulation. Findings indicated that the combination therapy significantly improved PI, GI, PPD, and MMP-9 levels compared to mechanical debridement alone, demonstrating the superior efficacy of laser-assisted treatment in controlling peri-implant inflammation.

Mechanical debridement remains a fundamental approach in peri-implantitis treatment, primarily focusing on disrupting pathogenic biofilms and reducing microbial load. However, conventional methods often fail to achieve complete decontamination due to the intricate microtopography of implant surfaces, which facilitates bacterial adherence (18). The present study observed a notable reduction in PI and GI across both groups, suggesting that mechanical debridement effectively minimizes biofilm accumulation and inflammation. Nevertheless, the greater reduction in the test group underscores the additional benefits of laser therapy in enhancing biofilm removal and mitigating inflammatory responses. Several clinical studies have reported that erbium lasers significantly reduce inflammation in peri-implantitis sites (19-22). Schwarz F et al. (19), demonstrated effective subgingival calculus removal and a reduction in bleeding on probing at three and six months following erbium-doped:yttrium, aluminum, and garnet (Er:YAG) laser treatment for peri-implantitis (19). Additionally, when comparing air-abrasive treatment and erbium laser monotherapy for severe peri-implantitis, a greater reduction in bleeding on probing and suppuration was observed in the laser-treated group (20). Research has also highlighted that erbium lasers effectively ablate both soft and hard tissue, including bacterial biofilm and calculus, without causing thermal damage to adjacent structures (21). Furthermore, a randomized controlled trial evaluating erbium laser irradiation for regenerative surgical therapy of peri-implant defects showed superior reductions in plaque index, gingival index, and an increase in bone gain over time (22). The results of the present study align with these findings.

Another key observation in this study was the significant decrease in PPD in the test group compared to the control, a clinically relevant outcome as deeper peri-implant pockets contribute to bacterial colonization and disease progression. The photothermal and ablative properties of Er,Cr:YSGG lasers facilitate bacterial reduction and decontamination, supporting tissue healing and pocket depth reduction. Additionally, the laser's biomodulatory effects, such as enhanced fibroblast proliferation and collagen synthesis (23), likely contributed to improved soft tissue response and peri-implant health stabilization. Previous studies have demonstrated that erbium laser treatment leads to reductions in Fusobacterium spp. and enhanced pocket depth reduction in peri-implant defects (24), and effective removal of granulation tissue, calcified deposits, and biofilm from implant surfaces (25). Lin T et al. (26), highlighted that reduction in pocket depth and gain in bone height was noted when erbium lasers were used alongside regenerative therapy in peri-implantitis defect sites. Additionally, the combination of erbium laser treatment with bone grafting has been shown to enhance probing pocket depth reduction and peri-implant defect fill after one year (27). Furthermore, a recent systematic review suggested that laser treatment may promote bone gain and reduce both bleeding on probing and PPD in peri-implantitis (28). The improvements in clinical parameters in this study are consistent with previous literature, reinforcing the effectiveness of laser therapy combined with mechanical debridement for peri-implant management. However, despite these positive outcomes, no significant differences in CBL

were observed between the groups. This could be due to the lack of radiographic bone gain, as the study focused on cases with horizontal bone loss, where regenerative interventions such as bone grafting or guided tissue regeneration were not applied.

MMP-9 plays a crucial role in extracellular matrix degradation and is closely linked to peri-implant inflammation and tissue destruction. Elevated MMP-9 levels are associated with disease severity due to their role in breaking down basement membranes and promoting osteoclastic activity. This study found a significant reduction in MMP-9 levels in both groups, with a more pronounced decrease in the test group. These findings suggest that Er,Cr:YSGG laser therapy may offer additional anti-inflammatory benefits by regulating proteolytic enzyme activity (29), thereby limiting tissue destruction and promoting peri-implant tissue stability. A study assessing protein biomarker changes and bacterial profiles after surgical reconstructive therapy of peri-implantitis demonstrated that Er:YAG laser use significantly reduced bacterial load and modulated the inflammatory response by lowering MMP-9 and VEGF levels during the post-surgical period (30). Another study examining the impact of laser-treated healing abutments on peri-implant connective tissue and extracellular matrix proteins found that tissue adjacent to laser-treated surfaces exhibited significantly lower MMP levels ($p<0.05$) and higher TIMP levels ($p<0.05$) compared to tissue surrounding conventional machined surfaces (31). The reduction in MMP-9 in the present study aligns with prior research demonstrating that laser therapy can suppress inflammatory mediators and promote wound healing.

Overall, the findings of this study support the adjunctive use of lasers in peri-implantitis management. Er,Cr:YSGG lasers have been extensively investigated for their ability to selectively decontaminate bacterial biofilms while preserving implant surfaces. Their ability to enhance cellular responses and regulate inflammation further underscores their utility as an adjunctive treatment in peri-implant therapies. However, discrepancies in laser parameters, treatment protocols, and follow-up durations across studies necessitate further research to establish standardized clinical guidelines for optimal outcomes.

Certain limitations of this study should be acknowledged. The three-month follow-up period provides valuable insight into the immediate effects of the interventions but does not capture long-term peri-implant health or disease recurrence. Future studies with extended follow-up periods are needed to evaluate the durability of treatment outcomes. Additionally, while MMP-9 serves as a key biomarker of inflammation, incorporating additional inflammatory markers could provide a more comprehensive understanding of the molecular mechanisms underlying peri-implantitis resolution. Histological analyses could also further elucidate tissue responses and healing processes following laser therapy.

CONCLUSION

The results of this study suggest that Er,Cr:YSGG laser therapy, when used as an adjunct to mechanical debridement, significantly enhances peri-implant health by improving clinical parameters and reducing MMP-9 levels. The superior efficacy of laser therapy in modulating inflammation and facilitating tissue healing underscores its potential as a valuable non-surgical treatment modality for peri-implantitis. Further longitudinal studies with larger sample sizes and additional biochemical markers are necessary to validate these findings and optimize peri-implantitis management protocols.

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