

Comparative Evaluation Of Patients Suffering From Chronic Periodontitis Treated With Open Flap Debridement With And Without Adjunctive Laser Therapy: A Clinical And Microbiological Study

Abstract

Aim: This study aimed to determine the number of edentulous residual ridges in stone casts of individuals with full dentures and to analyze the relationship between these ridges and mouth openness.

Methods: The sample consisted of eighty study casts, forty of which were upper and forty of which were lower. Measurements were taken with the assistance of a dry-point compass and a transparent ruler. The measurements for maximum mouth opening (MMO) were collected from volunteers who were instructed to open their mouths to their furthest extent until there was no more room to open. Utilizing a Boley gauge, the distance measured from the edge of the upper lip to the edge of the lower lip was determined.

Results: Correlations between mouth opening and arch size (length and width) were verified using Spearman correlation and simple linear regression. Individuals with larger maxillary and mandibular alveolar ridges had wider mouth openings.

Conclusion: A significant correlation was found between arch size (length and width) and mouth opening.

Keywords

Alveolar ridge dimensions; Complete dentures; Edentulous residual ridge; Maximum mouth opening; Oral morphometry; Prosthodontics.

Authors

Dr Sameer Chaudhary¹

Designation: Post-Graduate Student
Department of Periodontology and Oral Implantology, ITS Dental College & Hospital, 47, Knowledge Park III, Greater Noida, Uttar Pradesh, India
Email id : chaudharysameer177@gmail.com
ORCID ID- 0009-0007-6610-6720

Dr Gunjan Gupta²

Designation : Professor & Head
Department of Periodontology and Oral Implantology ITS Dental College & Hospital, 47, Knowledge Park III, Greater Noida, Uttar Pradesh, India
Email id : drgunjangupta22@gmail.com
ORCID ID- 0000-0001-9436-6895

Dr Shivesh Kumar Mishra^{3*}

Professor, Department of Periodontology and Oral Implantology. ITS Dental College & Hospital, 47, Knowledge Park III, Greater Noida, Uttar Pradesh, India
Email id : mishrashivesh@gmail.com
ORCID ID- 0000-0001-5264-1027

Dr Nishant Gupta⁴

Professor & Head, Department of Orthodontics & Dentofacial orthopedics, Teerthankar Mahaveer University, Moradabad
Email: nishantgupta2001@gmail.com
ORCID 0000-0002-1791-7224

Dr Kumar Saurav⁵

Professor, Santosh Dental College Ghaziabad, Uttar Pradesh, India
Email id: drsauravsingh4@gmail.com
ORCID 0000-0003-1334-523

Dr Mohit Bhatnagar⁶

Reader, Department of Prosthodontics, crown & Bridge & Oral Implantology
ITS Dental College & Hospital, 47, Knowledge Park III, Greater Noida, Uttar Pradesh, India
dr.mohitbhatnagar93@gmail.com
ORCID 0000-0001-7541-904X

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INTRODUCTION

Chronic periodontitis is a common disease that affects the gingiva and the tissues that support the teeth. If left untreated, it can lead to tooth mobility and tooth loss¹. The initiation of the disease is due to the presence of harmful bacteria in the subgingival plaque. However, the tissue destruction depends on how the body reacts to these bacteria². The interaction between bacteria and the host immune response leads to inflammation, periodontal pocket formation, loss of attachment, and destruction of alveolar bone³.

The main aim of periodontal treatment is to remove plaque, calculus, and bacteria from the root surfaces of teeth⁴. In early stages of the disease, non-surgical periodontal therapy is often effective. But, in moderate to severe chronic periodontitis, completely removing the bacteria may not be possible because of causes such as deep pockets, furcations involvements, and various multiple root anatomy⁵. In such conditions, surgical procedures like open flap debridement (OFD) are recommended. OFD is suitable for direct visibility and access to the root surfaces and bony defects, making thorough debridement possible and promotes better healing⁶.

Many studies have shown that OFD leads in decreasing the probing pocket depth and improves clinical attachment levels⁷. However, some bacteria may still remain in deeper tissues or areas that are inaccessible even after surgery⁸. These remaining bacteria may lead to delayed healing and increase in chances of disease recurrence⁹. Because of this, additional treatment methods have been studied to improve the results of periodontal surgery.

Lasers have been introduced in periodontal therapy because of their ability to reduce bacteria, detoxify root surfaces, and support wound healing¹⁰. Different types of lasers such as diode, Nd: YAG, and Er: YAG lasers have been used along with conventional periodontal treatment¹¹. Laser therapy has been reported to reduce inflammation, decrease bacterial levels, and improve clinical outcomes by selectively acting on diseased tissues while preserving healthy tissues¹².

The term LASER stands for “Light Amplification by Stimulated Emission of Radiation” and was introduced by Gordon Gould in 1959.¹³ In dentistry, laser use has

increased over the past few decades, especially after the development of low-level laser therapy, which is known for its role in pain reduction and wound healing.

Laser-based dental treatments are generally well accepted by patients because they are less invasive, cause less bleeding, and are associated with less discomfort.

The progression of periodontal disease is influenced by bacterial growth, the host inflammatory response, and individual risk factors.

Low-level laser therapy has shown anti-inflammatory effects by reducing inflammatory mediators such as nuclear factor kappa B, prostaglandin E₂, tumor necrosis factor alpha, cyclooxygenase-2, and interleukin-1 β ¹⁴. Several studies have also reported reductions in pocket depth and inflammatory markers following laser application.

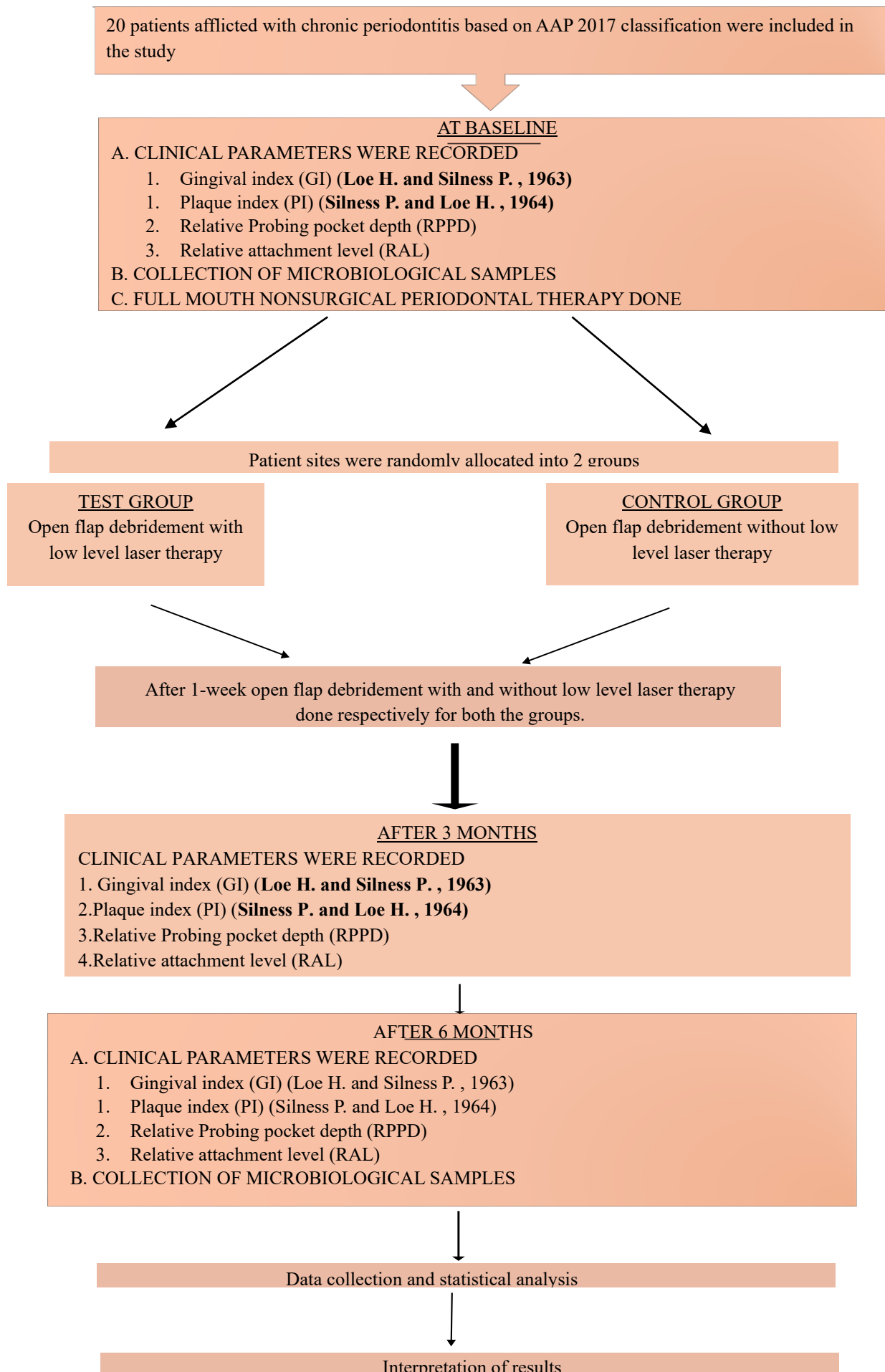
Considering the ongoing debate regarding the benefits of lasers in periodontal surgery, the present study was conducted to compare open flap debridement alone and open flap debridement with adjunctive diode laser therapy in patients with chronic periodontitis.

MATERIALS AND METHODS

This study was randomized controlled clinical trial carried out on patients visiting the Department of Periodontology and oral implantology and seeking dental implant treatment from I.T.S Dental College, Hospital & Research Centre, Greater Noida, Uttar Pradesh were selected. The protocol for the study was reviewed and approved by the Institutional Ethical Committee. (Ref. No. IEC/Perio/04/24) . The Inclusion Criteria –Patients with chronic periodontitis as per 2017 AAP classification, age group 18-50 years of both genders, probing depth > or equal to 4mm and < or equal to 7mm, Presence of atleast > or equal to 20 natural teeth in both upper and lower arches. The exclusion criteria was Subjects who smoke, Pregnant and lactating females, with history of uncontrolled systemic diseases affecting the periodontium, received surgical or non-surgical periodontal therapy 6 months prior to study.

STUDY METHODS/TOOLS

BRIEF STUDY OUTLINE:



Clinical Procedure

All procedures were carried out by a single, experienced investigator who was trained and had received standardization in the clinical with similar patient.



FIGURE 6: COLLECTING PLAQUE SAMPLE AT BASELINE



FIGURE 7: POCKET DEPTH MEASURED AT BASELINE



FIGURE 8: SULCULAR INCISION GIVEN



FIGURE 9: FLAP RAISED AND DEGRANULATION DONE IN CONTROL GROUP



FIGURE 10: LOW LEVEL LASER THERAPY APPLIED AFTER OPEN FLAP DEBRIDEMENT IN TEST GROUP

**FIGURE 11: SUTURES APPLIED****FIGURE 12: COE PACK PLACED**

RESULTS

Changes in clinical and microbiological parameters were then analyzed within and between the two groups over the study period.

Microbiological Assessment

Subgingival plaque samples were collected using sterile curettes from the deepest pocket in all quadrants. The samples were processed for colony-forming unit (CFU) counts, focusing specifically on *Porphyromonas gingivalis*, a key pathogen in chronic periodontitis. Microbiological samples were collected at baseline and 6 months post-treatment to assess changes in bacterial load.

Interpretation

Both groups showed improvements in gingival health and periodontal attachment, which means that open flap debridement (OFD) alone works to reduce inflammation and help tissues heal.

The test group that got diode laser treatment had better results in pocket depth, attachment level, and better gingival health.

The number of *P. gingivalis* bacteria went down more in the laser group, showing that diode lasers can kill these harmful bacteria in the gums.

The improvements in gum health matched the reduction in bacteria, which suggests that less bacteria leads to better healing.

Measuring at baseline, 3 months, and 6 months showed that healing continues over time.

The present study was conducted at ITS Dental college and hospital Greater Noida to compare the effects of low level laser therapy in patients suffering from Chronic Periodontitis treated with Open Flap Debridement with and without adjunctive laser therapy. A total of 20 patients were taken as per the inclusion criteria comprising of 40 sites which were included in the study. The subjects were randomly allocated into test group and control group using SPSS software with 20 sites in each group. Patients were screened for Periodontal diseases both gingivitis and Periodontitis as per the AAP Classification of 2017

Following results were found:

Table 1: Frequency distribution n (%) of patients by their Age

Age Group	n	(%)
18-34 years	8	40%
35-52 years	10	50%
53-65 years	2	10%

INTERGROUP COMPARISON

Intergroup comparisons were performed for Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), Bleeding Index (BI), and Relative Attachment Level (RAL), while microbial analysis was carried out by assessing colony counts at baseline, 3 months, and 6 months to evaluate the effect of Open Flap Debridement (OFD) with and without adjunctive low-level laser therapy (LLLT) in patients with chronic periodontitis.

Table 2: Intergroup plaque index comparison between test group and control group respectively

Group	0 (0%)	1 (5%)	2 (10%)	3 (15%)	4 (20%)
Group 1 (OFD)	0 (0%)	0 (0%)	0 (0%)	4 (20%)	16 (80%)
Group 2 (OFD + LLLT)	0 (0%)	0 (0%)	0 (0%)	6 (30%)	14 (70%)
Chi square test value = 0.533, p=0.465 (no significant difference)					

PI score – 3 month	Mild (0.1-1) N (%)	Moderate (1.1-2) N (%)	Severe (2.1 – 3) N (%)
Group 1 (OFD)	5 (25%)	13 (65%)	2 (10%)
Group 2 (OFD +LLLT)	8 (40%)	11 (55%)	1 (5%)
	Chi square test value = 3.412, p=0.043* (significant difference)		
PI score – 6 month	Mild (0.1-1) N (%)	Moderate (1.1-2) N (%)	Severe (2.1 – 3) N (%)
Group 1 (OFD)	11 (55%)	9 (45%)	0 (0%)
Group 2 (OFD +LLLT)	15 (75%)	5 (25%)	0 (0%)
	Chi square test value = 5.121, p=0.031* (significant difference)		

Table 3: Intergroup pocket depth score comparison between test group and control group respectively

Group 1 (OFD)	5 (25%)	8 (40%)	7 (35%)
Group 2 (OFD +LLLT)	2 (10%)	11 (55%)	7 (35%)
	Chi square test value = 1.759, p=0.415 (no significant difference)		
Pocket depth - 3 month	4-5 mm N (%)	6-7 mm N (%)	≥7mm N (%)
Group 1 (OFD)	7 (35%)	13 (65%)	0 (0%)
Group 2 (OFD +LLLT)	11 (55%)	9 (45%)	0 (0%)
	Chi square test value = 1.61 p=0.126 (no significant difference)		
Pocket depth -6 month	4-5 mm N (%)	6-7 mm N (%)	≥7mm N (%)
Group 1 (OFD)	15 (75%)	5 (25%)	0 (0%)
Group 2 (OFD +LLLT)	16 (80%)	4 (20%)	0 (0%)
	Chi square test value = 2.987 p=0.101 (no significant difference)		

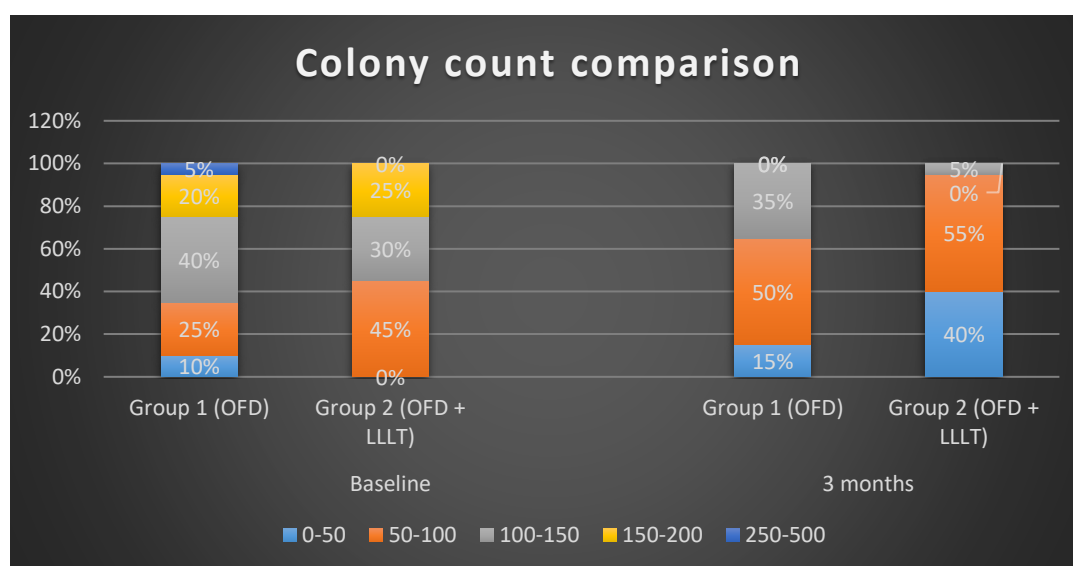
Table 4: Intergroup relative attachment loss (RAL) comparison between test group and control group respectively

Group 1 (OFD)	1 (5%)	14 (70%)	5 (25%)
Group 2 (OFD +LLLT)	3 (15%)	14 (70%)	3 (15%)
	Chi square test value = 1.5, p=0.472 (no significant difference)		
RAL -3 month	4-5 mm N (%)	6-7 mm N (%)	≥7mm N (%)
Group 1 (OFD)	10 (50%)	10 (50%)	0 (0%)
Group 2 (OFD +LLLT)	14 (70%)	6 (30%)	0 (0%)
	Chi square test value = 1.667, p=0.197 (no significant difference)		
RAL -6 month	4-5 mm N (%)	6-7 mm N (%)	≥7mm N (%)
Group 1	16 (80%)	4 (20%)	0 (0%)

(OFD)			
Group 2 (OFD +LLLTT)	18 (90%)	2 (10%)	0 (0%)
	Chi square test value = 3.109, p=0.048* (significant difference)		

Table 5: Intergroup colony count comparison between test group and control group respectively

Group 1 (OFD)	2 (10%)	5 (25%)	8 (40%)	4 (20%)	1 (5%)
Group 2 (OFD +LLLTT)	0 (0%)	9 (45%)	6 (30%)	5 (25%)	0 (0%)
Chi square test value = 4.540, p=0.338 (no significant difference)					
Colony count - 6 months	0-50 N (%)	50-100 N (%)	100-150 N (%)	150-250 N (%)	250-500 N (%)
Group 1 (OFD)	3 (15%)	10 (50%)	7 (35%)	0 (0%)	0 (0%)
Group 2 (OFD +LLLTT)	8 (40%)	11 (55%)	1 (5%)	0 (0%)	0 (0%)
Chi square test value = 6.820, p=0.033* (significant difference)					

**Graph1: Intergroup comparison of colony counts**

DISCUSSION

This study compared open flap debridement alone with open flap debridement combined with diode laser therapy in patients with chronic periodontitis. Chronic periodontitis is a complex disease caused by an imbalance of bacteria in the subgingival area and an exaggerated host immune response¹⁵. Conventional surgical therapy like OFD provides direct access to root surfaces and bone defects, which allows thorough removal of bacterial deposits, inflamed tissue, and calculus. In this study, both treatment groups showed significant improvements in clinical parameters, such as reduced probing pocket depth and increased clinical attachment levels, which is consistent with earlier studies showing that surgical access helps resolve inflammation and improve tissue healing¹⁶. Patients treated with adjunctive diode laser therapy showed greater improvements in both clinical and

microbiological outcomes, indicating that diode lasers offer additional benefits beyond mechanical debridement⁵⁵.

The microbiological results of this study showed a significant reduction in *P. gingivalis* counts in the laser group compared with OFD alone. *P. gingivalis* is a Gram-negative rod, anaerobic bacterium which is recognized as a key pathogen in periodontitis because it disrupts the balance of oral bacteria and triggers tissue-damaging inflammation¹⁷. The reason *P. gingivalis* was selected as it is the main causative periodontal pathogen, it produces several virulence factors, including gingipains, fimbriae and lipopolysaccharides, which allow tissue invasion, immune evasion, and destruction of extracellular matrix components¹⁸. This bacterium can also interfere with host immune responses by affecting Toll-like receptor and

complement pathways, leading to chronic but ineffective inflammation¹⁹. Because of its pigmented nature, *P. gingivalis* is particularly sensitive to diode lasers, which target pigmented bacteria and generate heat that can destroy bacterial cell walls and biofilms²⁰

The Plaque was obtained through sub gingival samples using a sterile curette and subjected through CFU samples.

The reduction of *P. gingivalis* observed in this study agrees with previous research. Khandekar et al. reported that adjunctive 980 nm diode laser therapy with OFD led to greater reductions in *P. gingivalis* than OFD alone.²¹ Abdullah et al. also found that diode laser therapy together with scaling and root planning resulted in reduced *P. gingivalis* more than non-surgical therapy at 1- and 3-month follow-ups¹⁹. Üstün et al. reported significant decreases in *P. gingivalis* and other red complex bacteria following diode laser use compared with mechanical therapy alone. These studies support the findings of the present research and suggest that diode lasers can effectively reduce key periodontal pathogens.

Other clinical studies also showed that diode lasers reduce multiple periodontopathogens. Üstün et al. demonstrated reductions in *Tannerella forsythia* and *Treponema denticola* after diode laser therapy. In vitro studies have also confirmed that diode lasers can reduce pigmented anaerobic bacteria in biofilms, providing a biological explanation for their clinical effectiveness.²⁰

In addition to reducing bacteria, diode lasers may also influence the host's inflammatory response.

Gonçalves et al.²⁰ found reduced levels of inflammatory substances such as TNF- α and IL-1 β in gingival tissues after diode laser therapy²¹. Although the present study did not measure these cytokines directly, the greater improvements in bleeding on probing and pocket depth in the laser group may reflect both reduced bacterial load and decreased local inflammation.

Some studies, however, have reported mixed results. Sgolastra et al.²¹ reviewed diode laser-assisted therapy and found that while laser use often improved clinical outcomes, results varied widely due to differences in laser type, settings, protocols, and follow-up periods²². Ozturk et al. found that 980 nm diode laser therapy reduced clinical inflammation but did not produce statistically significant differences in all clinical parameters compared with conventional therapy alone²³. These findings highlight the need for standardized laser protocols to achieve consistent results.^{24,25}

This study had some limitations. The number of patients included in this study was small. The 6-month follow-up allowed assessment of short-term improvements, but longer-term outcomes and microbial recolonization were not evaluated. Only *P. gingivalis* was analyzed, and a complete assessment of the entire subgingival microbiome was not performed. Histological evaluation was not done, so true regeneration of periodontal tissues cannot be confirmed. Patient-centered outcomes such as

pain, quality of life, and cost-effectiveness were also not assessed, and these are areas for future research.

Conclusion

In conclusion, the findings suggest that adjunctive diode laser therapy combined with OFD results in better clinical and microbiological outcomes, particularly in reducing *P. gingivalis*, compared with OFD alone. Comparisons with previous studies indicate that diode lasers can enhance microbial suppression and may improve healing. However, variability in protocols and the need for standardized techniques remain challenges. Future long-term randomized studies with larger sample sizes and comprehensive microbial and host response analysis are needed to establish clear clinical guidelines for the use of diode lasers in periodontal therapy

REFERENCES

1. The tendency of MMO (mm) to increase with the length, width, and size of the arches with denture prostheses was evident, as shown in Figure 3, which displays scatter and linear regression diagrams. This relationship was observed to be si
2. Newman MG, Takei HH, Klokkevold PR, Carranza FA. Carranza's Clinical Periodontology. 13th ed. Elsevier; 2019. p. 64–67.
3. Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. Periodontol 2000. 1997;14:9–11.
4. Offenbacher S. Periodontal diseases: pathogenesis. Ann Periodontol. 1996;1(1):821–878.
5. Lindhe J, Lang NP, Karring T. Clinical Periodontology and Implant Dentistry. 6th ed. Wiley-Blackwell; 2015. p. 574–576.
6. Waerhaug J. Healing of the dento-epithelial junction following subgingival plaque control. J Periodontol. 1978;49(3):119–134.
7. Kirkland O. The role of periodontal surgery. J Periodontol. 1952;23:16–21.
8. Becker W, Becker BE, Berg L. Periodontal treatment without maintenance. J Periodontol. 1984;55(9):505–509.
9. Slots J, Rams TE. New views on periodontal microbiota in special patient categories. J Clin Periodontol. 1991;18:411–420.
10. Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease. J Periodontol. 1992;63(4 Suppl):322–331.
11. Cobb CM. Lasers in periodontics: a review of the literature. J Periodontol. 2006;77(4):545–564.
12. Schwarz F, Aoki A, Becker J, Sculean A. Laser application in non-surgical periodontal therapy. J Clin Periodontol. 2008;35(8 Suppl):29–44.
13. Romanos GE, Weitz D. Therapy of peri-implant diseases. Where is the evidence? J Evid Based Dent Pract. 2012;12(3 Suppl):204–208.
14. Kreisler M, Haj H, d'Hoedt B. Temperature changes induced by diode laser irradiation. Lasers Surg Med. 2002;30:153–159.
15. Slot DE, Jorritsma KH, Cobb CM, Van der Weijden FA. The effect of laser therapy as an adjunct to non-

- surgical periodontal treatment. *J Clin Periodontol*. 2014;41:681–692.
16. Lindhe J, Lang NP, Karring T. *Clinical Periodontology and Implant Dentistry*. 6th ed. Oxford: Wiley-Blackwell; 2015. p. 574–610.
 17. Cobb CM. Lasers in periodontics: a review of the literature. *J Periodontol*. 2006;77(4):545–564.
 18. Holt SC, Ebersole JL. *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia*: The “red complex,” a prototype polybacterial pathogenic consortium in periodontitis. *Periodontol* 2000. 2005;38:72–122.
 19. Lamont RJ, Jenkinson HF. Life below the gum line: Pathogenic mechanisms of *Porphyromonas gingivalis*. *Microbiol Mol Biol Rev*. 1998;62 (4):1244–1263.
 20. Hajishengallis G. Immune evasion strategies of *Porphyromonas gingivalis*. *J Oral Biosci*. 2011;53(3):233–240.
 21. Gonçalves PF, De Oliveira RR, Novaes AB Jr, et al. Effect of diode laser therapy on inflammatory mediators in periodontal tissues. *J Periodontol*. 2010;81(12):1829–1837
 22. Moritz A, Schoop U, Goharkhay K, et al. Treatment of periodontal pockets with a diode laser. *Lasers Surg Med*. 1998;22(5):302–311.
 23. Sgolastra F, Severino M, Gatto R, Monaco A. Effectiveness of diode laser as an adjunct to periodontal therapy: A systematic review. *J Dent*. 2014;42(2):156–164.
 24. Khandekar S, Prakash S, Vyas T, et al. Effect of adjunctive diode laser therapy on *Porphyromonas gingivalis* in chronic periodontitis patients treated with open flap debridement. *J Lasers Med Sci*. 2017;8(4):182–187.
 25. Abdullah M, Al-Kheraif AA, Al-Habashneh R. Effect of diode laser adjunct to scaling and root planing on periodontal pathogens. *Photomed Laser Surg*. 2016;34(12):577–583.
 26. Öztürk G, Akyüz S, Erdemci B. Evaluation of the clinical effects of 980 nm diode laser as an adjunct to periodontal therapy. *Lasers Med Sci*. 2015;30(2):693–700.
 27. Üstün K, Erciyas K, Sezer U, et al. Clinical and microbiological effects of diode laser as an adjunct to non-surgical periodontal treatment. *Lasers Med Sci*. 2013;28(4):1259–1266.
 28. Üstün K, Erciyas K, Sezer U, et al. Effects of diode laser application on periodontal pathogens. *Lasers Med Sci*. 2014;29(2):613–620.