

Efficacy of Low Level Laser Therapy in Periodontal Surgery-An Original Research

**Dr. Abdul Rahman Khan Mohammed¹, Dr. Nazia Ali,² Dr. Lakshmi Vineela Yenikepati³,
Dr. Yash Pal Singh⁴, Dr. Ashank Mishra⁵, Dr. Sapnil Gaidhankar⁶**

¹Graduate Student, Department of BioHealth Informatics, Indiana University-Purdue University Indianapolis (IUPUI), USA.

²MDS, Consultant Conservative Dentist and Endodontist, Private Practitioner, House No-13, Sector A, Gulbahar Colony, Srinagar, India.

³BDS, MS, Rutgers biomedical and health sciences ,Newark, New Jersey, USA.

⁴BDS, MDS, PhD, Assistant Professor, Department of Restorative and Prosthetic dental Sciences, College of Dentistry, Dar Al Uloom university, Riyadh, KSA.

⁵Reader, Dept of Periodontics and implantology, RVS dental college and hospital, Coimbatore, Tamil Nadu

⁶Senior Lecturer, Department of Periodontology, Nanded Rural Dental College & Research Center, Nanded, Maharashtra, India.

Corresponding Author: Dr. Abdul Rahman Khan Mohammed, Graduate Student, Department of BioHealth Informatics, Indiana University-Purdue University Indianapolis (IUPUI), USA.

rahmanmohd0311@gmail.com

Abstract

Introduction: Lasers are the recent applications for the various procedures in dentistry. Hence in the present study we aim to evaluate the consequence of low-level laser therapy (LLLT) as an adjunct to periodontal therapy of those with the tobacco habits and having moderate to advanced chronic periodontitis.

Materials and Methods: Forty subjects were selected. They were divided to two groups of LLLT and control groups of 20 each. Diode laser was used for the adjunct non-surgical periodontal therapy applied to the gingival surface after periodontal treatment. These groups were further divided as those with tobacco habit and without habit. Gingival crevicular fluid was tested for Matrix metalloproteinase-1, tissue inhibitor matrix metalloproteinase-1, transforming growth factor-b1, and basic-fibroblast growth factor levels in the 1st, 3rd, 5th months after treatment.

Results: Better improvement levels were shown by LLLT group in sulcus bleeding index, clinical attachment, probing depth levels compared to the control group ($P < 0.001$). Significant progresses in the LLLT smokers' PD and SBI levels compared to smokers of controls at all the months was seen. ($P < 0.001$) Significant decrease in the marker levels at the time periods between both the groups was seen. ($P < 0.001$) Basic-fibroblast growth factor levels significantly diminished in both groups in the first month after the treatment, then increased in the third and sixth months ($P < 0.005$).

Conclusion: The periodontal non-surgical therapy with the low-level laser therapy can successfully be used for the periodontal health.

Key Words: Periodontitis, low level laser therapy, FGF, TGF, Probing Depth

Introduction

The periodontal therapy includes both the surgical and nonsurgical methods. The application of the laser has been used as an adjuvant for non-surgical therapies in the various dental procedures.¹ The low-level laser therapy (LLLT) is applied for its speed wound repair properties, lower pain, anti-inflammatory properties.² This laser works at molecular level and promotes the healing. There has been inadequate data on how the laser properties can be used for the periodontal surgeries. Some studies have evaluated the LLLT as an adjunct only in periodontal surgeries.³⁻⁶ Tobacco habits like smoking has been associated with poor prognosis with the periodontal surgeries. There are only few studies that have evaluated the use of low level lasers in the periodontal nonsurgical therapies.⁷ Hence in this study we intend to evaluate the application of the LLTE in nonsurgical periodontal therapy as an adjuvant, by measuring the molecular markers in the GCF for TGF-b1, TIMP-1, MMP-1, and fibroblast growth factors-beta levels. We also compared the subjects with and without smoking.

Materials and methods

We included 40 subjects for the study who had chronic periodontitis (moderate to advanced). These subjects were divided as LLLT and control group (n=20), and each group further divided into smokers and nonsmokers equally (n=10). The subjects were selected after ethics committee approval, based on exclusion of those who were without any systemic diseases, were not using the antibiotics, unwilling for surgery. Those subjects who have never smoked in life were considered as controls, and those who smoked 10 cigarettes/day were considered as smokers. Only scaling and root planing were done in Control, while LLLT with the diode laser was applied after scaling and root planing in case group. Clinical attachment level (CAL), Plaque index (PI), Probing Depth, and sulcus bleeding index (SBI) were recorded on six sites per tooth at baseline, 1, 3, and 6 months after the treatment. GCF was collected using filter paper strips from the deepest pre pocket sites of 5 mm depth of the incisors and premolars. Samples were collected to assess GCF level of MMP-1, TIMP-1, TGF-b1, and b-FGF, by using the ELISA. Statistical analysis was done keeping the $p < 0.05$ and using the appropriate statistical tools.

Results

We observed that the mean age was 42.3 ± 7.5 years. The gender distribution was M:F=11:9. In the follow up for the period of six months it was noted that all the patients were followed up through the 6 months period. No adversative effects with the laser therapy were noted. When the clinical parameters were compared no statistically significant differences in PI, SBI, PD, and CAL between the groups at baseline was seen. In both the case and the control groups the PI, SBI, PD, and CAL showed reduction. The SBI scores, PD, and CAL reduction were significantly better in the LLLT group between baseline and time points ($P < 0.001$). No statistically significant difference of reduction was observed in CAL changes at baseline and 6 months between the subgroups. We observed a statistically significant clinical betterment in the laser-applied smokers' PD and SBI levels compared to smokers to whom laser was not applied, between the baseline and all time points ($P < 0.001$) No statistically significant difference was seen in PD and CAL variations of the laser group among the smokers and non-smokers at different time points. (Table-1) When the biochemical analysis was done we observed no statistically significant difference in any analyzed biochemical marker level changes between the groups at any time points. MMP-1/TIMP-1 and TGF-b1 showed a statistically significant

decrease at baseline and all time points in the case and the control groups. A significant reduction between baseline and first month and a significant increase between baseline, third, and sixth months were observed in b-FGF levels. (Table-2)

Table 1: Comparison of the clinical parameters

| Variable | Baseline | 1st Month | Variance(0 to 1 month) | P Value [†] | 3rd Month | Variance(0 to 3 months) | P Value [†] | 6th Month | Variance(0 to 6 months) | P Value [†] |
|-----------------|-------------|------------|------------------------|----------------------|--------------|-------------------------|----------------------|-------------|-------------------------|----------------------|
| PI | | | | | | | | | | |
| Control | 1.790–0.66 | 1.701–0.67 | 1.090–0.80 | <0.001 | –0.590.60 | 1.201–0.750 | <0.001 | 0.680–0.620 | 1.101–0.704 | <0.001 |
| LLLT | 1.860–0.52 | 1.660–0.59 | 1.200–1.640 | <0.001 | 1.0610–0.504 | 1.250–0.640 | <0.001 | –0.6670.600 | 1.191–0.66 | <0.001 |
| P value* | | | <0.001 | | | <0.001 | | | <0.001 | |
| SBI | | | | | | | | | | |
| Control | 1.891–1.031 | 1.501–0.59 | 1.390–1.000 | <0.001 | 1.051–0.757 | 1.390–1.141 | <0.001 | 0.370–0.671 | 1.522–1.080 | <0.001 |
| LLLT | 1.810–1.041 | 1.301–0.57 | 1.501–1.018 | <0.001 | 1.021–0.406 | 1.610–1.18 | <0.001 | 0.118–0.408 | 1.613–1.111 | <0.001 |
| P value* | | | <0.001 | | | <0.001 | | | 0.001 | |
| PD (mm) | | | | | | | | | | |
| Control | 4.14–1.80 | 3.12–1.48 | 1.12–1.106 | <0.001 | 2.91–1.32 | 1.17–1.16 | <0.001 | 2.92–1.405 | 1.13–1.23 | <0.001 |
| LLLT | 3.79–1.61 | 2.18–1.11 | 1.30–1.20 | <0.001 | 2.41–1.26 | 1.48–1.2 | <0.001 | 2.47–1.20 | 1.41–1.25 | <0.001 |
| P value* | | | <0.001 | | | <0.001 | | | <0.001 | |
| CAL (mm) | | | | | | | | | | |
| Control | 4.63–2.07 | 3.60–1.87 | 0.83–1.105 | <0.001 | 3.71–1.912 | 0.85–1.206 | <0.001 | 3.534–1.937 | 1.11–1.17 | <0.001 |
| LLLT | 4.50–1.91 | 3.37–1.54 | 1.047–1.112 | <0.001 | 3.40–1.57 | 1.20–1.21 | <0.001 | 3.40–1.65 | 1.15–1.20 | <0.001 |
| P value* | | | <0.001 | | | <0.001 | | | | <0.001 |

* Mann-Whitney U test $P < 0.01$.

† Wilcoxon test $P < 0.05$.

Table 2: Comparison of the biochemical parameters

| Parameter (SD) | Baseline | 1 month | Difference (0 to 1 month) | P Value [†] | 3 Months | Difference (0 to 3 months) | P Value [†] | 6 Months | Difference (0 to 6 months) | P Value [†] |
|---|----------|---|------------------------------------|----------------------|---|----------------------------|----------------------------|---|----------------------------|--------------------------|
| MMP-1 (ng/sample) Control 1.021–0.57 LLLT 0.84–0.48 P value* 0.289 | | 0.57 – 0.31 0.44 – 0.21 0.194 | 0.48 – 0.56 0.41 – 0.51 | <0.001 <0.001 | 0.53 – 0.35 –0.42 0.47 – 0.28 –0.49 0.610 | 0.47 0.37 | <0.001 0.002 | 0.430 – 0.13 –0.551 0.441 – 0.14 –0.410 0.748 | 0.575 0.40 0.40 | <0.001 <0.001 |
| TIMP-1 (ng/sample) Control 1.81 – 0.92 LLLT 1.55 – 0.61 P value* 0.457 | | 2.11 – 1.10 1.78 – 0.82 0.27 | –0.48 – 1.01 –0.21 – 0.598 | 0.013 0.070 8 | 1.89 – 0.979 –0.112 – 0.500 1.515 – 0.416 –0.715 0.419 | – 0.00 | 0.064 0.528 | 2.023 – 1.300 –0.211 – 0.801 1.712 – 0.719 –0.15 – 0.804 0.286 | – – – – | 0.111 2 0.170 0 |
| MMP-1/TIMP-1 Control 0.67 – 0.45 LLLT 0.58 – 0.29 P value* 0.743 | | 0.321 – 0.109 0.289 – 0.107 0.664 | 0.402 –0.434 0.219 –0.246 | <0.001 <0.001 | 0.324 – 0.226 0.31 4 – 0.229 0.312 – 0.200 0.21 5 – 0.234 0.671 | – 0.31 – 0.21 | <0.001 0.007 | 0.206 – 0.102 0.41 1 – 0.328 0.218 – 0.008 0.31 0 – 0.217 0.362 | – – – – | <0.001 <0.001 |
| TGF-β1 (pg/sample) Control 196.97 – 115.798 66.406 – 45.512 121.010 – 101.967 LLLT 137.105 – 61.213 41.001 – 22.418 95.135 – 61.910 P value* 0.1510 0.1600 | | | | <0.001 <0.001 | 37.97 – 43.410 158.99 – 108.910 21.845 – 15.15 116.301 – 60.143 0.247 | | <0.001 1 <0.001 1 | 15.60 – 16.981 181.36 – 11.489 12.715 – 12.25 125.41 – 62.54 0.505 | | <0.001 <0.001 |
| b-FGF (pg/sample) Control 34.80 – 16.24 LLLT 30.10 0 – 14.808 P value* 0.338 | | 9.261 – 5.541 12.85 2 – 8.724 0.325 | 26.64 – 13.24 17.06 – 10.28 | <0.001 <0.001 | 22.01 – 12.64 12.81 – 10.45 23.84 – 12.12 6.04 – 7.75 0.627 | | <0.001 1 0.005 | 43.94 – 15.41 –9.122 – 7.31 38.54 – 15.15 –8.57 – 6.17 0.191 | | 0.001 0.001 |

* Mann-Whitney U test $P < 0.01$.† Wilcoxon test $P < 0.05$.

Discussion

The lasers have been used in the medical procedures extensively recently. Low level lasers have been associated with better healing in the various specialties like dermatology, ophthalmology, etc.^{1,7} These are known to work at molecular levels. These factors are Dose dependent. Biostimulation is reported with the LLLT at 0.001 and 10 J/cm².²⁻⁴ Mester et al.⁸ proposed that doses of 1 to 2 J/cm² are essential to see an outcome on wound healing. In the present study wavelength of 807 nm laser, output power of 0.24W, and 4.0 J/cm² energy density on the 1st, 2nd and 7th day after the treatment. At this dosage there has been noted wound healing and epithelization.^{9,10} In our study there were statistically significant enhancement in clinical parameters after non-surgical periodontal treatment in each group. This could be due to the reduction of clinical inflammation, microbial shifts to a less pathogenic sub-gingival flora, PD reduction, and gain of clinical attachment. We also noted significant improvement in the LLLT group in PD, CAL, and SBI compared to the controls. In the study of Qadri et al.¹¹ low-level lasers lowered the periodontal gingival inflammation and PD, similar observations are made in our study. However in the study of Lai et al.¹² low-power laser did not bring about any clinical advantages than routine. This is contradictory to our study. This could be due to research design variations to our study. LLLT ensued in significantly decrease of SBI score associated to the controls. This could be due to the anti-inflammatory cytokine levels increase and an upsurge of microcirculation by the low-level laser therapy.^{13,14} In our study the alteration of reduction of PD in both moderate and deeper sites was seen to be statistically significant among the groups. The LLLT group's reduction was significantly more in the 6 months post-treatment. Smoking impacts periodontal treatment outputs.¹⁵ We have also found additional improvements in the smoking LLLT group compared to control group smokers. Significant lowering was seen in SBI and PD in 6th month and CAL in 3rd month. The reason may be an increased microcirculation in the LLLT where it is hindered in smokers. When in the case group the bio-clinical parameter of SBI, PD and CAL was compared there was no statistical difference between the smokers and nonsmokers. MMPs play a vital part in the tissue repair and remodeling after surgical procedures. MMP-1 is the major type of proteolytic enzyme that can break interstitial collagens type I and type III.¹⁶ Its activity is further in control of controlled through TIMP-1. Their ratio MMP-1 to TIMP-1 acts as predictor of wound repair.^{17,18} In the study of Tuter et al.¹⁹ the decrease of the ratio of MMP-1 to TIMP-1 after non-surgical periodontal therapy was published. Similar observations were made in our study where we noted a reduction in the ratio of MMP-1 to TIMP-1 after LLLT was noted. But there was no significant variation between the control and LLLT groups. Fibroblast proliferation is enhanced in repair by the TGF-β1.¹⁶ When there is inflammation the levels of TGF-β1 levels rise like in situations of gingival tissues and GCF at sites of inflammation related to normal subjects.³⁵ This growth factor is also a subject of the cytokine levels. In the study of Skaleric et al.²⁰ the TGF-β1 levels in GCF positively correlated with PD. Similarly in the present study, total amount of TGF-β1 in GCF lowered in both the LLLT and control groups after removal of inflammation. However there was no significant difference between the LLLT and control groups. β-FGF from the gingival fibroblasts helps in fibroblast activation and collagen production.^{21,22} We observed that GCF level of β-FGF lowered in both the groups in the first month but later increase of β-FGF levels in GCF were noticed in the following months. This study is the first to report β-FGF in GCF variations using the LLLT. It can be concluded from our study that the low level laser therapy develops clinical results, after the routine scaling and root planing. The biochemical changes are in accordance with the clinical progress. Also the variations of the clinical parameter were evident when laser was used than

only the non-surgical periodontal treatment. Only the laser-applied group in smokers depicted statistically significant clinical improvements. Nevertheless, the variations between test and control group in all the parameters were least at 6 months.

Conclusion

The periodontal non-surgical therapy with the low-level laser therapy can successfully be used for the periodontal health. Also the clinical and the biochemical parameters have improved in the individuals with the smoking habits. Hence the LLLT can be used as an adjuvant in the periodontal nonsurgical therapies.

References

1. Badersten A, Nilve'us R, Egelberg J. Effect of non- surgical periodontal therapy. I. Moderately advanced periodontitis. *J Clin Periodontol* 1981;8:57-72.
2. Karlsson MR, Diogo Lo'fgren CI, Jansson HM. The effect of laser therapy as an adjunct to non-surgical periodontal treatment in subjects with chronic peri- odontitis: A systematic review. *J Periodontol* 2008;79: 2021-2028.
3. Marques MM, Pereira AN, Fujihara NA, Nogueira FN, Eduardo CP. Effect of low-power laser irradiation on protein synthesis and ultrastructure of human gingival fibroblasts. *Lasers Surg Med* 2004;34:260-265.
4. Silveira PC, Streck EL, Pinho RA. Evaluation of mitochondrial respiratory chain activity in wound healing by low-level laser therapy. *J Photochem Photo- biol B* 2007;86:279-282.
5. Alexandratou E, Yova D, Handris P, Kletsas D, LoukasS. Human fibroblast alterations induced by low power laser irradiation at the single cell level using confocal microscopy. *Photochem Photobiol Sci* 2002;1:547- 552.
6. Hawkins D, Abrahamse H. Effect of multiple exposures of low-level laser therapy on the cellular re- sponses of wounded human skin fibroblasts. *Photomed Laser Surg* 2006;24:705-714.
7. Pereira AN, Eduardo CdeP, Matson E, Marques MM. Effect of low-power laser irradiation on cell growth and procollagen synthesis of cultured fibroblasts. *Lasers Surg Med* 2002;31:263-267.
8. Mester E, Spiry T, Szende B, Tota JG. Effect of laser rays on wound healing. *Am J Surg* 1971;122:532-535.
9. Lagan KM, Clements BA, McDonough S, Baxter GD. Low intensity laser therapy (830nm) in the manage- ment of minor postsurgical wounds: A controlled clinical study. *Lasers Surg Med* 2001;28:27-32.
10. Ozcelik O, Cenk Haytac M, Kunin A, Seydaoglu G. Improved wound healing by low-level laser irradiation after gingivectomy operations: A controlled clinical pilot study. *J Clin Periodontol* 2008;35:250-254.
11. Qadri T, Miranda L, Tune'r J, Gustafsson A. The short- term effects of low-level lasers as adjunct therapy in the treatment of periodontal inflammation. *J Clin Peri- odontol* 2005;32:714-719.
12. Lai SM, Zee KY, Lai MK, Corbet EF. Clinical and radiographic investigation of the adjunctive effects of a low-power He-Ne laser in the treatment of moderate to advanced periodontal disease: A pilot study. *Pho- tomed Laser Surg* 2009;27:287-293.
13. Hughes TP, Caffesse RG. Gingival changes following scaling, root planing and oral

hygiene. A biometric evaluation. *J Periodontol* 1978;49:245-252.

14. Woodruff LD, Bounkeo JM, Brannon WM, et al. The efficacy of laser therapy in wound repair: A meta- analysis of the literature. *Photomed Laser Surg* 2004; 22:241-247.

15. Grossi SG, Zambon J, Machtei EE, et al. Effects of smoking and smoking cessation on healing after me- chanical periodontal therapy. *J Am Dent Assoc* 1997; 128:599-607.

16. Birkedal-Hansen H. Role of matrix metalloproteinases in human periodontal diseases. *J Periodontol* 1993; 64(Suppl. 5):474-484.

17. Pardo A, Selman M. MMP-1: The elder of the family. *Int J Biochem Cell Biol* 2005;37:283-288.

18. Muller M, Trocme C, Lardy B, Morel F, Halimi S, Benhamou PY. Matrix metalloproteinases and diabetic foot ulcers: The ratio of MMP-1 to TIMP-1 is a predictor of wound healing. *Diabet Med* 2008;25:419-426.

19. Tu'ter G, Kurtisx B, Serdar M. Effects of phase I peri- odontal treatment on gingival crevicular fluid levels of matrix metalloproteinase-1 and tissue inhibitor of metalloproteinase-1. *J Periodontol* 2002;73:487-493.

20. Skaleric U, Kramar B, Petelin M, Pavlica Z, Wahl SM. Changes in TGF-beta 1 levels in gingiva, crevicular fluid and serum associated with periodontal inflam- mation in humans and dogs. *Eur J Oral Sci* 1997;105: 136-142.

21. Cochran DL, Wozney JM. Biological mediators for periodontal regeneration. *Periodontol* 2000 1999;19: 40-58.

22. Yu W, Naim JO, Lanzafame RJ. The effect of laser irradiation on the release of bFGF from 3T3 fibro- blasts. *Photochem Photobiol* 1994;59:167-170.