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Evaluation of the effectiveness of diode laser therapy in conjunction with nonsurgical treatment of peri-implantitis

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ABSTRACT

Purpose: Peri-implantitis (PI) is an inflammatory condition associated with the destruction of bone tissue around a dental implant, and diode lasers can be used to treat this disease. In this study, we aimed to evaluate the effectiveness of a 940-nm diode laser for the nonsurgical treatment of PI.
Methods: Twenty patients (8 women and 12 men) were enrolled in a split-mouth randomized controlled study. In the control group (CG), mechanical debridement with titanium curettes accompanied by airflow was performed around the implants. The test group (TG) was treated similarly, but with the use of a diode laser. Clinical measurements (plaque index, gingival index [GI], probing pocket depth [PPD], bleeding on probing [BOP], clinical attachment level, and interleukin-1β [IL-1β] in the peri-implant crevicular fluid) were evaluated and recorded at baseline and 3 months. IL-1β levels were determined using the enzyme-linked immunosorbent assay method.
Results: The symptoms were alleviated in both groups at 3 months as assessed through clinical measurements. GI, BOP, and PPD were significantly lower in the TG than in the CG (P<0.05). The IL-1β level increased post-treatment in both groups, but this increase was only statistically significant (P<0.05) in the CG.
Conclusions: The diode laser enabled improvements in clinical parameters in the peri-implant tissue. However, it did not reduce IL-1β levels after treatment. Further studies about the use of diode lasers in the treatment of PI will be necessary to evaluate the effects of diode lasers in PI treatment.

Keywords: Cytokine; Dental implant; Diode laser; Interleukin-1beta; Laser therapy; Peri-implantitis

INTRODUCTION

Peri-implant diseases have been classified as periodontal diseases by the Consensus Report of the 2017 World Periodontology Workshop [1]. Peri-implantitis (PI) is an inflammatory condition associated with various degrees of bone destruction around a dental implant, with symptoms such as a peri-implant pocket depth of at least 4 mm, bleeding, and/or purulent flow [2]. Peri-implant diseases are common, and the prevalence of peri-implantitis has been

rising due to the increase in the use of dental implants. Dreyer et al. [3] reported that the prevalence of PI was between 1.1% and 85.0%. In addition, they reported that the incidence of PI was 43.9% within 5 years [3]. Although poor plaque control plays a major role in the etiology of PI, other risk factors have also been proposed to contribute to the improvement of peri-implant diseases, such as a history of chronic periodontitis, irregular dental visits after implant therapy, smoking, and diabetes [2]. Implant surface characteristics, such as its chemical composition, surface free energy, and roughness may affect bacterial attachment and proliferation [4].

Peri-implant infection control entails removing the biofilm from the implant site and performing a thorough mechanical debridement. Airflow, saline, citric acid, hydrogen peroxide, chlorhexidine, and lasers are used to decontaminate the titanium surfaces of implants [5,6]. Diode laser therapies have been shown to be effective for disinfecting implant surfaces and the biostimulation of peri-implant tissues without causing problems in the surrounding tissues because they do not interact with titanium or coated materials [7,8]. Various studies have evaluated the use of diode lasers in periodontal or peri-implant treatments [4,6,9-18]; however, some studies have reported that diode lasers may not provide significant clinical benefits for periodontal disease treatment [4,9,13]. A recent review by Mattar et al. [4] concluded that the data available in the literature did not support the use of diode lasers for PI therapy, but more studies were needed to confirm this result. Aimetti et al. [9] showed that diode laser treatment did not provide a statistically significant clinical benefit in managing peri-implant inflammation at 3 months compared to nonsurgical mechanical therapy alone. In another study, Meseli et al. [13] found that diode laser therapy did not have an additional clinical effect on residual pockets.

The importance of clinical signs of inflammation and an increase in the probing depth have been emphasized in the diagnosis of PI [2]. Clinical signs of inflammation include redness, edema, mucosal enlargement, bleeding on probing (BOP) with or without suppuration, increasing pocket depth, and radiographic marginal bone loss. It has been reported that the observation of progressive bone loss around an implant in the absence of other clinical manifestations is very rare [2]. Sánchez-Martos et al. [15] reported that the diode lasers caused a statistically significant improvement in BOP, while also ameliorating other clinical parameters, albeit not significantly. In addition to clinical parameters, the presence of inflammatory mediators in the peri-implant crevicular fluid (PICF) is important in the diagnosis of PI. One of the most researched indicators of periodontal and peri-implant disorders is pro-inflammatory cytokines. One of these cytokines is interleukin (IL)-1β, which plays an important role in peri-implant diseases since it regulates collagenase activity in inflammation and wound healing [18,19]. Yoshinari et al. [18] showed that IL-1β levels increase after periodontal treatment, while another study reported a significant increase in IL-1β levels in the crevicular fluid in PI compared to healthy implant surfaces [19]. To the best of our knowledge, no study has evaluated the effects of diode lasers on IL-1β levels in the treatment of PI.

Therefore, in this clinical study, we aimed to investigate the early effectiveness of 940-nm diode laser therapy as a supportive method for the nonsurgical therapy of PI.

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MATERIALS AND METHODS

Patient selection and clinical measurements

This study was approved by the Van Yuzuncu Yil University Clinical Trials Ethics Committee (10.01.2018/04) and performed in accordance with the principles of the Helsinki Declaration. Twenty partially dentate patients with at least 2 implants diagnosed with PI (defined below) were selected as the study population. All patients signed consent forms for voluntary participation in the study after being informed of the possible benefits and risks of the intervention. All implants included in the study were made by the same manufacturer (Implant Direct, Thousand Oaks, CA, USA), with a large-grit, sandblasted, acid-etched surface, and contained an internal conical connection and cement-retained fixed bridges supported by the implant.

Patients were selected according to the following PI case definition: 1) peri-implant bone loss >2 mm compared to baseline radiography taken at the time of bridge placement; 2) BOP (0.25 N); 3) presence of redness and edema with or without suppuration in the peri-implant zone; 4) increase in probing pocket depth (PPD) (≥4 mm); and 5) the use of implant-supported fixed bridge prosthesis for at least 6 months. The exclusion criteria were: 1) clinical implant mobility; 2) pregnancy or lactation; 3) presence of systemic disease; 4) use of antibiotics in the previous three months; or 5) use of tobacco.

The clinical study was planned as a randomized, controlled, split-mouth study in order to eliminate selection bias, which might have been made consciously or unconsciously at the stage of selecting patients, and variations in the host response between patients. Five patients were excluded because they did not fulfill the study criteria, and 2 patients chose not to participate in the study (**Figure 1**). Overall, 20 patients and 40 implants were included. Each patient provided 1 site for the test group (TG) and 1 for the control group (CG). The PI

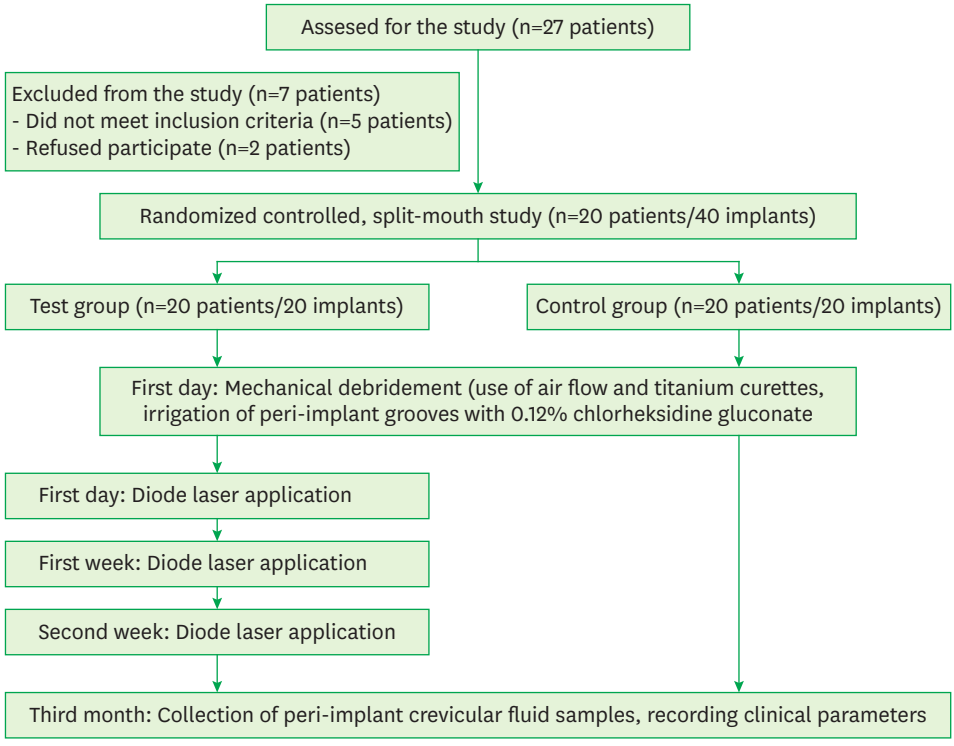

Figure 1. Study flow diagram.

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site with the deepest pocket in different quadrants was selected for each treatment group in each patient. If the TG or CG had more than 1 implant with PI, all regions with peri-implant disease were treated. The investigator (EA) performed clinical assessments for the TG (n=20) and CG (n=20) at baseline and after 3 months. Randomization was performed by tossing a coin by the same researcher. The treatments were conducted by DA.

The plaque index (PIn) [20], gingival index (GI) [21], BOP, PPD, and clinical attachment level (CAL) were measured at pre-treatment and the 3-month follow-up. A millimetrically calibrated Williams periodontal plastic probe (Hu-Friedy, Chicago, IL, USA) was used to perform and record measurements at 6 regions of each implant (mesiobuccal, distobuccal, mesiopalatinal/lingual, distopalatinal/lingual, midfacial, and midpalatal/lingual). PICF was collected 1 day after the periodontal parameters were recorded. The sampled areas were isolated using cotton rolls. Saliva contamination was avoided by a suction and air-water spray. For sampling, paper strips (Periopaper; OraFlow, Inc., Hewlett, NY, USA) were inserted into the deepest peri-implant pocket until slight resistance was felt. Each strip was kept in the pocket for 30 seconds. Samples contaminated with saliva and/or blood were not included in the study. PICF was collected at baseline and 3 months. The paper strips were kept in 500 μL of phosphate-buffered saline in Eppendorf tubes (SealRite 1.5 mL Microcentrifuge Tubes; USA Scientific, Inc., Ocala, FL, USA) at −40°C until the test day. The IL-1β levels in the PICF samples were evaluated by an enzyme-linked immunosorbent assay (ELISA) (Human Interleukin 1β ELISA kit, cat. no. SG-10260, lot no. 201803, manufactured by SinoGeneClon Biotech Co., Ltd., Hangzhou, China). The absorbance was read at 450 nm in a BioTek ELX-800 brand ELISA reader. The standard concentrations and absorbances were used, and a calibration graph was drawn using the Curve Expert 1.4 program. The sample concentrations were calculated from the calibration graph using the sample absorbance values.

Mechanical debridement and laser treatment protocol

A titanium curette (ImplaMate; Nordent, Elk Grove Village, IL, USA) and airflow (PROPHYflex-3; KaVo, Biberach an der Riss, Germany) were used during mechanical debridement, and peri-implant pockets were irrigated locally with 0.12% chlorhexidine gluconate in all groups. The CG and TG received the same mechanical treatment procedures. First, airflow was used for visibly contaminated implant threads and dental calculus, while the tip of airflow was applied in a non-contact mode circumferentially around the implant for 15 seconds at each aspect (mesial, distal, buccal, and lingual) of the treated implants. A high-speed aspiration system was used for this procedure. All pockets of each implant were curetted with a titanium curette. We applied vertical strokes on the implant surface with the edge of the curettes; and horizontal strokes with the point of the curettes in the valleys. After removing the granulation tissue, the inside of the pocket was irrigated locally with 0.12% chlorhexidine gluconate with 1 injector for each implant. Subgingival debridement was performed once for each implant.

After the mechanical debridement procedures, the tip of the laser was inserted into the peri-implant crevice for placebo treatment for the CG; however, it was not activated. For the TG, laser therapy was applied 3 times; on the same day of mechanical debridement, and 1 and 2 weeks after mechanical debridement according to previously published reports [14]. A 940-nm diode laser (Ezlase; Biolase Technology Inc., Foothill Ranch, CA, USA) was applied with an optic fiber tip that had a diameter of 300 μm (power: 1.5 W; energy density: 15 J/cm^2; pulse range: 20 ms; tip mode: uninitiated mode). The fiber tip was placed parallel to the axis of the implant, and it was pulled coronally with mesiodistal movements with laser application for

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30 seconds. In each laser therapy session, the laser tip was retracted 1 mm into the pocket with the aim of not negatively affecting recovery. During the application of the laser, the laser tip was inspected every 10 s and cleaned using isotonic solution, and the peri-implant pockets were cooled with saline to reduce the negative effects of the laser-induced heat.

No antibiotics were prescribed during treatment. A mouthwash containing chlorhexidine gluconate was prescribed to all patients to be used twice a day for 1 week. Patients received individualized oral hygiene instructions on tooth brushing, use of interdental brushes, and use of floss. All the patients were invited to follow-up visits at 1 and 3 months to check their oral hygiene habits, but PICF samples and clinical assessments were only collected at 3 months.

Statistical analysis

In order to determine intra-observer reliability, the intra-class correlation coefficient (ICC) was computed. For ICC computation, measurements were repeated by the same observer 2 times with a 20-minute interval in 10 individuals. The ICC was found to be 0.91 (P<0.01). In addition, the Dahlberg error was also computed to determine the random measurement error, and this value was found to be 0.023.

In this prospective study, the sample size was determined by considering an 80% power (α=0.05 and β=0.20), an effect size of 0.09 (d), and a Z value of 1.96. The standard deviation (s) was considered as 0.2 for IL-1β. Thus, the minimum sample size was determined to be 19 (approximately 20) by using the "$n=Z^2\ s^2/d^2$" equation for sample size calculation. Descriptive statistics for the continuous variables (characteristics) were shown as means and standard deviations, while counts and percentages were used for the categorical variables. The normality assumption of the continuous variables was analyzed with the Kolmogorov-Smirnov test. After the normality test, the Student's t-test was used to compare the means for normally distributed characteristics. In addition, the paired t-test was used to compare pre- and post-treatment values. The statistical significance level was considered 0.05, and SPSS version 21 (IBM Corp., Armonk, NY, USA) was used for all statistical computations.

RESULTS

The clinical study included 20 patients (8 women and 12 men, mean age: 51.40±10.15 years) diagnosed with PI. The flow chart of the study is shown in **Figure 1**, and the age and sex distribution of the patients is presented in **Table 1**.

Clinical periodontal parameters

Changes in the clinical parameters (GI, PIn, PPD, CAL, and BOP) and the IL-1β levels are shown in **Table 2**. The GI decreased from 2.44±0.56 to 1.28±0.60 (P<0.05) in the CG and from 2.45±0.51 to 1.07±0.54 (P<0.05) in the TG. There was no significant difference between the CG and TG in the pre-treatment GI values (P>0.05); however, the GI was significantly higher in the CG (P<0.05) after treatment.

Table 1. Demographic characteristics of the study groups

Variables	No.	Age (mean ± SD)	Min	Max
Male	12	49.75±11.50	36	68
Female	8	53.88±7.75	38	60
Total	20	51.40±10.15	36	68

SD: standard deviation, Min: minimum, Max: maximum.

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Table 2. Clinical and biochemical parameters (mean ± SD) at baseline (T_0) and post-treatment (T_1)

Variables	Baseline (T_0)	Month 3 (T_1)	Difference between T_0-T_1	*P* value
GI scores				
Control	2.44±0.56	1.28±0.60	1.15±0.84	0.001[a]
Test	2.45±0.51	1.07±0.54	1.38±0.76	0.001[a]
P value	0.842	0.006[b]	0.015[b]	
PIn scores				
Control	2.06±0.47	0.88±0.68	1.18±0.58	0.001[a]
Test	2.15±0.56	0.85±0.67	1.29±0.75	0.001[a]
P value	0.305	0.742	0.749	
PPD scores (mm)				
Control	4.50±1.15	3.94±1.14	0.55±0.81	0.006[a]
Test	5.11±1.04	3.73±0.95	1.38±0.95	0.001[a]
P value	0.028[b]	0.389	0.032[b]	
CAL scores (mm)				
Control	4.50±1.15	3.94±1.14	0.55±0.81	0.006[a]
Test	5.15±1.05	3.75±0.91	1.40±0.96	0.001[a]
P value	0.019[b]	0.457	0.711	
BOP Scores (%)				
Control	88.33±21.01	43.33±40.6	45.00±47.78	0.001[a]
Test	94.16±15.55	28.33±28.14	65.83±32.20	0.001[a]
P value	0.090	0.006[b]	0.061	
IL-1β (ng/L)				
Control	3.05±0.18	3.17±0.21	−0.11±0.21	0.028[a]
Test	2.99±0.16	3.11±0.21	−0.10±0.29	0.152
P value	0.013[b]	0.334	0.776	

SD: standard deviation, GI: gingival index, PIn: plaque index, PPD: probing pocket depth, CAL: clinical attachment level, BOP: bleeding on probing, IL-1β: interleukin 1β.
[a]Statistically significant difference within the group; [b]Statistically significant difference between the groups.

The PIn decreased from 2.06±0.47 to 0.88±0.68 (P<0.05) in the CG and from 2.15±0.56 to 0.85±0.67 (P<0.05) in the TG. There was no statistically significant difference between the 2 groups in post-treatment PIn values (P>0.05).

The pre-treatment PPD value was significantly higher in the TG than in the CG (P<0.05). In the CG, the PPD decreased from 4.50±1.15 to 3.94±1.14 (P<0.05). In the TG, the PPD decreased from 5.11±1.04 to 3.73±0.95 post-treatment (P<0.05). Although there was a greater decrease in the TG after the treatment, this decrease was not statistically significantly different from that observed in the CG. Considering the magnitude of the decrease in PPD value after treatment, the decrease in the TG was found to be statistically significant.

The mean pre-treatment CAL value was significantly higher in the TG than in the CG (P<0.05). The CAL values decreased from 4.50±1.15 to 3.94±1.14 (P<0.05) in the CG, and from 5.15±1.05 to 3.75±0.91 (P<0.05) in TG. Although there was a greater decrease in the TG after treatment, this decrease was not statistically significantly different from that observed in the CG.

The pre-treatment BOP rate was higher in the TG than in the CG, but this difference was not statistically significant. In the CG, the BOP rate decreased from 88.33%±21.01% to 43.33%±40.60% (P<0.05). In the TG, the BOP rate decreased from 94.16%±15.55% to 28.33%±28.14% (P<0.05). The post-treatment BOP rate was statistically significantly higher in the CG than in the TG (P<0.05).

Cytokine measurements

IL-1β levels are shown in **Table 2**. The mean IL-1β level in the CG was 3.05±0.18 ng/L before treatment and 3.17±0.21 ng/L after treatment. The mean IL-1β level in the TG was 2.99±0.16

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ng/L before treatment and 3.11±0.21 ng/L after treatment. The difference in IL-1β values was not found to be statistically significant.

DISCUSSION

In this clinical study, we investigated whether the addition of diode laser therapy to mechanical debridement is more effective in the therapy of PI than the use of mechanical debridement alone. Our results showed that using a diode laser as a supportive therapy in addition to the mechanical treatment of PI improved the clinical parameters. However, although both treatment methodologies provided clinical benefits, neither significantly changed the IL-1β levels.

PI has been a public health problem for several years, and the nonsurgical therapy methods of peri-implant diseases involve mechanical debridement, chemical modalities, antibiotic medicine, lasers, and oral hygiene instructions [22]. Many methods have been suggested for the treatment of PI [10,23-27]. A systematic review and meta-analysis showed that airflow had positive effects on reducing BOP in the short term [23]. Another systematic review and meta-analysis reported that systemic antibiotic administration in PI treatment did not reduce clinical parameters such as PPD and BOP [24]. Contrary to this study, it has been reported that antibiotics provide clinical benefits in the long term, especially in the treatment of PI [25]. Other methods used in the treatment of PI are photobiomodulation therapy and photodynamic therapy. It has been reported that the use of these methods in addition to mechanical debridement in PI treatment provides clinical benefits [26]. A recent study reported that the severity of peri-implant mucositis could be reduced by using an oral irrigator with 0.06% chlorohexidine in addition to mechanical debridement in the treatment of peri-implant mucositis [27]. Dental lasers are another modality for the treatment of PI [4,7-15,17]. Alpaslan Yayli et al. [10] used a diode laser and an erbium, chromium-doped: yttrium, scandium, gallium, garnet (Er,Cr:YSGG) laser for PI treatment. According to their results, the use of the 940-nm diode laser in addition to mechanical treatment was found not to be superior to mechanical therapy alone and did not provide any additional clinical benefits; however, the Er,Cr:YSGG laser seemed to be more efficient both at clinical and molecular levels.

Diode lasers are among the preferred lasers in therapy for peri-implant diseases [6,10,14,17]. Although the use of diode lasers in the detoxification of implant surfaces has increased recently, there are discordant outcomes in the literature regarding the additional benefits of lasers compared to traditional treatments. Several studies have argued that diode lasers do not provide additional clinical benefits [9-11,13], while others have suggested that diode lasers have beneficial results [14,15,28]. A systematic review by Mattar et al. [4] showed that the available data in the current literature did not support the use of 810-nm diode lasers in the treatment of PI. On the contrary, another review study concluded that 810-nm diode lasers with low average power and optimal irradiation time significantly improved the clinical parameters in PI [29]. This difference may be related to various factors such as laser settings, wavelengths, and study plans [30]. To the best of our knowledge, limited studies have used 940-nm diode lasers in PI therapy. One study reported that a 940-nm dose had an inhibitory effect on the viability of gingival fibroblasts, which might improve implant stability, and the researchers stated that lasers with different wavelengths may differ in the depth of penetration and the chromophores they can stimulate [31]. Mettraux et al. [14] reported that the application of an 810-nm diode laser to PI sites for 30 seconds at baseline and after 7 and

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14 days was significantly beneficial compared to mechanical debridement alone; thus, we utilized a similar strategy, with 940-nm diode laser application repeated 3 times over 2 weeks, in accordance with this procedure.

Aimetti et al. [9] reported that the addition of diode laser treatment to mechanical debridement produced comparable clinical improvements, with similar reductions in the amount of BOP-positive sites, plaque scores, and PPD values with respect to mechanical debridement alone. Alpaslan Yayli et al. [10] showed that the decrease in the PPD was similar between mechanical therapy alone and diode laser treatment in addition to mechanical therapy. A review article that analyzed data from 11 articles on the use of laser irradiation as an addition to peri-implant disease treatment concluded that the combination of laser irradiation with nonsurgical treatment had minimal benefits in reducing the PPD and PIn and improving the CAL and degree of recession [7]. Arisan et al. [11] concluded that the additional use of a diode laser did not have a favorable effect on peri-implant healing compared with the traditional method, and they observed greater marginal bone loss in the diode laser group than in the control group after 6 months. Meseli et al. [13] reported that the application of a diode laser in addition to mechanical periodontal therapy did not exhibit any additional clinical benefits on the residual pockets. However, Lerario et al. [28] observed that PPD and BOP rates showed a greater decrease when the diode laser was applied in addition to mechanical therapy compared to mechanical therapy alone. In the present study, both therapy modalities significantly improved the clinical parameters in PI therapy; however, the improvement in GI and BOP values after treatment was significantly greater in the TG than in the CG, while no significant difference was found between the 2 groups in PIn, PPD and CAL values. However, although the post-treatment PPD value did not differ statistically between the groups, the decrease in the PPD values post-treatment was significantly higher in the TG.

A recent study reported that cytokine levels were higher in implants affected by PI than in healthy implants [32]. IL-1β acts synergistically with other proinflammatory cytokines both in the initiation and propagation of inflammation [33]. Consequently, IL-1β plays an important role in the inflammatory response and alveolar bone resorption [34]. Evidence has demonstrated that the level of IL-1β is higher in the PICF on implant surfaces both in peri-implant healthy tissues and in areas with peri-implant mucositis [19]. In addition to clinical parameters, it has been reported that the levels of proinflammatory cytokines in PICF, such as IL-1β, could be used to differentiate healthy implants from peri-implant mucositis and PI [35]. Thus, the IL-1β level was evaluated as a biomarker in our study.
A diode laser was used in the treatment of PI in our study since diode laser systems are promising for implant decontamination and have not been reported to show any signs of surface changes [36]. A study examining IL-1β levels as an immune-related biomarker in the treatment of PI in smokers applied photochemotherapy using a diode laser [37]. The researchers followed the patients at baseline, the third month, the sixth month, and the 12th month [37]. The IL-1β levels showed a significant decrease only in the 12th month [37]. However, our study differs from this study since it was conducted among non-smokers. Consequently, to the best of our knowledge, there are limited studies investigating the level of IL-1β in PICF after the treatment of PI with diode lasers. However, the results from this study might be compared to the results obtained with a diode laser and mechanical therapy in cases of chronic periodontitis. Saglam et al. [38] found that the total IL-1β level decreased after therapy in both scaling and root planning (SRP) followed by diode laser irradiation and SRP alone groups. Qadri et al. [39] stated that a diode laser did not reduce IL-1β levels in gingival crevicular fluid (GCF) post-therapy. Yoshinari et al. [18] did not apply a diode laser,

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but they reported that the amount of IL-1β in GCF slightly increased after SRP. In the present study, while the clinical parameters improved after treatment in both groups, the IL-1β levels increased in both groups, exhibiting a lack of inhibition. The increase in the IL-1β level in the PICF might have been caused by inadequacies of measurement, since it has been reported that the measurement of IL-1β levels in GCF might not be reliable because IL-1β is expressed only in small quantities in the GCF and is produced locally at the interface between the production cells and target cells [18]. Furthermore, our results should be interpreted with caution, as a review reported that PI may have higher biomarker levels than periodontitis, which may indicate a difference in biomarker levels between these 2 diseases [40]. A lack of antibiotic supplementation might also have caused the increase in IL-1β expression in both groups since antibiotic treatment has been recommended in cases of bone destruction in the cumulative interceptive supportive therapy protocol but no drug supplements were used in this study to better evaluate the effectiveness of the laser.

The following limitations should be kept in mind when interpreting our results. First, the treatment results were evaluated at 3 months and not followed up in the long term, and the sample size was relatively small. Our study had no microbiological analyses and focused on a single biochemical marker. In addition, patients were not classified according to the etiology of PI and contributing factors. In addition, the wavelength, power, waveform, pulse duration, energy intensity, exposure time, target tissue, and tissue properties may all have strongly influenced the treatment results.

In conclusion, we observed that the use of a 940-nm diode laser in addition to the nonsurgical therapy of PI provided clinical, but not biochemical benefits. Long-term follow-up and increasing the sample size may change the results. Studies of different doses and durations of laser treatment are needed in the future since lasers have the potential to be applied in new therapeutic models in the treatment of PI.

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