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# Acute Low Back Pain with Radiculopathy: A Double-Blind, Randomized, Placebo-Controlled Study

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## Abstract

***Objective:*** The aim of this study was to investigate the clinical effects of low-level laser therapy (LLLT) in patients with acute low back pain (LBP) with radiculopathy. ***Background Data:*** Acute LBP with radiculopathy is associated with pain and disability and the important pathogenic role of inflammation. LLLT has shown significant anti-inflammatory effects in many studies. ***Materials and Methods:*** A randomized, double-blind, placebo-controlled trial was performed on 546 patients. Group A (182 patients) was treated with nimesulide 200 mg/day and additionally with active LLLT; group B (182 patients) was treated only with nimesulide; and group C (182 patients) was treated with nimesulide and placebo LLLT. LLLT was applied behind the involved spine segment using a stationary skin-contact method. Patients were treated 5 times weekly, for a total of 15 treatments, with the following parameters: wavelength 904 nm; frequency 5000 Hz; 100-mW average diode power; power density of 20 mW/cm$^2$ and dose of 3 J/cm$^2$; treatment time 150 sec at whole doses of 12 J/cm$^2$. The outcomes were pain intensity measured with a visual analog scale (VAS); lumbar movement, with a modified Schober test; pain disability, with Oswestry disability score; and quality of life, with a 12-item short-form health survey questionnaire (SF-12). Subjects were evaluated before and after treatment. Statistical analyses were done with SPSS 11.5. ***Results:*** Statistically significant differences were found in all outcomes measured ($p < 0.001$), but were larger in group A than in B ($p < 0.0005$) and C ($p < 0.0005$). The results in group C were better than in group B ($p < 0.0005$). ***Conclusions:*** The results of this study show better improvement in acute LBP treated with LLLT used as additional therapy.

## Introduction

Low back pain (LBP) is one of the most frequent health problems.[1,2] Specific low back pain represents only 15% of all back pain problems, and 50% of specific back pain is due to a prolapsed intervertebral disc (PID), in which the nucleus pulposus herniates through a tear in the annulus fibrosus, resulting in irritation of the adjacent nerve root and causing a typical radiculopathic pain.[3] Generally, pain that lasts less than 4 weeks is classified as acute pain.[4] The main clinical phenomena in acute LBP are pain and neurological disorders that affect daily activities.

There is increasing evidence that inflammation in itself and in association with root compression is the main pathological factor of radiculopathy associated with disc herniation. Disruption of the annulus fibrosus causes leaking of the nucleus pulposus into the spinal canal, which contains various irritants to tissues, including glycoproteins, phospholipase A2, and nitric oxide, which in turn cause an inflammatory response in and around the pain-sensitive nerve tissues.[5,6] Previous experimental and clinical studies have elucidated biochemical interactions between the affected disc tissue and nerve roots and demonstrated that inflammatory mediators can affect fibers in nerve roots at the same or neighboring segments without mechanical compression.[7] Another study recently demonstrated the effect of cyclic mechanical stress on the production of inflammatory agents and postulated a possible synergistic effect of simultaneous mechanical and chemical irritation of the annulus fibrosus cells on the production of pain mediators (PGE2).[8] Data strongly support the role of proinflammatory cytokines in the pain of herniated discs. Cytokines such as IL-3, IL-6, and IL-8 cause hyperalgesia in animals[9] and may play a role in the physiopathology of radiculopathy induced by disc herniation. The

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proinflammatory cytokines may do this by inducing expression of receptors within the dorsal root ganglion (DRG). In addition, axonal interactions with proinflammatory cytokines could increase electrical conductivity. Amaya and colleagues[10] present a combined inflammatory neuropathic explanation of (short-term) LBP based on their experiments in which pain was evoked by applying Complete Freund's Adjuvant (CFA) topically to a spinal nerve and DRG in rats. The pain etiology is supposed to be inflammatory, because CFA induces the local accumulation of inflammatory mediator molecules and triggers the expression of cyclooxygenase in the DRG.[10] The understanding of this process is becoming increasingly important to the nonsurgical treatment of disc herniation.

Many experimental and clinical studies, especially in similar syndromes, have shown the anti-inflammatory potential of LLLT in a dose-dependent manner.[11,12] The aim of this study was to investigate the clinical effects of LLLT as adjunctive therapy to pharmacology nonsteroid anti-inflammatory treatment in patients with acute LPB and associated radiculopathy.

## Materials and Methods

### Patients

The study was carried out between January 2005 and September 2008 at the Clinic for Rehabilitation of the Medical School University of Belgrade, Serbia. During this period, 960 patients with acute LBP and associated radiculopathy were admitted to the clinic for hospitalization or for outpatient treatment. The prospective double-blind, randomized, placebo-controlled study included 546 patients with acute LBP and unilateral radiculopathy who had symptoms for less than 4 weeks, which were caused by a prolapsed intervertebral disc (PID), confirmed by magnetic resonance imaging (MRI). In the study, 424 patients were not included because they had no response to initial contact or they had red flag symptoms,[13] diabetes mellitus, neurological problems or cancer; pregnant patients and patients treated surgically for the same problem or treated with oral corticosteroids and steroid injections for any reason in the previous month were also excluded.[14]

A diagnosis was made by clinical examination and additional neuroradiological and neurophysiological examinations.[15,16] The criteria for radicular pain were typical dermatomal pain radiating beyond the knee toward the foot, pain evoked by stretching of the sciatic nerve and worsening on Valsalva maneuver, and signs of nerve root dysfunctions such as sensory, motor, and reflex impairments.[17]

During the study, 2 patients dropped out of the study; however, the last recordings of their measured parameters were analyzed. The reason for drop outs was worsening of pain in both patients. Withdrawal from the study has not been registered.

All patients gave informed written consent to participate in the study, which was approved by the ethics committee of the Clinic for Rehabilitation of the Medical School University of Belgrade.

### Blinding

The patients were double-blinded and randomized into three groups following the allocation of 546 sequentially numbered, opaque, sealed envelopes (SNOSE) prepared earlier and a computerized table of random numbers. The table was balanced to ensure equal numbers in each group. Allocation concealment was maintained until statistical analysis was completed by the statistician (GH), who was blind to the coding.

### Treatment

The patients were randomized into one of the three treatment groups: group A ($n = 182$) was treated with cyclooxygenase COX-2 inhibitor nimesulide 200 mg/day and simultaneously with local active LLLT; group B ($n = 182$) was treated only with nimesulide at the same dosage; and group C ($n = 182$) was treated with nimesulide and simultaneously with local placebo LLLT. Laser units were manufactured by Enraf Nonius, Rotterdam, The Netherlands. Devices for LLLT were assigned as device A for active LLLT and device B for placebo LLLT. Patients were not aware of the active unit. Patients were treated 5 times weekly, for a total of 15 treatments with LLLT and 15 consecutive days with nimesulide. All patients were instructed on restricted and allowed activity (low aerobic activity). Treatment was applied by the same therapist, who was blind to the type of treatment.

### Active LLLT

The parameters of the laser beams are presented in Table 1. The choice of parameters was derived from previous studies.[18,19] Testing of optical output was performed before and after the end of the trial.

### Placebo LLLT

Placebo LLLT was applied in the same manner as for the active device by an identical device that was deactivated by a member of the Institute for Physics. The physicians and patients were unable to distinguish between the laser and placebo units.

### Outcomes

The primary outcome measures[20] were intensity of pain and lumbar mobility. Intensity of pain was measured by the visual analogous scale (VAS), which is a horizontal scale graded from zero, representing no pain, to 100 mm, representing the worst imaginable pain. Intensity of pain was separately measured for lumbar pain (VAS-Lu) and for leg pain (VAS-Le).[21] VAS scores were taken as an average of what the patient normally suffered a few days before evaluation. Lumbar mobility was measured by a modified Schober test, which represents lumbar flexion, and was assessed by measuring changes in the distance between the two spinal landmarks. For the modified Schober test, marks were made on the skin 10 cm above and 5 cm below the S1 as the participant stood in a neutral position. The participant then bent forward maximally, and the change in distance between these marks was measured and expressed in millimeters.[22] The second outcome measure was the highly validated Oswestry disability questionnaire,[23] which consists of 10 questionnaires about how pain affects daily activities, scored from 0 to 5 for each section, with higher values indicating more severe impact;[24] and a 12-item short-form health survey (SF-12) that consists of 12 questions concerning general health and can be

Table 1. Characteristics of Laser Beams

| | |
|---|---|
| Wavelength | 904 nm (red) |
| Laser frequency | 5000 |
| Power output | 100 mW |
| Spot size | 1 cm |
| Power density | 20 mW/cm$^2$ |
| Energy | 3 J/point |
| Energy density | 3 J/cm$^2$ on each point |
| Treatment time | 150 sec on each point |
| Number of points | 4 |
| Daily energy delivered | 12 J |
| Total energy delivered | 180 J |
| Application mode | Probe held stationary in skin contact |
| Anatomical site | Local transforaminal[a] |

[a]2.5 and 3.5 cm laterally of the spinous process of the involved nerve root (L4 or L5 or S1) and one distal-level segment.

divided into two aggregate summary measures: the physical component summary (PCS) and the mental component summary (MCS).[25]

Subjects were evaluated before and after the treatment by independent physicians who performed diagnostic assessment and were blind to the type of treatment. To systematically capture any adverse effects of treatment, subjects were asked to record any new symptoms.

### Statistics

The analysis was conducted on the basis of "intention to treat." Statistical analyses were done with SPSS 11.5. The results were expressed as mean ± SD for data that were normally distributed or median (25% and 75% percentiles) for data that were not normally distributed. We present two types of comparisons: (1) comparison of medians between the beginning and the end of the therapy for all groups on each measured outcome, and (2) comparison of differences in scores between the beginning and end of the therapy on each measured outcome between groups A and B, A and C, and B and C. Statistically significant differences were tested using the Wilcoxon signed-ranks test for paired observations (1). Statistically significant differences were tested using the independent *t* test, or the Wilcoxon Mann–Whitney test for two independent groups, or the chi-squared test, depending on the type of variable outcome (2). The level of statistical significance was set at a two-tailed *p*-value of 0.05. Effect-size analysis was done for an evaluation of the importance of measured changes, as post hoc power analysis.

## Results

The basic characteristics of subjects who entered the study are shown in Table 2. We did not find statistically significant differences among all groups in baseline characteristics.

### Outcomes

Median values with 25% and 75% percentiles for all groups on each outcome measured at the beginning and at the end of therapy are shown in Table 3. For comparing medians between the beginning and the end of therapy for all groups on each measured outcome, we used the Wilcoxon signed ranks test. For all groups, we found statistically significant differences in all measured outcomes ($p < 0.001$).

### Intergroup statistical analyses

Comparisons of mean values in the measured outcomes are presented in Table 4. The results of intergroup statistics show that group A results were better than those of groups B ($p < 0.0005$) and C ($p < 0.0005$). The results in group C were better than in group B ($p < 0.0005$), except for the Schober measure and the PCS score. Effect-size statistical analyses have shown the importance of the measured differences to be high for pain intensity in the leg between groups A and B ($d = 4.78$).

Detailed analyses between categories of pain (Table 5) at the end of therapy show statistically significance differences between groups ($\chi^2$ test), however, with high effect size between groups A and B ($d = 0.85$).

Detailed analyses of changes in the Oswestry score at the end of therapy (Table 6) also show statistically significant differences between all groups ($\chi^2$ test), however, with medium effect size between groups A and B ($d = 0.71$).

Systematic capture of adverse effects shows transitional worsening of pain in 27 of 182 (14.8%) patients in group A and persistent worsening of pain in 2 patients, 1 in group A and 1 in group C. Transitional worsening of pain was registered immediately after the first 3 sessions with a maximum duration of 6 h. Persistent worsening of pain was registered for 10 consecutive days. Patients with persistent pain were excluded from the study. Results of capture for side effects show the low-risk nature of LLLT.

Table 2. Basic Subject Characteristics

| | *Groups* | | | *Statistics* | | |
|---|---|---|---|---|---|---|
| *Variables* | *A* | *B* | *C* | *AB* | *AC* | *BC* |
| Age[a] | 43.5 ± 7.7 | 44.84 ± 9.22 | 41.87 ± 8.37 | $\chi^2 = 0.101$ | $\chi^2 = 0.01$ | $\chi^2 = 0.0113$ |
| Male[b] | 75/182 | 77/182 | 79/182 | $p = 0.245$ | $p = 0.053$ | $p = 0.055$ |
| Female[b] | 107/182 | 105/182 | 103/182 | $p = 0.75$ | $p = 0.92$ | $p = 0.92$ |
| Duration[a] (days) | 14.9 ± 5.3 | 15.52 ± 5.72 | 17.81 ± 5.6 | $p = 0.335$ | $p = 0.227$ | $p = 0.331$ |
| Nonhospitalized[b] | 36/182 | 40/182 | 45/182 | $\chi^2 = 0.15$ | $\chi^2 = 1.02$ $p$ | $\chi^2 = 0.25$ |
| Hospitalized[b] | 146/182 | 142/182 | 137/182 | $p = 0.69$ | $p = 0.313$ | $p = 0.62$ |

[a]Data are normally distributed.
[b]Data are not normally distributed.

Table 3. Median Values (25% and 75% Percentiles) of Measured Outcomes

| | Group A | | Group B | | Group C | |
|---|---|---|---|---|---|---|
| Outcomes[a] | Pretherapy | Posttherapy | Pretherapy | Posttherapy | Pretherapy | Posttherapy |
| VAS Le[b] | 78.5 (76.4; 80.6) | 34.0 (30.5; 38.0) | 78 (75; 80.5) | 60 (56; 65) | 76 (70; 78.1) | 54 (50; 56) |
| VAS Lu[c] | 66 (60; 69) | 35 (34; 38) | 67 (62.5; 70) | 50 (46; 55) | 65 (60; 67) | 45 (40; 46) |
| Oswestry[d] | 32 (31; 33) | 20 (19; 21) | 31 (30; 32) | 24 (22; 26) | 32 (31; 34) | 22 (20; 24) |
| Schober[e] | 35 (34.5; 36) | 38.5 (38; 39) | 35.5 (35; 36) | 38 (37; 38.5) | 36 (35; 37) | 38 (37.5; 39) |
| PCS[f] | 10 (9; 10) | 14 (13; 14) | 10 (9; 11) | 12.5 (12; 13) | 10 (9; 11) | 13 (12; 14) |
| MCS[g] | 12 (11; 14) | 18 (17; 19) | 11.5 (11; 12) | 15 (14; 16) | 12 (11; 14) | 17 (15; 18) |

[a]Statistically significant differences were found for all measured outcomes.
[b]Visual analogue scale of pain intensity in the leg.
[c]Visual analogue scale of pain intensity in the lumbar region.
[d]Oswestry score.
[e]Schober test.
[f]Physical component summary.
[g]Mental component summary.

## Discussion

Although LBP is prevalent and has a high risk of chronicity and recurrence, evidence on effective treatment of acute phase patients is generally lacking. The requirement for optimum treatment is emphasized by the fact that effective treatment of the acute phase reduces chronicity.[26,27] A broad spectrum of therapy approaches is being used in clinical practice today, ranging from pharmacological and physical agents to exercise and manipulative practice. Various types of physical agents are not sufficiently supported, which is reflected in the clinical practice guidelines for treatment of acute lumbar pain. The general recommendation is that further studies are required or that physical agents as a form of therapy could be potentially useful in patients for whom no improvement has been achieved by previous treatments.[28–30]

This study included patients with severe pain and moderate disability and discomfort during daily activities on baseline examination, associated with acute radicular lesion and discus herniation. Our results show statistically significant improvement in all groups, with better results for all investigated parameters in group A compared with other groups. Detailed analyses of categorized parameters with more specified clinical meaning have shown very clear differences between different treatment groups. The most prominent are the results on reduction in pain intensity. In a study of acute pain, a minimum clinically relevant change in pain intensity was found to be 13 mm,[31] and in this study it was more than 40 mm (Tables 4 and 5), switching from the moderate severe to the moderate class of intensity of pain,[32] and with high effect size between groups A and B. In addition, in group A the mean values of the Oswestry score (Tables 4 and 6) switched to the better functional class (from moderate to minimal disability) on control examination, with improvement in more than 30% (accounted for as a difference between percentage of normal values), and this was designated as a clinically important difference.[24]

Table 4. Statistical Analyses of Measured Changes

| | Groups | | | Intergroup statistics | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | A | B | C | A–B | | A–C | | B–C | |
| Outcomes | Mean (SD) or median | Mean (SD) or median | Mean (SD) or median | t or Z p | d[d] | t or Z p | d[d] | t or Z p | d[d] |
| VAS Le[a] | 43.81 ± 5.78 | 21.33 ± 6.03 | 16.54 ± 5.65 | 45.5 0.00[c] | 4.78 | 36.3 0.00[c] | 3.81 | −7.81 0.00[c] | 0.82 |
| VAS Lu[a] | 29.97 ± 6.69 | 20.81 ± 6.08 | 15.69 ± 5.99 | 21.43 0.00[c] | 2.25 | 13.66 0.00[c] | 1.44 | −8.08 0.00[c] | 0.85 |
| Schober[b] | 3.3 (2.5; 4) | 2 (1; 3) | 2 (1; 3) | −8.3 0.00[c] | 0.44 | −8.01 0.00[c] | 0.42 | −0.15 0.88 | No |
| Oswestry[b] | 12 (10.8; 13) | 10 (8; 12) | 6.5 (5; 8) | −11.3 0.00[c] | 0.59 | −5.98 0.001[c] | 0.31 | −10.7 0.00[c] | 0.56 |
| PCS[b] | 4 (4; 4) | 3 (2; 4) | 2 (2; 3) | −11.4 0.00[c] | 0.6 | −8.88 0.00[c] | 0.47 | −1.85 0.064 | No |
| MCS[b] | 6 (4; 7) | 4 (2; 5) | 3 (2; 4) | −11.9 0.00[c] | 0.63 | −8.21 0.00[c] | 0.43 | −3.58 0.00[c] | 0.19 |

[a]For data normally distributed, mean ± SD and *t*-value are shown.
[b]For data not normally distributed, median (25% to 75% percentiles) and Z-value are shown.
[c]Statistically significant difference.
[d]*d* (Cohen effect size: $d < 0.2$, low; $0.2 < d < 0.8$, medium; $d > 0.8$, high).

Table 5. Distribution of Improvement in Pain Intensity in the Leg

| | Groups | | | Comparison | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| *Improvement (mm)*[a] | *A* | *B* | *C* | *AB* | | *AC* | | *BC* | |
| ≤50 | 0 | 0 | 0 | $\chi^2 = 265.25$ | *d* | $\chi^2 = 222.26$ | *d* | $\chi^2 = 35.96$ | *d* |
| 30–50 | 122 | 37 | 87 | $p = 0.000$ | 0.85 | $p = 0.000$ | 0.78 | $p = 0.000$ | 0.32 |
| 10–30 | 18 | 135 | 93 | | | | | | |
| ≥10 | 1 | 10 | 2 | | | | | | |

[a]Improvement in pain intensity was defined as greatly improved (≤50 mm), much improved (30–50 mm), somewhat improved (10–30 mm), the same or worse (≥10).

The main problems in comparing the results of this study with others are the differences in the included patients and applied laser parameters. A meta-analysis by Yousefi-Nooraie and colleagues[33] considered nonspecific LBP, and there were no consistent conclusions. In addition, many other clinical studies have used LLLT for nonspecific chronic LBP; however a group of patients with nonspecific chronic LBP is very heterogenic, and the genesis of their pain is caused not only by pathological changes in the spinal and paraspinal structures, but also by complex neurophysiologic and psychosocial mechanisms. Studies by Gur and colleagues[34] and Klein and colleagues[35] compared the effects of LLLT with exercise effects in chronic LBP. Basford and colleagues[36] used doses recommended by WALT, and for the source of laser beams NdYAG[36] was used in a group of patients with nonspecific LBP. Soriano and Rios[37] examined patients with chronic LBP, and Toya and colleagues[38] conducted a study with the idea of achieving additional anti-inflammatory effect in a group of patients with acute pain. However, this group of patients was very heterogenic in the pathophysiological aspect, which made the results difficult to compare. In a study that investigated patients with acute pain and in which the effects of different therapies were compared, a 830-nm laser unit at a dose of 1 J was used. No changes in results were observed compared with ultrasound and traction therapy.[39] The classification of acute LBP to subtypes, based on symptoms and signs by Delitto and colleagues[40] with the idea of developing optimal exercise therapy treatment, could be important in further phases of treatment; yet it is not substantial enough for our study.

Hypothetically, the biological actions of LLLT are multiple: the reduction of inflammation as a primary effect with consecutive improvement in neurophysiologic features of comprised of nerves; the direct effect on nerve structures that accelerates recovery of the conductive block; and reflective effects with changes in the endorphin level. The results of clinical and experimental studies have shown that the anti-inflammatory effects are the most important. Studies have documented changes in biochemical markers of inflammation, distribution of inflammatory cells, and reduction in the formation of edema, hemorrhage, and necrosis after local laser irradiation with different sources of laser beams at 660, 684,[41] 780,[42] and 904 nm,[43] respectively, in experimentally induced inflammation models. Reduction in inflammation infiltrates, at the level of 30–50%, reaches its peak in 3–4 h following irradiation. This reduction is in positive correlation with a TNFa level decrease and is dose-dependent.[44] Comparison with anti-inflammatory drugs like meloxicam and indomethacin has shown similar anti-inflammatory effects.[45] The direct impact of LLLT on neural structures that are damaged by compression or inflammation should be considered an important additional effect.[46] These additional effects should be regarded as important in acute lesions of neural structures, such as acute lumbar radiculopathy, which are risk factors for neuropathic pain development.[47] Laser phototherapy at different wavelengths, especially at the 780-nm wavelength, of injured peripheral nerves significantly improves nerve recovery in animal[48] and clinical studies[49] and does not support positive effects for 904 nm. A study by Chen and colleagues,[50] who used a superpulsed 904-nm laser, reported negative effects on nerve recovery in rats.[50] This should be emphasized, considering the applied mode, timing, and pretreatment conditions of the irradiated tissue, especially for the superpulsed 904-nm laser. The influences of LLLT on the activity of antioxidative enzymes could also be a part of a modulation mechanism, considering the role of these enzymes in increasing the nonspecific resistance of cells to different damages.[51,52] The possibility of some positive interactions between LLLT and COX-2 inhibitors should be considered.[53]

Placebo response in this study is also very impressive, and it differs for the investigated parameters (for pain in leg about 30%, for Oswestry disability about 45%, accounted for as the difference between percentages of normal values). The placebo group showed better results compared with the only pharmacologically treated group. This fact highlights the roles of other factors in shaping the experience of pain. Some investigations have shown that cognitive expectations for pain relief and affective processes are involved in pain processing

Table 6. Distribution of Changes in the Oswestry Score Categories

| | Groups | | | Comparisons | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| *Improvement*[a] | *A* | *B* | *C* | *A–B* | | *A–C* | | *B–C* | |
| Yes | 151 | 33 | 98 | $\chi^2 = 180.4$ | *d* | $\chi^2 = 24.7$ | *d* | $\chi^2 = 86.2$ | *d* |
| No | 31 | 149 | 84 | $p = 0.000$ | 0.71 | $p = 0.000$ | 0.26 | $p = 0.000$ | 0.49 |

[a]Improvement was designated as switching from the moderate to the minimal disability category of the Oswestry score at the end of the therapy.

through the nervous system and in placebo analgesic responses.[54,55] Additional management in the placebo group compared with the only pharmacologically treated group might induce cognitive and affective response in treated patients.

The results of this study must be considered in light of the several limitations and the choosing of patients using relatively strict clinical forms: severe levels of pain and moderate levels of disability, because of the typical flow of patients for clinical treatment (selection bias). Randomization did not include nuclear magnetic resonance (NMR) findings, duration of symptoms, or other clinical and psychosocial characteristics of patients that could have influenced therapeutic response. Evidence from this study suggests only the short-term effects. Determination of the placebo effect could be considered controversial owing to the experimental conditions and without the untreated group.

Future studies could include patients randomized by levels of baseline disability and duration of symptoms. For long-term studies and consideration of long-term effects, randomization should include subgroups of patients in relation to functional findings, despite the lack of evidence for identification tests.[56] Future studies should evaluate many factors related to the disease, the patient's psychosocial characteristics, and procedure management that may reflect on treatment response and capability for recovery. The possibility for complete substitution of anti-inflammatory drugs by LLLT, in patients that are at high risk, should also be targeted in future studies. Moreover, for further confirmation of the anti-inflammatory effectiveness of phototherapy and the promotion of nerve recovery, future clinical trials need to define optimal therapeutic protocols for various clinical situations, in particular with respect to the characteristics of the laser setup, site of irradiation, and length of treatment.

## Conclusions

Treatment of acute LBP with radiculopathy at 904-nm LLLT at a dose of 3 J/point, proposed as additional therapy to nonsteroidal anti-inflammatory COX-2 drugs, has shown better improvement in local movements, more significant reduction in pain intensity and related disability, and improvement in quality of life, compared with patients treated only with drugs and with a placebo LLLT procedure, and with no side effects.

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## Author Disclosure Statement

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