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The Clinical Efficacy of Low-Power Laser Therapy on Pain and Function in Cervical Osteoarthritis

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Abstract: Pain is a major symptom in cervical osteoarthritis (COA). Low-power laser (LPL) therapy has been claimed to reduce pain in musculoskeletal pathologies, but there have been concerns about this point. The aim of this study was to evaluate the analgesic efficacy of LPL therapy and related functional changes in COA. Sixty patients between 20 and 65 years of age with clinically and radiologically diagnosed COA were included in the study. They were randomised into two equal groups according to the therapies applied, either with LPL or placebo laser. Patients in each group were investigated blindly in terms of pain and pain-related physical findings, such as increased paravertebral muscle spasm, loss of lordosis and range of neck motion restriction before and after therapy. Functional improvements were also evaluated. Pain, paravertebral muscle spasm, lordosis angle, the range of neck motion and function were observed to improve significantly in the LPL group, but no improvement was found in the placebo group. LPL seems to be successful in relieving pain and improving function in osteoarthritic diseases.

Keywords: Cervical osteoarthritis; Low-power laser therapy

Introduction

Pain is a major symptom in COA [1], the source of which is not unique. It originates from the posterior and posterolateral external fibres of the annulus fibrosus, the posterior longitudinal ligament, nerve roots, dura mater, apophysial joints and muscles [2]. Central pain mechanisms also contribute [3]. Pain reduces functional status by causing spasm in the surrounding muscles and by limiting the range of motion (ROM) of the neck. Thus it is reasonable to assume that an appropriate pain treatment will lead to an improvement in functional status by relieving these underlying problems.

According to experimental and clinical studies that have been performed since 1967, LPL has been claimed to have biostimulation and pain reduction effects [4]. The pain reduction effect of LPL is known to be due to its effect on pain sensation in the sensorial nerve endings. Moreover, it has been reported to have an increasing effect on β-endorphine stimulation [5]. LPL also widens the arterial and capillary vessels, stimulates electrolyte interchange in the cell protoplasm, increases oxygen consumption and enhances nucleic acid and protein synthesis [6].

Many authors have reported significant pain reduction with LPL in acute and chronic painful conditions such as rheumatoid arthritis, osteoarthritis, soft tissue disorders and postoperative pain [7]. However, some have failed to show such an effect in painful musculoskeletal pathologies [8,9]. Our aim was to evaluate the analgesic efficacy of LPL therapy and related functional changes in COA before putting this device to routine use.

Materials and Methods

Sixty patients between 20 and 65 years of age, admitted to Trakya University Hospital Physical Medicine and Rehabilitation Polyclinic with painful neck were included in the study. Patients with symptoms indicating COA, such as a history of mechanical localised neck

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pain, osteophytes, joint space narrowing, sclerosis of the vertebral margins and subchondral cysts were diagnosed as having COA [10]. Exclusion criteria were: patients with brachialgy and pure cervical disc herniation diagnosed by physical examination and CT; muscle weakness due to COA; other rheumatologic, neurological, metabolic, endocrine and neoplastic diseases; patients with myofascial syndrome, having painful taut bands in the cervical muscles.

Laser Device and its Application

The LPL device used in this study was the Endolaser 476 supplied by Enraf Nonius. It is a solid-state laser (Ga-As-Al) with a power output of 50 mW and wavelength 830 nm. The diameter of the laser beam at the focal treatment point was 1 mm. The laser was set to deliver a continuous form of energy at 0.90 J for each 1 cm^2 area. The beam was applied to 12 application points, equidistant and parallel to each other, descending in the midline of the paravertebral muscles. Six of the points were on the right side and six on the left. No special attention was paid to treating the trigger points. Each application point was treated for 15 s, and so the session lasted for 3 minutes a day. The therapy was stopped after the 10th consecutive day. Patients were randomised by a simple systematic method into two equal groups according to the therapy applied, i.e. LPL or placebo laser. The same applications were performed in the control group with the same device but without the laser beam working.

Analysis

Patients in each group were investigated in terms of pain and pain related physical findings, such as increased paravertebral muscle spasm, loss of lordosis and range of neck motion restriction before and after therapy. The functional changes were also evaluated after therapy. Changes with the therapy were evaluated by a physician blinded to the type of therapy.

Overall pain was evaluated by a visual analogue scale (VAS). Pain levels were labelled on a line in 10 categories, 10 points indicating unbearable pain and 0 no pain at all. Paravertebral muscle spasm was examined by the physician with manual pressure and was noted as either present or not. The lordosis angle of each patient was found by measuring the angle between a line connecting the top of zygomatic tubercle and the middle point of posterior margin of the foramen magnum, and a line tangential to the posteroinferior corner of the seventh cervical vertebra in lateral cervical radiograms [11]. Neck ROM was evaluated in both flexion and extension. Flexion was measured in terms of the distance from the midpoint of the chin to the apex of the sternal manubrium, in centimetres. Extension range was evaluated as the distance from the occipital tuberosities to the spinous process (PS) of C7. Functional status was assessed by the 'Neck Pain and Disability Scale' (NPDS) [12] before and after therapy.

Statistics

The findings were evaluated by using SPSS Version 8.0 for Windows. The average and standard deviation values were calculated by Student's *t*-test. The presence of differences in muscle spasm after therapy were evaluated by Pearson's χ^2 test.

Results

No significant difference was found between the groups in terms of age and gender. The mean age was 40.13 ± 10.31 and 40 ± 11.23 in the LPL group and the placebo laser group, respectively, ($p > 0.05$). The LPL group consisted of 26 females and 4 males; there were 24 females and 6 males in the placebo group ($p > 0.05$). Baseline values of pain, muscle spasm, lordosis angle, ROM and function were comparable in both groups ($p > 0.05$).

Pain levels in the LPL group decreased significantly after therapy, but no change was observed in the placebo

Table 1. Changes in pain levels, mean lordosis angles, ROM values and NPDS scores in the groups after therapy

Parameters	LPL group		Placebo group	
	Before therapy	After therapy	Before therapy	After therapy
Pain levels (VAS ± SD)	7.7 ± 1.3	2.4 ± 1.3[§]	7.3 ± 1.43	6.8 ± 0.98[†]
Lordosis angle (°)	39 ± 10.8	45.3 ± 10.8*	39.3 ± 10.7	41.1 ± 10.7[†]
Chin-manubrium distance (cm)	2.2 ±1.6	0.9 ± 0.8[§]	2.1 ± 1.7	2 ± 1.3[†]
Occiput-C7 PS distance (cm)	3.2 ± 1.7	1.4 ±1.1[§]	3.2 ± 1.7	3.1 ± 1.4[†]
NPDS[‡] score	82.6 ± 15.6	24.5 ± 7.6[§]	81.6 ± 14.8	74.8 ± 13.6[†]

Baseline values of all parameters did not differ significantly between groups.
[‡] NPDS, neck pain and disability scale.
[†] NS.
* $p < 0.05$.
[§] $p < 0.001$.

Table 2. Paravertebral spasm change after therapy

	LPL group		Placebo group	
	Before therapy	After therapy	Before therapy	After therapy
0 (absent)	3	23*	5	8†
1 (present)	27	7*	25	22†

Baseline values did not differ significantly between groups.
† NS.
* $p < 0.001$.

group (Table 1). Lordosis curves tended to improve after LPL, but the placebo group showed no significant improvement (Table 1). The flexion and extension ranges improved significantly in the LPL group, but not in the placebo group (Table 1). Paravertebral muscle spasm improved significantly in the LPL group, but not in the placebo group (Table 2). NPDS scores also improved significantly after LPL therapy, but no change was observed in the placebo group (Table 1).

Discussion

The analgesic effects of LPL therapy in musculoskeletal disorders are still being debated. Some authors report the efficacy of LPL therapy to be superior than placebo treatment in rheumatoid arthritis, post-traumatic joint disorders and myofascial pain syndrome [7]. Tam [6] reported excellent results in symptomatic COA as well, which was the subject of this study. However, not all authors have observed beneficial effects on pain, for example Krasheninnikoff et al. [8], who reported that LPL had had no superior effect over placebo in lateral epicondylitis. Moreover, the author concluded that studies investigating the analgesic effect of LPL therapy in musculoskeletal pain would be useless. This controversy may be related to the various efficacies of LPL therapy in different painful musculoskeletal conditions. However, opposite reports exist in many of the same pathologic conditions. For instance, Haker et al. [13] and Vasseljen et al. [14] reported beneficial effects from LPL therapy over placebo in tennis elbow. On the other hand, Siebert et al. [15] reported no superior analgesic effect of LPL therapy in tendinopathies. The same controversy can be noticed between Vecchio et al.'s [16] and van der Heijden et al.'s [17] reports on shoulder soft tissue pathologies. The latter have reported that LPL therapy was even better than ultrasound in the shoulder, whereas the former have suggested no beneficial effect over placebo. This list may be enlarged with the other musculoskeletal disorders, such as rheumatoid arthritis and myofascial pain syndrome.

Reports on the beneficial effect of LPL on osteoarthritis have been more consistent. Marks et al. [5] reported that LPL therapy seemed to be an extremely successful method of relieving symptoms in osteoarthritis and related disability in Russia and eastern Europe. Similarly, excellent results have been reported for COA and hip osteoarthritis, parallel to Walker et al.'s findings on the analgesic effect of LPL in sciatica and osteoarthritis [6,18]. The success of LPL therapy in osteoarthritis may be due to several mechanisms, one of which may possibly be through its positive effects on chondrocyte proliferation and matrix synthesis [5]. Reed et al. [19] observed macroscopic and microscopic smoothing of the fibrillated cartilage surface after laser irradiation in adult rabbits with mechanically induced degenerative knee arthritis. Skinner [20] reported that low-power Ga-As pulse laser had significant stimulatory effects on fibroblast function and enhanced connective tissue repair. These effects seem to be related to the biostimulation effect of LPL at the cellular level [21]. LPL has been suggested to increase the activation of cytoplasmic enzymes, oxygen consumption, ATP production and the synthesis of nucleic acids and proteins [22,23]. It has also been reported that LPL therapy had anti-inflammatory and anti-oedematous action owing to its reduction of prostaglandin synthesis. In particular its inhibition effect on prostacyclin has been reported to provide pain and inflammation regression, especially in acute exacerbations of osteoarthritis and sciatica [24]. It has also been suggested that LPL has effects on peripheral nerve stimulation and microcirculation regulation, interrupting the pain mechanisms and thereby providing analgesia [5]. The normalisation of the microcirculation and the speed of nerve transmission obtained have been reported to interrupt the vicious circle of the origin and development of the pain [4]. In some experimental studies pain thresholds have been shown to increase owing to laser application [25]. Konstantinovic et al. [24] demonstrated a spasmolytic effect of LPL in muscles. Thus LPL seems to break the pain–spasm–pain cycle by a dual effect. We found paravertebral spasm and pain levels to decrease in COA patients in this study, thereby supporting these claims. We also found lordosis angle to increase as a result of improvement in paravertebral spasm.

Bliddal et al. [26] demonstrated the pain reduction effect of LPL therapy, but could not manage to show its favourable effects on morning stiffness and joint performance in fingers with rheumatoid arthritis. In these cases pain did not seem to be the only deleterious factor on function. However, if the pain and spasm can be improved ROM limitation will dissolve and the function will improve in COA, as we managed to demonstrate in our patients.

Our success in pain reduction may be due to the laser modality, dosage and wavelength selection we used. In this study 10 sessions of laser therapy were applied in COA: the duration of therapy was similar to that of England et al. [27], in which nine sessions of laser therapy had been used in bicipital tendonitis pain. Some authors could not demonstrate a clear relationship between laser application methods and the dosages applied [7]. However, Tam [6] suggested a semiconductor or laser diode (Ga-As), which we used in this study, to be the most appropriate choice for pain reduction.

In conclusion, LPL therapy seemed to be an effective method of pain reduction in COA. It seems that a beneficial effect can be obtained by a specific power density and wavelength that can penetrate tissues. However, the mechanism of analgesia has not been clearly identified, and this study did not attempt to discover this point. Further investigations on the exact beneficial mechanisms of LPL, especially its effect on chondrocytes should be performed in the future.

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