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Effect of 850 nm He-Ne Laser Therapy on Nerve Conduction and Foot Planter Pressures Distribution of Painful Diabetic Neuropathy: A Randomized Controlled Trial

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Abstract

Introduction: Diabetic neuropathy patients are at very high risk for developing foot ulcer that may lead to lower extremity amputation and threaten the patient's life.

Objective: To evaluate the effects of scanning 850 nm He-Ne infrared laser on nerve conduction, pain intensity, and foot planter pressure distribution of painful diabetic polyneuropathy patients.

Methods: Thirty diabetic neuropathy patients with pain and reduced nerve conduction velocity were randomly divided into two groups; an experimental group (active laser group, n=15) and a control group (placebo laser group, n=15). Peak static and dynamic planter pressure were measured under heel, big toe and little toe. Sural and Peroneal nerves conduction velocity and amplitude and pain level were measured before and after treatment in both groups. The active laser group had got scanning 850 nm He–Ne infrared laser on foot planter surface and lumbosacral area with 5.7 J/cm^2 for 15 min/site/session, 3 session /week for four weeks.

Results: All measured parameters improved significantly in the active laser group, while no significant changes obtained in the control group. Comparison of post treatment measurements between groups showed that sural nerve conduction velocity and amplitude, pain level, and peak static and dynamic planter pressure were significantly higher in the experimental group compared with the control group. On the other hand there was no significant difference between groups for peroneal nerve conduction velocity and amplitude.

Conclusion: 850 nm He–Ne therapy with the applied parameter and technique was an effective modality for improving nerve conduction, redistributing foot plantar pressures and relieving pain of painful diabetic polyneuropathy patients.

Keywords: Diabetic neuropathy; Laser therapy; Foot pressure; Nerve conduction

Introduction

One of the most common complication of diabetes mellitus is Painful Diabetic Neuropathy (PDN). Within 10 to 15 years of diabetes approximately 50% of patients will develop PDN [1]. In neuropathy, there is a progressive degeneration of the peripheral nerves in the lower limbs especially, that leads to sensory and motor deficits [2]. This affects the biomechanics of the foot, that seen in the ankle kinematics [3], gait kinetics [4] and plantar pressure distribution [5]. According to literature review, there is a strong association between diabetic neuropathy and higher plantar loads that could be responsible for foot ulceration [6] and re-ulceration [7].

The onset of PDN could be delayed by excellent control of blood glucose [8]. After the onset of PDN, medical management focuses on prevention of foot ulcer and amputations [9]. Symptomatic relief of PDN was reported with the use of non-invasive medical treatments including near-infrared phototherapy [10], low level laser therapy (LLLT) [11], magnetic and electro-therapy [12]. It is still unknown the efficacy of most conservative treatment for PDN. The plantar pressure mapping technology can be utilized when evaluating the possible effect of specialized physiotherapy or a surgery operation by measuring the pressure distributions before and after the treatment. Among the different treatment option, LLLT may induce biostimulational effect on nervous system [13,14]. LLLT with different wave length was used for treatment of peripheral nerve injuries and for treatment of other diabetic complications [15,16] as it promotes nerve regeneration, improve neural function and vascularity. 850 nm He-Ne infrared laser therapy is a mixed laser that emit both He-Ne continuous with wavelength 850 nm and infra-red pulsed with wavelength 905 nm. There wasn't available research about this type of laser for treatment of PDN. The purposes of this study were to investigate the effect of scanning 850 nm He- Ne infrared laser therapy on nerve conduction, pain intensity, and foot planter pressure in PDN patients.

Methods

The study design and protocol were approved by the Ethics Committee of Faculty of Physical Therapy, Cairo University. The study design was a randomized control trial. It was started in January 2011 and ended in April 2011. Seventy DPN patients were recruited from the diabetic clinic in El Kasr EL Einy Hospital, Faculty of Medicine, Cairo University. Thirty patients were selected (20 women and 10 men) according to specific criteria.

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Inclusive criteria

- Longstanding Type 2 controlled diabetes with duration ≥10 years and blood glucose level ranged between 130-350 mg/l.
- Patients suffered from peripheral neuropathy for ≥6 months in lower limb with symptoms of pain, glove stock hyposthesia, burning sensation and spasm of foot muscles.
- Patients had abnormal nerve conduction study.

Exclusive criteria

Patients were excluded if they had;

- History of pedal ulcer, amputation and/or peripheral vascular diseases.
- Fractures or deformity of any bones of lower limb.
- Significant scar tissue or calluses on the feet.
- Osteoporosis.

Selected patients were randomly allocated to one of two groups using sealed envelopes with matching of age, sex, duration of diabetics and duration of neuropathy. There were two envelops A and B. A was active and B was placebo. The first patient chose one envelop either A or B and the matched patient of him located to the other group. Fifteen patients were allocated in experimental group (active laser group) and another fifteen were allocated into control group (placebo laser group). A diagram of the patients' retention and randomization is shown in Figure 1. The study sitting was Neurological outpatient clinic of the Faculty of Physical Therapy, Cairo University. All the patients signed consent forms. Patients was allowed to use analgesic medications but had to be unchanged for at least four weeks prior entering and during the study.

Prior to initiating the study, a sample size of 15 subjects per group was calculated to provide 80% power and a test size of 0.05 (two-sided) [17] to detect differences of treatment effect on the main investigation parameters (nerve conduction velocity and amplitude and peak static and dynamic planter pressure) between the 2 groups based on previous studies [18,19].

Physical and neurologic examinations were performed for patients by the same neurologist. Peak static and dynamic planter pressure, pain intensity and peroneal and sural nerve conduction studies were measured for both lower limbs before enter the study and after four weeks of the treatment by the same investigators.

The Toennis Neuroscreen Plus device was used to measure nerve conduction velocity of peroneal and sural nerves by conventional nerve conduction studies standard testing protocol [20].

Pain intensity was measured by Visual analog scale. RS scan foot plate system was used to measure the absolute peak static and dynamic plantar pressure under 3 areas on each foot (center of the heel, first metatarsophalngeal and fifth metatarsophalangeal). The system offer full details colour coded printout of the amount and percentage of peak pressure under the selected areas of the foot. The calculated area was referred the contact surface between the foot plantar surface and sensors (Figure 2).

For treatment, The Laser Scanner device (Italy ASA Co., Bravo Style) was used. This device emits mixed light of both He–Ne gas laser and infrared. He–Ne continuous with wavelength 850 nm, while infrared pulsed with wavelength 905 nm. The maximum power of device was 10 W. The device output was calibrated at each frequency with a power meter (Omega Laser Systems), and an I.R. Laser Detection Card.

Treatment procedure

The patient lay in comfortable prone position. Both the plantar surface of the feet and the lumbosacral area were treated by laser therapy. The dimensions of the lumbosacral area was marked by four points, one on the L2, one on the S1 and two points laterally to the spine by about 2 cm (Figure 3). Laser head was fixed at 30 cm away from treatment areas (shape of laser beam: round, r=0.6 cm, area=1 cm^2). These two areas were exposed to laser therapy through a sweeping computerized scanning at an angle of 30° ± 15° that deliver 850 nm He-Ne with 905 nm IR laser. The power density was 6.3 mW/cm^2 and irradiation time was set to 90 sec/cm^2 to achieve the total dose of 5.7 J/cm^2 through 15 min/site/session. Each patient received 12 sessions at a rate of 3 sessions /week. The same procedures were taken for the control group with the laser device OFF. All patients were treated under the same conditions, and individually to avoid the influence of one another.

Statistical analyses

All data are expressed as mean ± SD. Statistical significance was evaluated by two tailed Students t test (for paired and unpaired values). Analyses were performed using Graph Pad Prism, Version 18.0 on a personal computer. The significance level was set at $p \leq 0.05$.

Results

The demographic data of the patients is shown in Table 1. At beginning of the study, there were no statistically significant differences between groups in age, weight, height, BMI, duration of diabetes, duration of neuropathy and pain intensity.

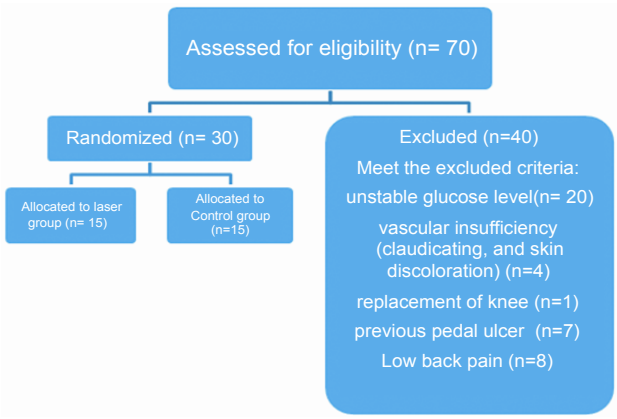

Figure 1: Flow chart of patient elicitaion.

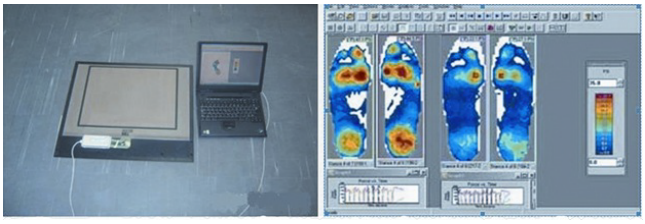

Figure 2: Foot scan plate system.

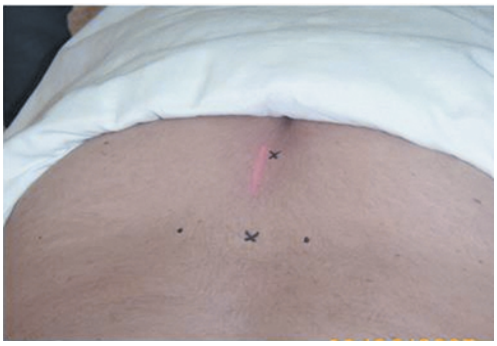

Figure 3: Application of laser therapy on lumboscral area.

Variables	Groups	Mean ± SD	MD	t	p- value
Age (years)	LG	53.13 ± 3.356	1.93	1.114	0.2[a]
	CG	51.2 ± 5.69			
Weight (kg)	LG	84.0 ± 7.64	0.06	1.33	0.3[a]
	CG	84.06 ± 6.76			
Height (cm)	LG	167.5 ± 4.98	0.07	1.48	0.2[a]
	CG	168.2 ± 3.58			
BMI (kg/m^2)	LG	29.9	0.2	1.4	0.1[a]
	CG	29.7			
Duration of diabeties mellitus (months)	LG	12.26 ± 2.9	0.6	0.4861	0.6[a]
	CG	11.66 ± 3.8			
Duration of Neuropathy (months)	LG	11.73 ± 0.51	0.27	1367	0.18[a]
	CG	12 ± 0.57			
Sex n (male/ female)	LG	05-Oct		-	1:00 AM
	CG	05-Oct			
Pain level intensity (cm)	LG	7.33 ± 0.61	0.13	0.61	0.61[a]
	CG	7.2 ± 0.77			

M ± SD: Mean ± Standard Deviation; MD: Mean Difference; LG: Laser Group; CG: Control Group; [a]Non-significant. [b]Significant.

Table 1: Demographic characteristics of patients.

Electrophysiological parameters results

Pre-treatment, there was no significant difference between the groups for either nerve conduction velocity or amplitude for peroneal nerve (Table 2). Sural nerve was absent in 22 patients and present in 8 patients of the two groups but with abnormal values (reduced conduction velocity and amplitude).

Post-treatment, sural conduction velocity was present in all patients in the active laser group and still absent in the control group.

In the active Laser group, the peroneal and sural nerve conduction velocity and amplitude were increased significantly (p=0.001, 0.0001, 0.002 and 0.0001 respectively). In the control group, there was no significant change (p=0.09 and 0.07, respectively) (Table 2).

Comparing the post-treatment results between the two groups found that the sural nerve conduction velocity and amplitude were significantly higher in the active laser group compared to placebo laser group (p =0.0001), while there was no significant difference for peroneal nerve conduction and amplitude between the group (p = 0.1) (Table 2).

Pain level results

Both groups noted a decrease in mean pain scores after four weeks of treatment, with a statistically significant difference between groups post treatment in favour of the laser group (p= 0.0001) (Table 2).

Peak planter pressure results

At pre-treatment measurements, there were no significant difference (p > 0.05) between groups at heel, big toe and little toe for both static and dynamic planter pressure (Table 3).

In the laser group, the static and dynamic peak planter pressure at the three points were decreased significantly (p = 0.001) with no significant change in the control group (p > 0.05) (Table 3).

Post-treatment measurement comparison between groups found a highly significant difference at heel, big toe and little toe for both static and dynamic planter pressure (p = 0.001, 0.001 and 0.009 respectively) in favour of the laser group (Table 3).

Discussion

High foot plantar pressures in association with sensory and motor deficit have been ascertained to be the important risk factors for developing foot ulcer in the PDN patients [21,22]. This study was

Variables	Groups		Pre M ± SD	Post ± SD	t	p- value
Peroneal nerve MCV (m/sec)	LG		46.3 ± 4.6	50 ± 6.7	4.097	0.001[b]
	CG		47.1 ± 5.3	46.6 ± 5	1.807	0.009[a]
	MD	pre	-0.8		0.372	0.7[a]
		Post		3.373	1.55	0.1[a]
Peroneal nerve amplitude (mv)	LG		1.3 ± 0.7	1.7 ± 0.6	3.788	0.002[b]
	CG		1.6 ± 0.78	1.5 ± 0.8	1.910	0.07[a]
	MD	Pre	0.27		0.976	0.3[a]
		post		0.316	1.24	0.2[a]
Sural nerve SCV (m/sec)	LG		5.1 ± 9.2	36.2 ± 12.4	10.234	0.0001[b]
	CG		5 ± 9.1	4.5 ±8	1.258	0.2[a]
	MD	pre	0.133		0.0397	0.9[a]
		post		31.813	8.355	0.0001[b]
Sural nerve amplitude (mv)	LG		5.6 ± 10.1	26.1 ± 12.7	7.851	0.0001[b]
	CG		1.6 ± 2.9	1.7 ± 3	1.835	0.08[a]
	MD	pre	4.353		1.6111	0.1[a]
		Post		23.240	6.375	0.0001[b]
Pain intensity level (cm)	LG		7.2 ± 0.77	5.33 ± 0.9	7.29	0.0001[b]
	Maximum		9	8		
	Minimum		6	3		
	Maximum		9	9		0.08[a]
	Minimum		6	4		
	MD	pre	0.13		0.48	0.63
		Post	1.6		5.23	0.001[b]

M ± SD: Mean ± Standard Deviation; MD: Mean Difference; LG: Laser Group; CG: Control Group; [a]Non-significant. [b]Significant.

Table 2: Electrophysiological parameters and pain level mean values of the groups pre and post treatment.

Variables		Groups		Pre M ± SD	Post ± SD	t	p-value
Peak static planter pressure (N/cm²)	Heel	LG		10.73 ± 2.46	7.46 ± 1.64	10.87	0.001[b]
		CG		10.0 ± 2.0	9.73 ± 1.79	1.16	0.26[a]
		MD	pre	0.73		1.79	0.09[a]
			Post		2.27	5.55	0.001[b]
	Big toe	LG		6.66 ± 2.25	5.0 ±1.73	6.61	0.001[b]
		CG		7.06 ± 1.33	6.86 ± 1.18	1.14	0.27[a]
		MD	Pre	0.4		0.7	0.49[a]
			post		1.86	4.29	0.001[b]
	Little toe	LG		6.06 ± 1.43	4.46 ± 1.43	9.79	0.001[b]
		CG		6.53 ± 1.72	6.2 ± 1.2	1.58	0.13[a]
		MD	pre	0.47		3.74	0.45[a]
			post		1.74	3.74	0.001[b]
Peak dynamic planter pressure (N/cm²)	Heel	LG		24.73 ± 3.93	20.33 ± 3.65	12.13	0.001[b]
		CG		25.6 ± 5.27	25.53 ± 5.22	0.29	0.77[a]
		MD	pre	0.87		0.65	0.52[a]
			Post		5.2	3.99	0.001[b]
	Big toe	LG		22.8 ± 4.69	17.4 ± 3.96	14.89	0.001[b]
		CG		22.46 ± 4.79	22.6 ± 4.56	0.61	0.54[a]
		MD	pre	0.34		0.25	0.8[a]
			Post		5.2	4.98	0.001[b]
	Little toe	LG		17.6 ± 2.35	13.33 ± 2.16	15.02	0.001[b]
		CG		17.06 ± 4.93	16.86 ± 4.91	0.89	0.38[a]
		MD	pre	0.54		0.46	0.65[a]
			Post		3.53	3.01	0.009[b]

M ± SD: Mean ± Standard Deviation; MD: Mean Difference; LG: Laser Group; CG: Control Group; [a]Non-significant. [b]Significant.

Table 3: Peak static and dynamic planter pressure distribution mean values of the groups pre and post treatment.

designed to examine the effects of scanning 850 nm He–Ne IR laser to treat patients with diabetic polyneuropathy. These effects were measured objectively by foot scanning to determine peak static and dynamic planter pressure, electroneurography to measure peroneal and sural NCV and amplitude and subjectively by VAS to reflect pain intensity level. The results of foot scan are reliable, reproducible and accurate [23]. The electrophysiological evaluation of both nerves sural and peroneal serve as a simple and effective diagnostic tool for diabetic polyneuropathy [24]. Peroneal conduction velocity correlates well with sural conduction velocity, and sural nerve latency is often absent in patients with reduced peroneal conduction velocity [25]. VAS it is the most common and reliable type of pain scale [21].

The study found that in the active laser group, both peroneal and sural nerves conduction velocity and amplitude were significantly increased, while no significant changes in the control group. Also foot planter pressure and pain intensity level were significantly decreased in the laser group only. Post-treatment comparisons between groups found that sural conduction velocity and amplitude, foot static and dynamic planter pressure and pain intensity were significantly higher in the active laser group than the placebo laser group, while no significant difference was recorded in either peroneal nerve conduction velocity or amplitude between groups.

The improvement in the electrophysiological parameters in the Laser group could be explained as follows; laser therapy can stimulate nervous system [13]. The typical aetiology of DPN starts as injury to a peripheral nerve. Research findings suggested that LLLT enhances re-innervation of nerve injury [13]. Prathap et al. [26] found that LLLT of 3 and 4 j/cm² was effective for regeneration of both motor and sensory nerve conduction velocity of experimentally induced diabetic neuropathy as compared with control group. Also Rochkind et al. [27] found that 15 min application of laser therapy transcutaneously to both the site of nerve injury and to the corresponding segments of the spinal cord improves function recovery and recruitment of voluntary muscle activity. Other studies concluded that laser irradiation allows higher metabolism for nerve, prevents degeneration of motor cell, induces proliferation of Schwann cell, allows and increases myelinization and axon regeneration [13,28,29]. The other possible mechanisms of laser action toward tissue regeneration were due to: (1) increased activity of leukocytes and phagocytes, and increased calcium in the cell cytoplasm; (2) interacted with cytochromes that stimulating redox activity in the cellular respiratory chain and resulting in cell activation, (3) accelerated cell division and growth; (4) activation of protein and cytokine synthesis; (5) enhances production of adenosine triphosphate (ATP), which stimulates the cells' mitotic activity; (6) enhances vasodilatation and cutaneous blood microcirculation [30,31] by photolysis of complexes such as nitric oxide [13,30]. Furthermore an intriguing hypothesis could be that the improvement in cutaneous blood flow might be mirrored by a similar effect at the endoneural level. Carmeliet [32] demonstrated that blood vessels and nerves use similar signals and principles to differentiate, grow, and navigate towards their targets, therefore they could also show synergistic responses to a common stimulus such as that induced by laser.

The better result found in sural nerve than in peroneal nerve may be indicate that (1) Laser therapy started its effects peripherally in small superficial nerve fibers which reflected on improvement of sural conduction velocity and amplitude [18]. (2) Sural conduction velocity is measured through one site of stimulation and recording while peroneal conduction velocity is measured through two sites of stimulation and recording that subtract the distal latency of peroneal nerve, which may reflect the peripheral laser effect [20]. This finding correlates with the results obtained by Khullar et al. [33] who found actual function recovery in rats with compressed sciatic nerve without significant change in the evoked compound action potentials of the common peroneal nerve; (3) Anatomically, the sural nerve is superficial sensory nerve that is easily influenced by laser therapy through both direct application to its branches on the plantar surface and to its origin through lumbosacral application. While the peroneal nerve is a deep motor nerve and the laser therapy could influenced it only indirectly through lumbosacral application. Parallel to the study's findings, statistically significant improvements were found in sensory conduction velocity, sensory and motor distal latencies of median nerves in carpal tunnel syndrome treated by laser [34]. This finding is consistent with some results of Peric´ and Cvetkovic [19] who concluded that LLLT had an indirect influence on the sensory axons function of the ulnar nerve in patients with painful DPN, where LLLT significantly increases the neural potential amplitude of ulnar nerve. Lazovic et al. [35] reported a significant improvement in sensory nerve velocity, and sensory and motor distal latencies in patient with carpal tunnel syndrome treated by 830 nm and 780 nm LLLT with intensity 3.6 J/cm² and 2.7 J, 3.4 J/cm²/point respectively.

Regarding the effect of 850 nm He–Ne laser on foot planter pressure distribution, the study found that the peak static planter pressure was decreased about 30.47%, 24.92% and 26.40% under the heel, big toe, and little toes respectively. Also the peak dynamic planter pressure was decreased about 17.79%, 23.68% and 24.26% under the three areas respectively in comparing with baseline.

Diabetic patient with neuropathy has diminished or lose of sensation and pain beside atrophy of intrinsic muscles of the foot. This causes instability of the metatarsophalangial joints (MTPJs), anterior migration and displacement of the fat pad which is normally

located under the metatarsal heads that leads to change in distribution of weight on planter surface and makes the foot more vulnerable to injury from accumulated trauma during walking [4]. The reduction in static and dynamic planter pressure after 850 nm He Ne laser therapy treatment can be explained as laser improve peripheral neural function of both sensory and motor nerve with significant decrease in the pain level. Previous researchers found that athermic laser therapy induced a significant reduction of pain and increase in skin microcirculation in diabetic microangiopathy patients [18,36]. All of these effects can be reflect clinically and functionally to readjust distribution of planter pressure during standing through decrease static planter pressure and during walking by decrease dynamic planter pressure.

The alleviation of neuropathic pain in diabetic neuropathy is very difficult, at the same time the previous studies had not used a treatment program based on anatomic, neuropath- physiological and vascular source of neuropahtic pain. In this study patients receiving 850 nm He Ne scanning laser by the applied technique had a 35% decrease of pain level after four weeks of treatment. This result is consistent with results of Nakamura et al. [37] demonstrated that LLLT was an effective form of treatment for pain.

The exact mechanisms how LLLT relieves pain is unknown. It may be by increasing production of ATP, serotonin and endorphins and consumption of cellular oxygen, anti-inflammatory effects and improving blood circulation in some cases [38]. Also *in vivo* and in vitro studies evidence that 830 nm laser inhibits Ad and C nerve fibre transmission [39]. The aetiology of the painful diabetic neuropathy is through abnormal activation of damaged nerve fibres, and the perception of this pain is dependent on neurotransmission in the dorsal horn of the spinal cord [40]. Laser treatment would block the abnormal activity in the affected peripheral nerve or block neurotransmission in the somatotopically related dorsal horn through application of laser on the corresponding segment of the spine (lumbosacral application) and direct stimulation of sensory peripheral nerves (foot plantar surface application) is believed to produce analgesia through both of these application.

This study was limited by small sample size and short time treatment due to the ethical consideration as the control placebo group didn't receive treatment to limit the variable that could affect the result. Future research is need with large sample size using different Laser wave length with the same technique of application.

Scanning He–Ne laser therapy with 850 nm that applied peripherally and centrally was an effective modality for improving nerve conduction, relieving pain and redistributing foot plantar pressures of painful diabetic polyneuropathy patients.

Declaration of Interest

There are none.

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