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Effect of Low-Intensity Laser on the Neuropathic Common Peroneal Nerve Post Burn.

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Abstract : Purpose: to determine the effect of low intensity laser therapy (LILT) on the neuropathic common peroneal nerve postburn. **Methods of evaluation:** Measurement of the motor conduction velocity(MCV) of the common peroneal nervein meter/ second. **Methods:-** Thirty patients (20 males and 10 females) ranging in age from 20 to 35 years, they were selected from the out-clinics of Kasr-El-Aini (Cairo University hospitals) and Om-Al-Misrieen hospital (Ministry of Health),patients were notfamiliar with the technique LILT and suffering from burns of chronic phase (post-hospitalization period), affecting lower limbs, with the percentage of total body surface area (TBSA) ranging from 20% to 30% and their early diagnosis was a burn of 2nd or 3rd degree and complicated with peripheral mononeuropathy affecting the common peroneal nerve. They were randomly divided into 2 equal groups in number, one study group (A) and a control one (B). the study group formed of 15 patients to which the LILT was applied (20 minutes in each session 3 times per week for 2 monthsas a total period of treatment), while the control group was formed of 15 patients to which the placebo LILT was applied. Measurements were conducted before starting the treatment as a first record and at the end of the second month of treatment as a second (final) record. **Results and conclusion:-** Results showed that application of the LILT had a valuable improving effects on the neuropathic common peroneal postburn as evidenced by the highly significant increases in the common peroneal nerve motor conduction velocity in meter/ second.
Key words : (Low intensity laser therapy, Neuropathic common peroneal nervepostburn and Motor conduction velocity).

Introduction

Burned patients suffer from many problems due to disruption of the normal protective functions of the skin, injury to the vascular tree and blood elements, and severe metabolic stress with abnormal capillary permeability, protein-rich fluid extravascular oedema and low cutaneous blood flow. A burn injury can have devastating effects on the neuromuscular system. Patient complaints regarding weakness or lack of sensation often are rationalized as generalized sequelae of the burn injury and healing process. However, these symptoms may be due to peripheral neuropathies resulting from either impaired nerve axons, or myelin sheaths, or both,[1, 7,8.]

All of the above burn complications plus the lymphatic damage will delay healing of the burn wound in the acute phase (before wound closure), as well as in the chronic phase which begins with wound closure and continues until the full maturation of the wound (from 1 to 2 years post -hospitalization) and lead to the development of contractures which affect the physical function of the burned patient finally. Peripheral neuropathies have been observed following thermal injury and most often affect nerves under the area of the burn, peripheral neuropathies are usually seen in patients with burns greater than 20 % of total body surface area (TBSA). The occurrence of multiple mononeuropathy after thermal burns covering greater than 20 % of TBSA is common and the number of nerves involved per patient ranged from 3 to 7. The source was believed to be due to a multiple crush syndrome, in which multiple different neuropathic factors in each patient summate to cause a multiple mononeuropathy, [1,9, 10.]

Also the hypermetabolic response of the burned patients, has been suggested as a cause of the peripheral neuropathies, as the basal metabolic rate (B.M.R) of the burned patients increase in excess of 2 to 2.5 times normal (Normal B.M.R. equals 40 ± 10 % $C/m^2/hr$), which contributed to the excess of the circulating catecholamines, that increase the sympathetic tone in burned patients, leading to increased systemic vascular resistance, decreased cutaneous, muscular and endoneurial blood flow (Nerve blood flow) resulting in nerve function alterations, [1,7,8,9,14,15,16,22.]

Clinical electrophysiology began towards the end of the eighteenth century with Galvani's discovery of animal electricity and has since progressed steadily during the past two centuries. Electrophysiological assessments of muscle and nerve are now considered indispensable in the practice of neurology, physical-therapy, and other related clinical disciplines. The clinical measurement of nerve conduction velocities has become increasingly popular since the late 1940s, a 1948 article by Hodes et al, contained the first clinically relevant discussion of conduction velocity Testing and created great interest in the subject of electrophysiological testing. Form the mid-1940s to the present, there has been an expanding use of the clinical electrophysiological testing in the form of nerve conduction velocities and electromyography studies, where both are good examples of how technology has been applied in clarifying clinicalproblems, [2,3,4,5,6,11,12,13,17,18,19,20]

Physical therapists use low-intensity laser therapy (LILT) to treat patients with chronic inflammatory and fibrotic conditions. The literature on reduction mammoplasty, however, does not mention the use of LILT as a potential treatment for fibrotic breast lumps. Cellular studies support the use of LILT to improve absorption of extracellular fluid, increase neutrophil activity and chemotaxis, increase secretion of macrophage growth factors, enhance DNA synthesis, decrease pain, enhance electronrespiratory chain reaction, increase endothelial PGI_2 secretion and degrade fibrin networks. Degradation of clotted blood and enhanced absorption of hematomas by improved circulation were the mechanisms by which LILT improved survival of grafted skin in cases where hematoma had developed, [23,24, 25,26.]

High dosages of LILT generally have an inhibitory effect on tissue metabolism. Dosages of 8to 32 J/cm^2 are used to treat chronic inflammation and reduce the risk of scar formation in musculoskeletal injuries. To reduce true keloid and hypertrophic scars, 830-nmLILT is applied at a dosage of 30 J/cm^2, in combination with the use of pressure tapes or dressings and steroid injection. Patients usually received LILT twice weekly, and treatment frequency was reduced once they judged that "real improvement" had occurred; total treatment was for periods of up to 1 year. The effect of LILT on scar formation has not been evaluated in a controlled study, and treatment of fibrosis or calcification secondary to hematoma or fat necrosis is not specifically mentioned in the LILT literature, [27,28,29.]

Material and Methods

Subjects:

Thirty patients (20 males and 10 females) ranging in age from 20 to 35 years, they were selected from the out-clinics of Kasr-El-Aini (Cairo University hospitals) and Om-Al-Misrieen hospital (Ministry of Health),patients were not familiar with the technique LILT and suffering from burns of chronic phase (post-hospitalization period), affecting lower limbs, with the percentage of total body surface area (TBSA) ranging from 20% to 30% and their early diagnosis was a burn of 2nd or 3rd degree and complicated with peripheral mononeuropathy affecting the common peroneal nerve. They were randomly divided into 2 equal groups in number, one study group (A) and a control one (B). the study group formed of 15 patients to which the LILT

was applied (20 minutes in each session 3 times per week for 2 months as a total period of treatment), while the control group was formed of 15 patients to which the placebo LILT was applied. Measurements were conducted before starting the treatment as a first record and at the end of the second month of treatment as a second (final) record, [4,5,8,9,16,21,24,25.]

Treatment instrumentation:

The treatment protocol was achieved by using laser unit that was a small hand held machine((Gallium arsenide (Ga-As)), class III laser product under the existing requirements of the United States food and drug association regulation. It manufactured by Laserex technology pty ltd Australia. The machine offers two types of laser therapy (continuous and pulsed). Continuous laser therapy is of a common use and many studies had found it effective. The maximum output is determined by the probe connected and the power could be settled. The device has the following treatment parameters: Laser type: Gallium arsenide (Ga-As), maximum average power: 5 milli Watts, wave length: 820nm, maximum repetition rate: 5 kHz, energy density: up to50 J/ cm^2, beam spot size: 0.1256 cm^2 and power density: 0.39 W/ cm^2 in addition to the Protective goggles: Safety glasses type (5213693) for patients and the physical therapist while applying the treatment, [26,27,28,29.]

Procedures

Evaluation:

For measuring the motor conduction velocity of the common peroneal nerve, The Neuropack 2 MEB-7102K, will be utilized to obtain an objective evaluation of the motor conduction velocity.The Neuropack 2 MEB-7102K is designed to be a compact, self-contained unit. It is composed of a main unit featuring high-performance 2-channel amplifiers, a multi-purpose stimulator, a floppy disk drive, a high-resolution thermal array recorder, a sophisticated electrode junction box with isolation amplifiers and an articulated arm; and an optional cart with a shelf for optional keyboard and a drawer for accessories is available. This configuration was satisfactorily provided the patient care staff with the capabilities for both evoked response testing, including somato-sensory evoked potential, auditory evoked response, visual evoked response, trending of the said evoked phenomena, nerve conduction velocity, and a range of EMGs. This compact mobile tower is easily transported by a single person to a patient who needs evoked response testing/EMG examination,[2,4, 5,8,10,11.]

Position of subject and electrodes: Upon arrival, the subject was asked to lie in supine position on a therapeutic plinth for approximately 5 minutes for rest and relaxation and to be familiar with the environment.

Recording electrodes: The active recording electrode was placed over the main bulk of the extensor digitorum brevis (EDB) muscle (located in the anterolateral aspect of the proximal midtarsal area). The reference electrode was placed distally over the small toe, while the ground electrode was placed over the medial portion of the foot.

Stimulating electrodes: In the distal stimulation: the stimulating cathode was placed 8 cm proximal to the active recording electrode to provide a standardized distal latency segment.**In the proximal stimulation:** The stimulating cathode was placed distally in the lateral part of the popliteal fossa (just medial to the biceps femoris tendon).Recording electrodes as well as the ground electrode were moistened with jelly and firmly fixed in their places by an adhesive plaster, [12,13,14, 15,16,17.]

Experimental technique of the common peroneal nerve MCV measurement: The MCV recording technique was conducted in an air conditioned room, where a thermometer was available during the whole time of the experiment to notice the ambient testing room temperature which was adjusted within a comfortable, reasonable and narrow range between 24 ^oC to 28 ^oC by setting the thermostat of the air-condition, and thus the temperature gradient along the course of the tested nerve was minimized**.** The controlled temperature of the testing room and warming the tested extremity by a deep stroking massage for 5 minutes was conducted, and thus the temperature-related variability was eliminated. Subject was asked to lie supine for approximately 5 minutes for leg massage, rest and relaxation as well as to be familiar with the environment of the testing room. The on-off switch of the EMG machine was turned on and the EMG12 program disk was inserted to load the EMG12 software program. All needed electrodes for the test were applied, so electrodes were connected to the positive and negative input of the active channels of the EMG head-box as well as the common electrode. The program was moved automatically to the ready condition of the neurography 1 motor NCV test after pressing any key for one time plus the enter key for 2 times on the alphanumeric keyboard**.** The electrode impedance for the active

channels was checked, to be less than 5 k Ohm and the data acquisition process was started after pressing the run key on the keyboard. The stimulus was gradually increased till the figure (trace) was obtained and then the stop key was pressed and the cursor was moved to measure latency. Print key was pressed as well as the paper key that allow paper to be advanced. During all recordings the sensitivity used was 1 mv/ division, and the sweep speed was 3 ms, while the stimulus intensity was ranged from 0.1 to 99 mA and stimuli were used to gain supramaximal recording. Recording of the segmental distances between the points of stimulation (marked by a marker pen midway the 2 stimulating electrodes) was measured by a tape measure**.** Nerve conduction velocity was calculated by the following formula: Conduction velocity = L1-L2 ÷ distance in (cm) × 10 Where: L1 = proximal latency and L2 = Distal latency, [2,4,5, 10,12,15.]

Treatment protocol including position of patient and position of the low-intensity laser therapy (LILT) in the study group (A): Patients were treated as outpatients and received full explanation about the purpose of the treatment, the therapeutic and physiological benefits of the low-intensity laser therapy (LILT). The subject was relaxed in supine position with the hips were adjusted in slightly flexed and laterally rotated position, knees were adjusted also in slightly flexed position (only 10^{o}) and slightly planter flexed ankles, with a pillow under the subjects head and so the comfortable patient's position was obtained. Lateral boundary of the popliteal fossa and lateral surface of the fibular head were irradiated by the continuous mode and direct contact method with the laser probe was applied without pressure to avoid the patient's complain of tenderness, 3 times per week for 2 months. Each region was irradiated for 10 minutes, the laser applicator was applied to the surface before switching on the machine, it was important to maintain the laser applicator in contact with the surface, so that the beam was applied perpendicular to points to achieve maximum penetration. The device was switched off before removing the applicator from contact point, [23,24,25, 26,27,28,29.]

Data analysis:

Common peroneal nerve motor conduction velocity was measured before and after the treatment program and the collected data were fed into computer for the statistical analysis; descriptive statistics as mean, standard deviation, minimum and maximum were calculated for each group. The t-test was done to compare the mean difference of the two groups before and after application and within each group. Alpha point of 0.05 was used as a level of significance, [30.]

Results

As shown in table (1) and figure (1), the mean value of the MCV before treatment was (33.123 ± 2.321) meter/ second in the study group (LILT group), while after treatment was (38.232 ± 2.861) meter/ second. These results revealed a highly significant increase in MCV ($P < 0.0001$). While in the control group (Placebo LILT group), the mean value of the MCV before treatment was (33.120 ± 2.011) meter/ second, while after treatment was (33.122 ± 2.013) meter/ second. These results revealed non-significant reduction in the MCV ($P > 0.05$).

Table (1): Comparison of the mean values of the motor conduction velocity (MCV) in meter/ second before and after treatment in the study and control groups

	Before treatment		After treatment		Mean difference	T-value	P.value	Level of significance
	Mean	± SD	Mean	± SD				
Study group (LILT group)	**33.123**	**2.321**	**38.232**	**2.861**	**-5.10900**	**-5.37**	**0.0001**	**Highly significant decrease**
Control group (Placebo LILT group)	**33.120**	**2.011**	**33.122**	**2.013**	**-0.002000**	**-0.00**	**0.998**	**Non-significant**

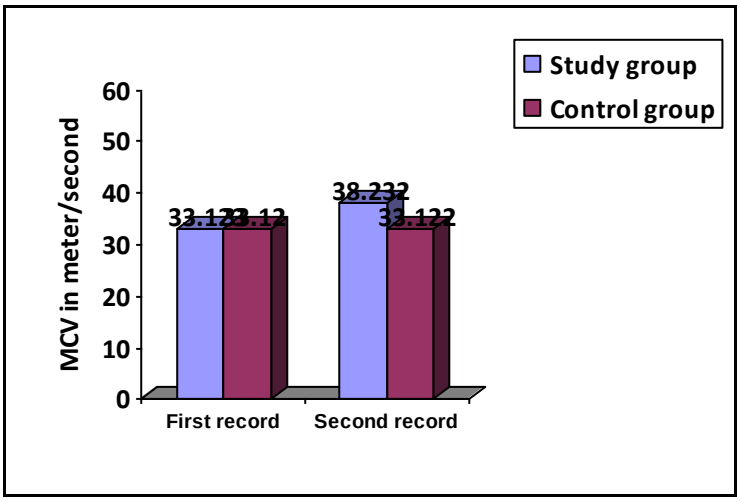

Fig (1): Mean values of themotor conduction velocity (MCV) before and after treatment in both groups.

Discussion

The basic pathophysiological consequence of the burn injury is the loss of the capillary integrity, localized increase in the micro vascular permeability, generalized impairment in the cell membrane resulting in cell swelling and increase osmotic pressure of the burned tissues leading to further fluid accumulation and oedema formation, which is a result of the outpouring of the intravascular fluid into the interstitial spaces. This process occurs at all areas of partial skin thickness burns and at the areas which are adjacent to and subjacent to the full skin thickness burns,[1,7,8.]

Burn patients suffer from many problems due to disruption of the normal protective functions of the skin, injury to the vascular tree and blood elements, severe metabolic stress with abnormal capillary permeability, protein rich fluid extra vascular or edema and low cutaneous blood flow. A burn injury can have devastating effects on the neuromuscular system. Patient's complaints regarding weakness or lack of sensation often are rationalized as generalized sequelae of the burn injury and healing process. However, these symptoms may be due to peripheral neuropathies and entrapment syndromes resulting from impaired nerve axons, or myelin sheath or both[9, 10,11].

Mononeuropathies and entrapment syndromes have been observed following thermal injury and most often affect nerves under the area of the burn, and they are usually seen in patient with burn greater than 20% of total body surface area (TBSA). The occurrence of entrapment syndromes or multiple mononeuropathies after thermal burns covered greater than 20% of TBSA is common and the number of nerves involved per patient ranged from 3 to 7 nerves. The source was believed to be due to multiple crush syndromes, in which multiple different neuropathic factors in each patientsummate to cause a multiple mononeuropathies or entrapment neuropathies[12, 13, 14, 15].

Electrodiagnostic studies (e.g., EMG, nerve conduction studies) remain the criterion standard for objective evaluations of neuropathic conditions. These studies are not without flaws; they are highly operator-dependent and the results do not always correlate with the severity of symptoms or patient outcomes. Despite these drawbacks, they may help confirm equivocal physical examination findings or help isolate the specific site of compression preoperatively. EMGs also may be used to verify progression or resolution in neurophysiology following surgical release. Common compressive neuropathies affect the median nerve in the form of pronator syndrome, anterior interosseous syndrome and the carpal tunnel syndrome; also the common compressive neuropathies affect the ulnar nerve in the form of cubital tunnel syndrome and ulnar tunnel syndrome. Common compressive neuropathies also affect the radial nerve in the form of radial tunnel syndrome, posterior interosseous syndrome and superficial radial nerve syndrome. Sites of compression of the pronator syndrome as an example of the median nerve entrapment syndrome includes the lacertus fibrosus, bicipital aponeurosis, superficial forearm fascia , the Struthers ligament, thickened or aberrant origin of pronator teres from distal humerus, the pronator teres (musculofascial band or compression between 2 muscular heads), and the proximal arch or the flexor digitorum superficialis.[17,18,19.]

The common peroneal nerve is most susceptible to trauma in the lateral aspect of the knee. Leg-crossing causes compression of the nerve against the head of the fibula. Injury at this level most frequently affects the deep branch, and less commonly, the whole nerve. Selective involvement of the superficial branch is rare. A ganglion in the same location may damage the nerve. Common peroneal nerve palsy may also occur as a result of prolonged squatting, which compresses the nerve among the biceps tendon, lateral head of the gastrocnemius, and the head of the fibula, [12, 13,14,15.]

Laser phototherapy uses radiation both in the visible (400-700 nm) and in the near-infrared (700 - 1000 nm) regions of the spectrum. When a photon is absorbed by a molecule, the electrons of that molecule are raised to a higher energy state. This excited molecule must lose its extra energy, and it can do this either by re-emitting a photon of longer wavelength (i.e., lower energy than the absorbed photon) as fluorescence or phosphorescence, or it can lose energy by giving off heat, or it can lose energy by undergoing photochemistry. Photobiological responses are the result of photochemical and/or photophysical changes produced by the absorption of non-ionizing radiation[, 23,24, 25]**.**

LLLT devices include the gallium arsenide(Ga-As), gallium aluminum arsenide infrared semiconductor (Ga-Al-As), and helium neon (He-Ne) lasers. The 632.8 nm wavelength He-Ne laser emits visible red light and may have a shallow penetration into skin. The Ga-Al-As, infrared laser has a longer wavelength than red beam laser and may have deeper tissue penetration. The 904 nm wavelength Ga-As laser is most commonly used for pain and inflammation because it has the deepest tissue penetration. As a result, it may be less suited for wound healing[, 26,27.]

The amplification process allows the emission of high-energy level laser.Two of the most common misconceptions about lasers are that 1) all lasers are high powered, and 2) their beams are always parallel. Conversely, low-level lasers are most often designed with divergent beams as a safety precaution, and they operate at very low levels of power (0.05 to 0.5 W). LLLT emits no heat, sound, or vibration. Instead of producing a thermal effect, LLLT may act via nonthermal or photochemical reactions in the cells[, 28,29.]

Findings of the present study showed non-significant difference in the pre-treatment records of the MCV, between the mean values of the study and the control groups. Results of this study revealed a highly significant increase in the mean values of the MCV in the study group after the application of LILT, also comparing second records of the MCV, between the mean values of the study and the control groups showed highly significant increase indicating that LILT was fruitful and beneficial inincreasing the MCV of the neuropathic common peroneal nerve postburn.

Significant differences showed in this study were consistent with those observed and recorded by Feller et al., 2005; Gama, 2008; Karu, 2008; Smith, 2005; Baxter et al., 2007; Bolton et al., 2008;Zawacki et al., 2000; Delisa et al., 2008; Helm et al., 2002; Hoffmann et al., 2004 and Redford, 2004.

Results of this study supports the expectation that application of LILT was fruitful and beneficial inimproving the neuropathic common peroneal nerve postburn as evidenced by the highly significant increase in the MCV of the neuropathic common peroneal nerve postburn.

Conclusion

Application of LILT was fruitful and beneficial in improving the neuropathic common peroneal nerve postburn as evidenced by the highly significant increase in the MCV of the neuropathic common peroneal nerve postburn.

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