Laser Therapy in Relation to Bone Mineral Density in Postmenopausal Women with/without Osteoporosis

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ABSTRACT

The aim of this current study was to investigate the effect of low power laser irradiation (LPLI) on bone mineral density (BMD) of lumbar vertebrae in postmenopausal women with/without osteoporosis. Twenty postmenopausal women free from lumbar vertebrae osteoporosis served as group (I) and twelve postmenopausal women with lumbar osteoporosis served as group (II), participated in this study. Each group was subdivided equally into subgroups (a) and (b). Group (Ia) and (IIa) received Helium-Neon combined with infrared (He-Ne/IR) laser irradiation and group (Ib) and (IIb) received Helium-Neon (He-Ne) laser irradiation. Laser therapy was done three times/week for six weeks. CT densitometry was used for assessment of BMD before and after treatment. The results of the study demonstrated the superiority of a combination of He-Ne/IR laser to increase lumbar BMD than He-Ne laser in lumbar vertebrae in postmenopausal women with/without osteoporosis. Further research is required to examine long term effectiveness of this treatment and also when combined with physical activity that reported its effectiveness on bone repair in literature.

Key words: *Postmenopausal, Bone Mineral Density , Laser therapy,Osteoporosis.*

INTRODUCTION

Recently postmenopausal osteoporosis has become a burgoeing area of interest in terms of medical, social and economic costs[7]. It is a significant cause of women s morbidity and mortality leading to fractures of the hip, spine and wrist[19]. Osteoporosis is a primary metabolic disease of bone and a major public health problem that mostly occurs in the elderly.

It has been reported by Riggs and associates[23], that there is disproportionate loss of trabecular bone from the axial skeleton about 47% throughout life depending on the peak bone mass. It was reported in the literature that peak bone mass in the human skeleton is achieved in the third to fourth decade of life[20]. However, the adult skeleton is undergoing a continual process of remodeling in which bone resorptions is coupled with bone formation. At each remodeling site (approximately 0.1mm^3 of bone), a stereotyped sequence of events has been described. An initial stimulus activates the remodeling cycle. Osteoclasts bone resorbing cells that originate in the monocytemacrophage cell line, resorb an

apparently predetermined volume of bone. Having completed this task, the osteoclasts then disappear and are replaced by osteoblasts, which lay down osteoid refilling the cavity. Mineralization of osteoid completes the repair process[24]. In the aging skeleton, however, there is an imbalance between the resorptive and formative process[20,24].

There are two types of osteoporosis[28], type I due to a decrease in cumulating estrogens which affects trabecular bone (especially vertebral bone) and affects females more than males in a ratio of 1:6. Type II, senile osteoporosis, which is age related and occurs in cortical and trabecular bone, affects females and males in a ratio of 2:1.

It is evident that low bone mass is the most potent factor leading to fracture[24]. Estrogen deficiency is well established as a risk factor for osteoporosis[2]. There are several risk factor reported in the literature which accelerate the development of osteopenic process includes negative calcium balance, sedentary life style, immobilisation, menopause (surgical or natural), amenorrhea, family history of osteoporosis, high alcohol intake, smoking, high caffeine consumption and steroid therapy[5].

Although, there is no cure for osteoporosis, therapy should be directed primarly toward increasing physical activity, reducing the risk of falling and secondarily toward stabilizing bone mass. Halting or reversing the osteoporotic process require therapy in the form of hormonal replacement[24]. Calcitonin which is peptide hormone mediator for estrogen action, produce inhibition of osteoclasts activity and therefore decrease the bone resorption[11]. Also maintaining a high dietary intake of calcium, vitamin D, reduction of excessive consumption of protein and phorphorous are indicated as therapeutic options[28]. Calcium must be given with sodium fluoride to allow mineralization of the new osteoid. Problems with this modality, include the questions of abnormal bone architecture and the high incidence of side effects[24].

The impact of physical activity on BMD was established via reducing and/or preventing the volitional bone loss in both recently postmenopausal and very elderly women[22]. The role of electro-therapy in the management of menopausal osteoporosis is very limited in the literature. Zati et al.,[30] concluded that pulsed electromagnetic field has an effect to slow down the bone mass loss in osteoporosis induced by ovariectomy in rats and clinical application of the same current in women s osteoporosis was also reported[25].

Although high-power laser therapy for surgery and hemostasis is well known, the effect of low-power laser irradiation (LPLI) which usually means less than 60-100 mW power intensity and is regarded as showing a non-thermal effect, still remains surrounded by skepticism in spite of more than 20 years of clinical use and investigation. Observed and reported effects cover alteration of nerve function, acceleration of wound and fracture healing and treatment for pain control[1,18,27]. Also, widespread effects were revealed on cellular functions in vitro experiments[16,29], even in animal experiments, there are several reports on the action of lasers to enhance osteogenesis[3,27].

A critical review of the literature has revealed a gap in the effect of LPLI in postmenopausal osteoporosis. From this view, this study was conducted to investigate the effect of LPLI on BMD of lumbar vertebrae in postmenopausal women with / without osteoporosis.

SUBJECTS, MATERIALS & METHODS

Subjects:

Thirty two consecutive postmenopausal women were recruited from Kaser El-Aini and Mgd El-Eslam Hospital between 1996-1998. The criteria for inclusion were as follows: (a) CT densitometry diagnosis of normal BMD and osteoporosis in lumbar vertebrae with no evidence of vertebral compression fractures, (b) age between 51 to 60 years (to avoid inclusion of older patients with multiple medical problems), (c) no history of cancer, renal disease, gastrectomy, metabolic bone disease or any condition (such as a neurogenic, myopathic or connective tissue disorder) that could cause secondary osteoporosis, (d) no intake of any medications associated with accelerated bone loss (steroids) or any medications affected bone metabolism (estrogen, calcium, vitamin D, ...etc), (e) body mass index not exceeding 30 Kg/m^2, non smoker, parity from 1-3 times and led sedentary life style without participation at any exercise training during this study, and, (f) had natural menopause at least 1 year before entry into the study with no history of ovariectomy. Subjects were divided into two groups: group (I) consists of 20 subjects with normal BMD in lumbar vertebrae, while group (II) consists of 12 subjects with BMD in lumbar vertebrae below normal level for each subject (osteoporosis). Then each group was further subdivided into two equally groups (a) and (b).

Instrumentation:

(1) Somatom HiQ-S (siemens), for the qualitative assessment of BMD in the vertebral bodies of the lumbar spine for both groups.

(2) Levelaser M300: was used to deliver laser therapy. The apparatus provided the following options:

* He-Ne laser 632.8 nm, minimum power 12 mW.
*He-Ne and IR1 laser 904 nm, minimum power 22/35 mW.
*He-Ne and IR2 laser 780-870 nm, minimum power 1 W.
*He-Ne, IR1 and IR2 laser.

Procedures:

A. Evaluation

Initially a screening test including careful history taking and gynecological examination were conducted for each subject before entry in this study. After that BMD of lumbar spine (L_{1-5}) was measured by osteo CT densitometry. Evaluation of lumbar BMD was performed before and after the end of six weeks of treatment.

B. Treatment

All subjects in this study underwent three sessions per week for six successive weeks period of treatment. The treatment procedure was explained to all subjects. Skin was cleaned with alcohol to remove fat. During the irradiation, the position of the subjects was the same for both groups (prone lying position with a pillow under her abdomen). LPLI was irradiated to the lumbar vertebrae (L_{1-5}) using the following low power laser treatment parameters. Group (Ia) and (IIa) received He-Ne/IR laser 904 nm, 22-35 mW, while group (Ib) and (IIb) received He-Ne laser 632.8 nm, 12 mW. The delivery technique for both group was automatic scanning with energy density of 4 J/cm^2.

C. Statistical analysis

Data were collected and statistically analyzed using the arithmetic mean, standard deviation and paired t test at level of significance of 0.05.

RESULTS

In the present study, the response of BMD to LPLI was investigated. The data collected from both groups after six weeks of laser irradiation were compared with the pre treatment.

As revealed from table (1) and figure (1) there was a highly statistically significant increase ($P<0.001$) in the mean value of lumbar BMD between pre and post treatment in group (Ia) which represent 10.32% of the pre treatment value, while other groups (Ib) and (IIa) showed significant increase ($P<0.04$) in the mean values of lumbar BMD between pre and post treatment which represent 5% and 6.1% respectively. While in group IIb the difference was non significant ($P<0.07$) in spite of showing a percentage of change equal to 4.1%.

Table (1): represents the mean values of lumbar BMD for all groups.

Groups	Subgroups	BMD of L_{1-5} (mg/cm^2/years)		Level of significance	% of change mean
Group (I)	Ia	pre	128.47±858	0.001	10.32%
		post	141.73±6.09		
	Ib	pre	126.23±6.83	0.04	5%
		post	132.60±5.69		
Group (II)	IIa	pre	86.74±6.31	0.04	6.1%
		post	92.05±3.99		
	IIb	pre	86.48±3.46	0.07	4.1%
		post	90.03±4.25		

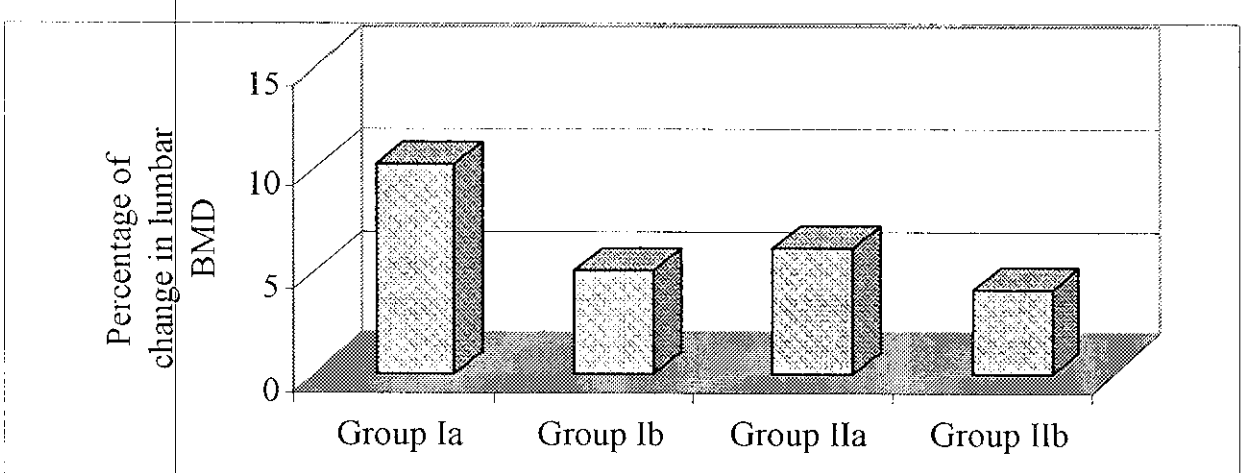

Fig. (1): Percentage of change in lumbar BMD from pre to post treatment for all groups.

Also, as observed from table (2), there was a statistically highly significant difference ($P<0.0001$) at pre and post treatment values between either group (Ia) and (IIa) or group (Ib) and (IIb).

Table (2): Comparison of lumbar BMD between groups at pre and post treatment.

Date of assessment	Groups	t value	Level of significance
pre treatment	Group Ia Group IIa	2.81	0.0001
	Group Ib Group IIb	4.39	0.0001
Post Treatment	Group Ia Group IIa	6.7	0.00001
	Group Ib Group IIb	4.96	0.00001

DISCUSSION

All through the history of humanity, attempts to combat pain have not stopped and will never stop, as long as, there is life on earth. The primary problem in postmenopausal with / without osteoporosis is thought to be enhancement of bone resorption, with consequent net loss of bone mass as osteoblasts fail to repair the defect completely[23] which increase the risk to fracture. The basic problem in the remodeling of bone is directly related to the stimulation, multiplication and proliferation of the extraperiosteal, periosteal and medullar connective tissue that forms reparative blastemas leading to the consolidation of the bone[9].

In our field, this is the first study to report the effect of LPLI on BMD of lumbar vertebrae in postmenopausul women with / without osteoporosis.

Osteo CT densitometry was used for assessment of bone density since a critical review of the literature has revealed a gap in this area of information.

The results of CT densitometry revealed a highly significant increase in lumbar BMD of postmenopausal women and a significant increase in lumbar BMD of postmenopausal osteoporotic women with He-Ne/IR laser. While He-Ne laser effects on lumbar BMD in postmenopausal women was found to be increased significantly compared to non significant increase in postmenopausal osteoporotic women.

The process of bone healing or remodeling is very similar to that of soft tissues healing. It would therefore seem reasonable to suggest that bone healing and/or remodeling might be stimulated by laser which is reported in our results. LPLI has been found to modulate various processes in various biological system[13] according to the photochemical theory which stated that absorption of laser not a sentence light take place in tissue chromophores (photoreceptors). These chromophores may be enzymes, a membrane molecule, or any other cellular or extra cellular substances[13]. Light energy was converted to metabolic energy involving the respiratory chain via production of a transmembrane electrochemical proton gradient[12]. This energy activates metabolic process such as an increase of calcium release from mitochondria and ATP production which enhance and moderate cell activity[13,15]. The magnitude of this effects seems to depend on wavelength[18]. Cell in cultures communicate with each other by means of electromagnetics energy, which influences metabolic and catabolic cell processes. In case of an impairment or disorder like our design, the energy state of a cell is changed so altering the accompaining communication, laser therapy is thought to influence this communication[12].

Since the precise mode of action of LPLI is not clearly understood, it is difficult to

interpret its dramatic effect on the process of bone regeneration. The particular properties of laser light would create a series of environmental conditions that accelerate the remodeling of bone[27]. It was reported that osteogenesis has been considered as depending directly on local circulation[26]. Bone develops better in a well vascularized environment therefore it would be desirable to produce this situation through therapeutic means in order to achieve faster bone consolidation.

The results registered in this study coincide with many research studies investigating the effect of LPLI on tissue and bone repair in vivo and vitro models and were in agreement and supporting our finding.

Kokino et al.,[14] investigated the callus formation in both fibulae of albino rats macroscopically and microscopically and found that laser had stimulating effects on callus formation. Trelles and Mayayo[27] applied He-Ne laser in doses of 24 J at one point to experimental tibial fracture in mice. The treated group displayed increased vascularisation, fastened formation of osseous tissues when compared with control group suggesting that laser might affect the function of osteocytes and promoting faster metabolism and reduction of bone callus which explained our findings in this study. They concluded also that, remodeling of the bone affects the totality of bone tissue with increase in trabeculae and modification of their disposition and denisty showed an active participation of the osteoblasts due to laser action which promote more osteosynthesis produced by increase vascularisation and anti-inflammatory action of it. Trelles 1982 according to Trelles and Mayayo[27] reported the improvement of osteogenesis in localized osteoporosis when radiating the periosteum with He-Ne laser which agrees with our results. Chen and Zhou[4] demonstrated that calcium, phosphorous and hydroxyproline quantities were greater in irradiated mandibular osteotomy sites, suggesting that laser could speed the process of bone healing.

Lubart et al.,[16] concluded that laser biostimulation probably starts the cascade of metabolic events by being absorbed by endogenous photosensitizers in the cell generating small amount of reactive oxygen species which activate the cell. Laser can accelerate tissue repair by activation of mast cell leading to degranulation and release of mediators[8]. Activation of nuclear transcription factor in human keratocytes was also reported[17]. It was stated in the literature that LPLI have a positive effect on proliferation, differentiation and calcification of clonal osteoblastic cells[29].

Barushka et al.,[3] found that He-Ne laser irradiation on hole injuries in the tibia of the rat affected the population of osteoblasts and osteoclasts by alterations in alkaline phosphate and tartrate resistance acid phosphatase activities. They also found that LPLI caused an approximately two fold enhancement in bone repair in the hole injuries of the rat tibia as revealed by histophotometry. Glinkowsky and Rowinski[10] used low-level diode laser therapy on tibial fractures in mice, and evaluation of the bone radiographs by a laser densitometer, demonstrated higher optical denisty in the irradiated group compared to controls.

Luger et al.,[17] used a biomechanical methods to investigate the effects of LPLI on bone fracture healing in rats. They concluded that gross non union of fracture after four weeks post trauma was found in four of 19 rats (21%) in the control group but non of the LPLI group. According to Trelles and Mayayo[27], the fracture region after LPLI showed predominance of fibroconnective tissue

with aboundant active osteoblast and osteocites trapped in their osteoid of the fracture region whereas in the bone of the control animals there was only cartilagenous consolidation which would be due to the poor vascularisation since cartilage predominates in the avascular regions.

It was reported in the literature that the use of prostaglandin E_2 activates the healing process of fracture. Mester et al[18], observed increase of prostaglandin after laser irradiation which in turn would contribute to earlier onset of osteosynthesis. It was reported in the literature that laser irradiation accelerated proliferation of the cells only in the growing stage. The cellular photosensitivity generally depends on the physiological state of the cell during irradiation[29]. Cells in the growing phase are more photosensitive than those in the stationary phase i.e. when the cells considered to be undifferentiated osteoprogenitor cells. Also, Ohshiro[21] noted the importance of Na-K-ATPase in regulating Na-K pump activity and modulating cell membrane gradiants and cited it as a possible mechanism of laser irradiation. Stimulation of collagen synthesis in human skin fibroblast culture was also reported[15]. In addition, LPLI can stimulates mitotic activity, increase metabolism and favorably influence immune process in the tissues[8].

David et al.,[6] failed to support the previously reported enhancing effect of He-Ne laser irradiation on fracture healing in rats with the same energy level. These results are in contradiction to those of Trelles and Mayayo[27] who reported faster formation of osseous tissue with dense trabecular net in the irradiated fractures which used histology as the only parameter for fracture healing. Most investigators used He-Ne laser or IR laser for their experiment and reported their positive effects on enhancing bone repair and other effects previously reported. In this study combination of He-Ne and IR laser demonstrated a higher significant increase on BMD than therapy with He-Ne laser alone.

CONCLUSION

Although osteoporosis is a primary metabolic disease of bone and a major public health problem that mostly occurs in postmenopausal period, there is no cure of it. Therapy should be directed primarily toward increasing physical activity, reducing the risk of falling and secondarily toward stabilizing bone mass. The results of this study demonstrated the superiority of combination of He-Ne and IR laser to increase BMD of lumbar vertebrae in postmenopausal women with/without osteoporosis.

Although the findings of this study are highly significant, but treatment of low bone mass might not be effective enough to guarantee that any gains in mass will be of sufficient magnitude to reduce fracture risk significantly, so further research is required to examine long term effectiveness of this treatment and combine it with physical activity that reported its effectiveness on bone repair in literatures.

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الملخص العربي

تأثير العلاج بالليزر على كثافة العظام

لدى السيدات بعد انقطاع الطمث مع وجود/عدم وجود هشاشة في العظام

الهدف من هذا البحث دراسة تاثير العلاج بأشعة الليزر على كثافة العظام لدى السيدات بعد انقطاع الطمث مع وجود/عدم وجود هشاشة في عظم المنطقة القطنية. اشترك في الدراسةاثنين و ثلاثون مريضة تم تقسيمهم إلى مجموعتين. المجموعة الأولى تتكون من عشرون سيدة ليس لديها هشاشة في العظام والمجموعة الثانية اثنتى عشرة سيدة لديها هشاشة في العظام. ثم قسمت المجموعتين مرة أخرىبالتساوى كلا إلى (اب) وتم علاج المجموعة الأولى والثانية (ا) بالهليوم نيون مع الانفراريد ليزر أما المجموعة الأولى والثانية (ب) تم علاجها الهليوم نيون فقط.كانت مدة العلاج ثلاث مرات في الأسبوع لمدة ستة أسابيع. تم تقييم الحالات قبل وبعد العلاج عن طريق الأشعة المقطعية (دنسيوتوميترى). وقد أكدت نتائج هذه الدراسة كفاءة العلاج بالهليوم نيون مع الانفراريد ليزر على زيادة كثافة العظام في المنطقة القطنية لدى السيدات بعد انقطاع الطمث في وجود/عدم هشاشة في العظام.