



Pressure Ulcer Surface Area and Volume Response to Gallium- Arsenide Laser

Authors

**Ahmed Mamdouh Mohamed Abd Al-Kader, Maha A.Hassan*, Hisham Galal Mahran
Elsayed*, * Zakaria Mowafy Emam Mowafy**

National Cancer Institute – Cairo University

*Physical Therapy Department For Surgery, Faculty Of Physical Therapy, Cairo University, Egypt

Abstract

This study has been conducted to evaluate the efficacy of the Gallium Aluminum Arsenide (Ga-As) laser in accelerating pressure ulcers healing. Methods of evaluation (wound surface area and wound volume).

Methods:- *Thirty patients (16 males and 14 females) with complete or incomplete spinal cord injury patients and complain from pressure ulcers were randomly divided into two groups. Group (A) received Ga-As laser plus the regular wound care, duration of treatment was 10 minutes daily for two months. Group (B) (Control group) received only the regular wound care.*

Results:- *Result showed that the Ga-As laser was effective in decreasing ulcer surface area and ulcer volume as well as improving healing of pressure ulcers.*

Conclusion: *- Ga-As laser was effective in accelerating pressure ulcer healing.*

Keywords *(Laser, Pressure ulcers, wound surface area and wound volume).*

Introduction

Many factors can impair healing. Local factors include the presence of foreign bodies, tissue maceration, ischemia and infection. Systemic factors as diverse as advanced age, malnutrition, diabetes and renal disease may be important. In addition to local and systemic factors that impair healing, the wound becomes chronic because of reduction in tissue growth factors or an imbalance between proteolytic enzymes and their inhibitors. Chronic wounds are thus defined as wounds, which have “failed to proceed through an orderly and timely process to produce anatomic and functional integrity, or proceeded through the repair process without establishing a sustained anatomic and functional result,^{3, 4, 5.}

Reduced levels of active growth factors in the wound environment may partly explain why certain wounds fail to heal. Chronic ulcers are known to have reduced levels of platelet derived growth factor, basic fibroblast growth factor, epidermal growth factor and transforming growth factor β compared with acute

wounds. It has been suggested that growth factors may become trapped by extracellular matrix molecules or may be degraded by proteases to an excessive degree, resulting in non-healing. Imbalance between proteinases and their inhibitors—excessive proteinase activity in chronic wounds probably from over expression of matrix metalloproteins, results in abnormal degradation of the extracellular matrix. Many new treatment strategies are directed at modifying the imbalance—by the topical application of proteinase inhibitors, by inducing the expression of endogenous inhibitors or by combining proteinase inhibitors with growth factors,^{7, 8,9.}

Pressure ulcer constitutes one of the major problems confronting the Healthcare professionals who are called upon to supervise the care of the severely disabled or debilitated patient. Every care provider is well aware of the complications manifested by the occurrence of ulceration in the chair or bed-ridden patient. Pressure ulcer prolongs patient morbidity and interferes with rehabilitation and medical maintenance. It may also frequently be implicated as a major contributing factor leading to the patient demise. Ulceration of the skin, especially over bony prominences, has undoubtedly plagued the disabled and debilitated patient since the beginning. Before the advent of antibiotic therapy, secondary infection of the ulcerations led to an early death, whereas today the patients usually survive for prolonged period^{11,13, 15.}

Pressure ulcers are localized areas of cellular necrosis, which usually occur over bony prominences, which are subjected for prolonged periods of time to pressure in excess of capillary pressure. Since normal cellular metabolism is dependent on the reception of nutrients and the elimination of metabolism, any condition which interferes with this exchange will affect the function of the cell. The circulating peripheral blood fulfills the metabolic needs of the cells so that alteration of the circulation in cellular changes. Prolonged circulatory interference ultimately leads to death of the cell. Pressure ulcers may be caused by inadequate blood supply and resulting reperfusion injury when blood re-enters tissue. A simple example of a mild pressure sore may be experienced by healthy individuals while sitting in the same position for extended periods of time: the dull ache experienced is indicative of impeded blood flow to affected areas. Within hours, this shortage of blood supply, called ischemia, may lead to tissue damage and cell death. The sore will initially start as a red, painful area, which eventually turns purple. Left untreated, the skin may break open and become infected. Moist skin is more sensitive to tissue ischemia and necrosis and is also more likely to get infected,^{15, 17, 19.}

Shearing force is an important factor in the production of ischemic ulcers, shearing and stretching of blood vessels tended to compound the ischemic changes produced by external pressure, thereby increasing the rate of tissue breakdown. Working with swine, demonstrated significant skin breakdown when the tissue was subjected to both pressure and friction, at a pressure significantly less than that when necrosis was caused by pressure alone (45mmHg versus 290mmHg),^{26,29.}

The salutary effect of laser therapy in medical practice connects with the improvement of microcirculation and the activation of cell proliferation. The concepts of free radical mechanism of low level laser irradiation (LLLI) stimulating action to the endogenous porphyrins, which are chromophores of LLLI in the red

spectral range and known as photo sensitizers, localized in blood cells membrane and absorb photons of the LLLI. This process is the basis for initiation of photosensitized free radical reaction including lipid peroxidation of blood leukocyte membranes with subsequent formation of lipid hydroperoxides. Peroxidative modification of membrane lipids increases cell membrane ionic permeability for calcium ions, 1,2,6,21,22,23,24,25.

Normal wound healing requires both destructive and reparative processes in controlled balance. Proteases and growth factors play an important role in regulating this balance, and if disrupted in favour of degradation then delayed healing ensues, which is a trait of chronic wounds. It has been shown that low-level laser irradiation at certain fluencies and wavelengths can enhance the release of growth factors and stimulate cell proliferation. The effects of LLLT include wound epithelialization, reduction of oedema and inflammation, and re-establishment of arterial, venous and lymph microcirculation. Increased rates of ATP, RNA and DNA synthesis are also observed. Major changes seen in wounds treated with LLLT include increased granulation tissue, early epithelialization, increased fibroblast proliferation, increased extracellular matrix synthesis and enhanced neovascularisation, all of which lead to better tissue oxygenation and nutrition, and, in turn, enhanced wound healing. It has been shown that low-level irradiation of fibroblasts stimulates the production of basic fibroblast growth factor and stimulates the transformation of fibroblasts into myofibroblasts. LLLT also affects immune cells, and acts directly and selectively on the immune system. Stimulation of the immune system means that infected wounds can be cleared more readily. The use of low-level lasers in wound healing has been shown to speed up the healing of leg ulcers and burn wounds; it has also been shown to improve skin-healing capabilities^{10,12, 14,16,20,27,30.}

Material and Methods:

Subjects

Thirty volunteer patients (16 males and 14 females) with their ages were ranged from 30 to 50 years. They were diagnosed as complete or incomplete spinal cord injury by a neurologist in El-Kasr El-Aini, Cairo University Hospitals. They had sacral pressure ulcers classified between grade 2 and grade 3 according to the European Pressure Ulcer Advisory Panel. Subjects were randomly divided into two equal groups in number: **Group (1):** (the study group) (**Ga-As laser group**). This received the Ga-As laser and the regular wound care. **Group (2):** (the control group): This received only the regular wound care.

Instrumentation and tools:

In this study the measuring equipment were the ulcer surface area (USA) measurement in cm² and the ulcer volume measurement (UVM) in CC, while the therapeutic equipment was the Ga-As laser. The laser device used in this study is manufactured by: LTU-904n m (**uniphy, phyaction 796 S/N 20381, MADE IN HOLLAND**). Also protective eye glasses wear during application of laser to avoid permanent eye damage resulting from direct exposure to the beam,^{1,2,6,10,12.}

Procedures:**Evaluation:****1 Measurement of ulcer surface area (USA):****in cm²**

The measurement of wound surface area conducted by tracing method by a sterilized transparency film placed on the wound or ulcer area. The ulcer parameter was traced by using the fine tipped-transparency marker. Each wound area traced three times to establish measurement reliability. After tracing, the transparency film face, which faces the ulcer wound, was cleaned by a piece of cotton and alcohol. The carbon paper placed over the metric graph paper 1mm². The traced transparency film was placed over a carbon paper with a white paper in between and transcribes the tracing on metric graph paper. The number of square millimeters on the metric graph paper within ulcer wound traced counted to determine the ulcer wound area. This area was converted to cm²; the mean of the three trails calculated and considered as a wound surface area (USA). The measurements of ulcer surface area will be conducted pre-treatment as a first record and after 30 days (one month) as a second final record, ^{13,17}.

2- Ulcer volume measurement (UVM): in CC.

The measurements of volume of the ulcer was conducted by isotonic solution by the following steps: The sore prepared and the surrounding skin was cleaned and dried. A transparent adhesive film was applied tightly over the sore and surrounding skin. The film was extended sufficiently beyond the same margin to ensure good adhesion. The ulcer then was filled with sterile physiological saline by injection through the film. Another needle was placed at the highest point of the ulcer to allow air to escape. The volume of solution required to fill the ulcer was recorded to indicate the volume of the ulcer. The measurements of ulcer surface area were conducted pre-treatment as a first record and after 30 days (one month) as a second final record, ^{19,26,29}.

Steps of the Ga-As laser treatment procedures:

Position of the patient: Prone lying position is appropriate for sacral pressure ulcers, the wound cleaned at first. Some abscesses were opened and pockets of pus drained, and necrotic tissue was removed with scrubbing the wound with a soft tooth brush followed by hydrogen peroxide, saline rinse and betadine. Laser device preparation: the plug of laser unit was inserted into the main current supply, the on/off switch switched on; now therapist must wear the protective eye glasses. Then set the treatment parameters of laser. Ga-As Laser application: Non-contact technique of laser application used with the laser probe perpendicular 2-millimeters away from the ulcer surface, treating the ulcer according to the grid technique 90 seconds for every centimeter square of the ulcer surface with 10 minutes as the total time of the session. It is advisable to prolong the Ga-As Laser application for one or two weeks if wound closure occurred before the end of the treatment month in order to strengthen the treated area. Frequency of application: daily for two months, ^{1,2,6,10,12,14}.

Data analysis:

ulcer surface area (USA) in cm^2 and ulcer volume measurement (UVM) in CC were measured pre-treatment as a first record and after 2 months as a second final record in both groups. 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,^{18,28}.

Results:

As shown in table (1) and figure (1), the mean value of the USA before treatment was $(16.45 \pm 6.22) \text{ cm}^2$ in the study group, while after treatment was $(7.22 \pm 2.33) \text{ cm}^2$. These results revealed a highly significant reduction in USA ($P < 0.0001$). While in the control group, the mean value of the USA before treatment was $(16.43 \pm 6.19) \text{ cm}^2$, while after treatment was $(16.40 \pm 6.16) \text{ cm}^2$. These results revealed non-significant difference in the USA ($P > 0.05$).

Table (1): Comparison of the mean values of ulcer surface area (USA) measurement in cm^2 before and after treatment in both groups

	Before treatment		After treatment		Mean difference	T-value	P.value	Level of significance
	Mean	SD	Mean	SD				
Study group	16.45	6.22	7.22	2.33	9.23000	5.38	0.0001	Highly significant
Control group	16.43	6.19	16.40	6.16	0.030000	0.01	0.989	Non-significant

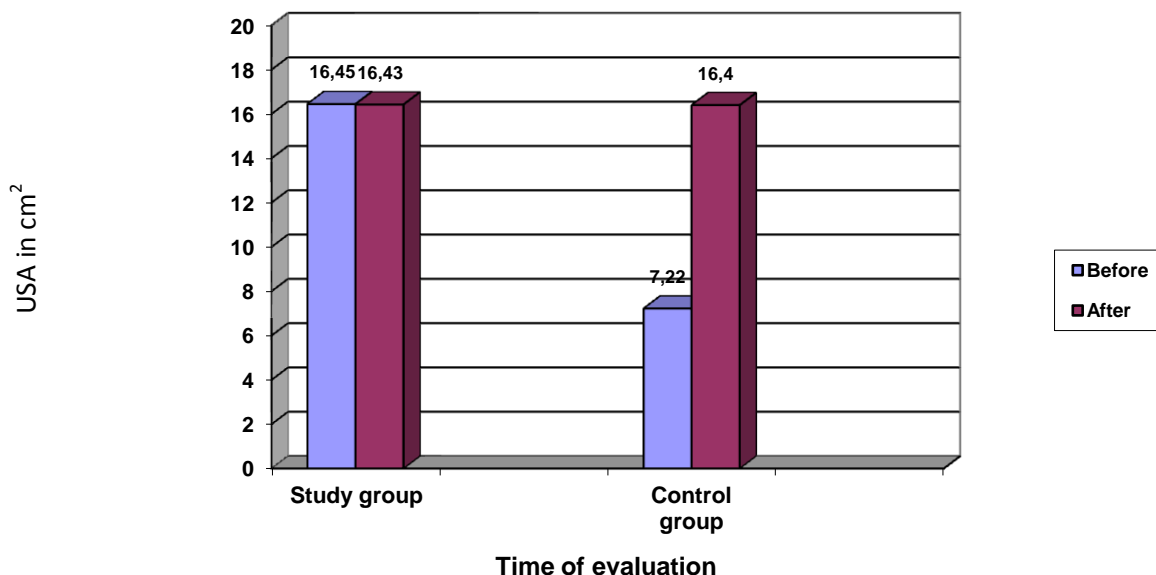


Fig (1): Mean values of the USA before and after treatment in both groups.

Also, as shown in table (2) and figure (2), the mean value of the ulcer volume measurement (UVM) in CC before treatment was (21.99 ± 6.44) CC in the study group, while after treatment was (8.56 ± 2.32) CC. These results revealed a highly significant reduction in UVM ($P < 0.0001$), while in the control group, the mean value of the UVM before treatment was (21.93 ± 6.41) CC, while after treatment was (21.90 ± 6.38) CC, these results revealed non-significant difference in the UVM ($P < 0.05$).

Table (2): Comparison of the mean values of UVM before and after treatment in both groups

	Before treatment		After treatment		Mean difference	T-value	P.value	Level of significance
	Mean	SD	Mean	SD				
Study group	21.99	6.44	8.56	2.32	13.4300	7.60	0.0001	Highly significant
Control group	21.93	6.41	21.90	6.38	0.030000	0.01	0.990	Non-significant

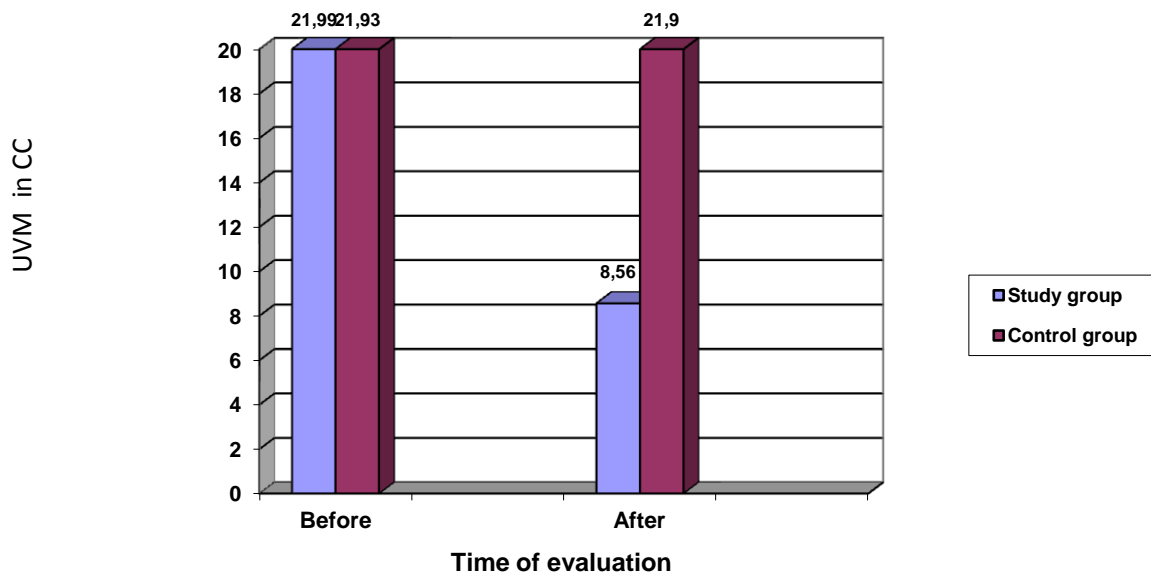


Fig (2): Mean values of the UVM before and after treatment in both groups.

Discussion

Successful wound healing requires more than closing the wound with sufficient tensile strength. Remodeling requires the scar to change to fit the tissue. Remodeling of the scar maturation represent the final aggregation, orientation and arrangement of the collagen fibers. Two processes arc involved under the umbrella of the remodeling phase; Synthesis-lysis balance and the collagen fiber orientation. Synthesis-lysis balance means the balance between the new collagen production process and the break down process of old collagen. If collagen formation is higher than old collagen breakdown this will lead to hypertrophic scar and Keloid formation. Collagen destruction or breakdown is occurred by the action of the collagenase enzyme. Collagenase enzyme is capable of cleaving strong cross-links of the tropo-collagen molecule to become soluble and excreted from the body as waste products^{3, 4,5}.

Elderly patients and individuals with spinal cord injuries, traumatic brain injury, and neuromuscular disorders are high risk populations for pressure sore development, especially in the acute hospital seeing. Immobility, malnutrition, fecal and urinary incontinence, and level of consciousness are important associated risk factors. Chronic systemic illness and the presence of a fracture may also be predictive of ulcer development. All of these factors are more prevalent among the elderly, compounding the risk age it confers. A number of changes occur in normal skin aging which predisposes older individuals to the development of pressure sores. Epidermal turnover decreases the dermal-epidermal junction flattens, and there are fewer dermal blood vessels. In addition, there is an increase in dermal collagen, a decrease in elastic fibers, and a loss of cells that synthesize vessels of basement membranes and an increase in skin permeability. Older individuals may also

experience in the perception of pain. There are currently two major theories about the development of pressure ulcers. The first and most accepted is the deep tissue injury theory which claims that the ulcers begin at the deepest level, around the bone, and move outward until they reach the epidermis. The second, less popular theory is the top-to-bottom model which says that skin first begins to deteriorate at the surface and then proceeds inward^{7, 8, 9.}

Pressure sores can trigger other ailments, and cause patients considerable suffering and financial cost. Some complications include autonomic dysreflexia, bladder distension, osteomyelitis, pyarthroses, sepsis, amyloidosis, anemia, urethral fistula, gangrene and very rarely malignant transformation. Sores often recur because patients do not follow recommended treatment or develop hematomas, infections, or dehiscence. Paralytic patients are the most likely people to have pressure sores recur. In some cases, complications from pressure sores can be life-threatening. The most common causes of fatality are from renal failure and amyloidosis^{11, 13, 15.}

The use of light for therapeutic purposes dates back to the ancient Egyptians, Greeks and Romans. Current research into the physiological benefits of light therapy has developed an area of great interest which is the laser. Most research in the uses of laser was reported by European sources. Only during the past decade have American research workers begun to add the results of their studies. The notion that light, in the visible and near infrared ranges, can produce photo chemical and photo biological changes that ameliorate pain and inflammation as well as promote tissue repair was first observed in the late 1960s. At this time the prevailing notion was that lasers were uniquely photo destructive, promoting attempts to develop powerful lasers that may yield military superiority. Thus, the mood was not right and neither were medical minds ready to accept the idea that a tool that can cut, vaporize, and otherwise destroy tissue could be used for beneficial purposes^{1, 2,6,12, 14.}

Laser is an acronym for Light Amplification by Stimulated Emission of Radiation; it is a form of phototherapy which involves the application of monochromatic light over biological tissue to elicit a biomodulative effect within that tissue. The biomedical laser, light amplified by stimulated emission of radiation, may be classified as either high and low powered or hot and cold respectively, high power lasers are used to cut through tissue. Low-level lasers, on the other hand, are used to stimulate tissue repair through a process of bio-stimulation,^{16, 20,22,23.}

The findings of the present study showed non-significant differences in the pre-treatment records of the USA between the mean values of the study and the control groups. As well as in the pre –treatment records of the UVM, between the mean values of both groups.

Results of the study group revealed a highly significant decrease in the mean values of USA and UVM, after application of the Ga-As Laser , when compared against the pre-application results.

Also results of the study group revealed a highly significant decrease in the mean values of USA and UVM, after application of the Ga-As Laser, when compared with the control group results after application of the regular ulcer care.

Significant differences showed in the study and control groups were consistent with those observed and recorded by Amir et al., 2000; Antonio et al., 2007; Brosseau and Morin , 2008; Cervo and Cruz, 2000; Damante et al., 2004; Demir et al., 2004; Franek et al., 2002; Lagan et al., 2002; Lagan et al., 2000, Lucas et al., 2003; Luther, 2004; Pereira et al., 2002 and Simon, 2004.

Results of this study support the expectation that application of the Ga-As Laser had valuable effects in enhancing healing of pressure ulcers in patients with complete or incomplete spinal cord injury, as manifested by the highly decreases USA and UVM.

Conclusion

Ga-As Laser was effective in enhancing healing the pressure ulcers in patients with complete or incomplete spinal cord injury as manifested by the highly decreases in ulcer surface area and ulcer volume.

References

1. Amir AS, Solomon AS and Cordoba MB, (2000): "The influence of helium-neon laser on the viability of skin flaps in the rat." *Br.J.Plas.Surg*; 53:pp.66-72.
2. Antonio LB, Pinheiro KH and Rodrigues FM, (2007): "Effects of Laser Therapy on Experimental Wound Healing Using Oxidized Regenerated Cellulose Hemostat" *Photo-medicine and Laser Surgery* 26(1): 10-13. doi:10.1089/pho.2115.
3. Aplaslan GG and Takano YE, (2007):" Wound Healing treatment: Wounds, 11 (9): Pp 202-15.
4. Berg SS, (2008): "Pressure sore treatment". *Lancet*; 444-51.
5. Bohannon AW, (2009): "Wound surface area and wound perimeters" *Lancet*; 212-16.
6. Brosseau LF and Morin MP, (2008):"Low level laser therapy and polarized light for treating rheumatoid arthritis". *Cochrane Database of Systematic Reviews*, Issue 3. Art. No.: CD002049. DOI: 10.1002/14651858.CD002049.pub2
7. Bucalo BW, Eaglstein WH and Falanga VS, (2003): "Inhibition of Cell Proliferation by Chronic Wound Fluid". *Wound Repair Regen.* 1: Pp 181-186
8. Cervo FA and Cruz AC, (2000): "Pressure ulcers analysis of guidelines for treatment and management". *Geriatrics*, 55: 55-60.

9. Cuddigan JA, (2001): "Pressure ulcers in America: Prevalence, incidence, and implications for the future," in An Executive Summary of the National Pressure Ulcer Advisory Panel Monograph, Jul/August.
10. Damante CA, Greggi SL and Passanezi EF, (2004): " Clinical evaluation of the effects of low-intensity laser (Ga-As) on wound healing after gingivoplasty in humans". J. Appl. Oral Sci; 12 (2): PP.1-6.
11. David JH, Chapman JT and Chapman EA, (2003): "An investigation of the current methods used in the nursing for the care of patients with established pressure sores". Nurs. Res; 23:110-113.
12. Demir HJ, Balag HB and Kirnap MK, (2004): "A comparative study of the effects of electrical stimulation and laser treatment on Experimental wound healing in Rats": J.R.R.D. 41 (2): pp 147-154.
13. Dutcher JA, Ratliff CR and Bryant DE, (2002): "Guideline for prevention and management of pressure ulcers". Wound Ostomy and Continence Nurses Society (WOCN) Clinical Practice Guideline Series.
14. Franek AS, Krol PA and Kucharzewski MF, (2002): "Does low output laser stimulation enhances the healing of crural ulceration?" Some critical remarks. Med Eng Phys 2002; 24(9):607-15.
15. Gold-Stin BG, Sanders JE and Berson BK, (2006): "Pressure ulcer in SCI: Dose tension stimulates wound healing. Am. J. Phys. Med. Rehabil; (2) Pp: 130-133.
16. Greco MK, Vacca RA and Moro LT, (2001): "Laser therapy of hepatocytes can trigger increase of the mitochondrial membrane potential and can stimulate c-fos expression in a Ca^{++} dependant manner." Laser Surg. Med; 29; pp.433-441.
17. Harding GF, Morris HL, and Patel GK,(2002): " Healing Chronic Wounds." Br. Med. J, 324: Pp 160-163.
18. Hinton PR, (2004): "Statistics Explained"2nd Ed. Routledge Taylor &Francis Group London Pp149-155.
19. Irion LI, (2002): "Normal Wound Healing" in "Comprehensive Wound Healing and Wound Management" 1st ed., Slack Incorporated Thorofare USA. ch(2): Pp 13-18.
20. Lagan KM, McDonough SM and Baxter GD, (2002): "A case report of low intensity laser therapy in the management of venous ulceration". Lasers Surg Med; 18 (1): pp.15-22.
21. Lagan KM, McKenna TA and Witherow AD, (2000): "Low-intensity laser therapy/combined phototherapy in the management of chronic venous ulceration: a placebo-controlled study". J Clin Laser Med Surg; 20(3):109-16.
22. Lee SA, Talavera FD and Meyers AD, (2001):"Lasers, general principles and physics". Medicine. Com. Inc; pp.1-7.

23. Lopes LA, Zangaro RA and Jaeger MM, (2001): "Comparison of the low level laser therapy effects on cultured human gingival fibroblasts proliferation using different irradiance and same flounce". Lasers Surg. Med; 29: pp. 179-184.
24. Lucas CA, Gemert MJ and Haan RJ, (2003): "Efficacy of low-level laser therapy in the management of stage III decubitus ulcers: a prospective, observer-blinded multi-centre randomized clinical trial". Lasers Med Sci 2003; 18(2):72-7.
25. Luther VD, (2004): "Low-Level Laser Therapy Facilitates Superficial Wound Healing in Humans". A Triple-Blind, Sham-Controlled Study by the National Athletic Trainers' Association, Inc, 39(3): 223–229
26. Niezgodna JA and Susan ME, (2006): "The Effective Management of Pressure Ulcers. Advances in Skin & Wound Care." The Journal for Prevention and Healing, Volume 19, Number 1 - Supplement: 3-15.
27. Pereira AN, Matson ED and Marques MM, (2002): "Effect of low power laser on cell growth and procollagen synthesis of cultured fibroblasts". Lasers Surg Med; 31: pp. 263-267.
28. Pipkin FB, (1984): "Medical statistics made Easy" Edinburgh. London. Mel Bourne and New York.
29. Rutledge JB, (2004): "Obstacles to Wound Healing". Cosmetic Surgery Times, 67: Pp 356-361.
30. Simon AA, (2004): "Low level laser therapy for wound healing". Health Technology Assessment Unit. Alberta Heritage Foundation For Medical Research.

استجابة مساحة وحجم قرح الفراش للجاليوم- ارسينايد ليزر

أحمد ممدوح محمد عبدالقادر

المعهد القومي للاورام – جامعة القاهرة

د/مها عبد المنعم حسن ، د/ هشام جلال مهران السيد ، ا.د/ زكريا موافى إمام موافى، جامعة القاهرة ،كلية العلاج الطبيعي، قسم العلاج الطبيعي للجراحة.

المستخلص

كان الهدف من البحث هو استكشاف تأثير العلاج بالجاليوم- ارسينايد ليزر في تعجيل التئام قرح الفراش. اشترك في هذه الدراسة 30 مريضاً من مرضى اصابات النخاع الشوكى ولديهم قرح فراش، وكانت أعمارهم بين 30-50 سنة وتم تقسيمهم إلى مجموعتين متساويتين في العدد . المجموعة الأولى الضابطة تتلقى الرعاية الدورية للقرح ومجموعة علاجية تتلقى العلاج بالجاليوم- ارسينايد ليزر. ولقد أظهرت النتائج فروق ذات دلالة إحصائية عالية بين المجموعة العلاجية والمجموعة الضابطة في نهاية مدة العلاج، وكان العلاج بالجاليوم- ارسينايد ليزر مثمر ومؤثرفي تحسين وتعجيل التئام قرح الفراش كما هو مثبت بتقليل كلا من مساحة وحجم القرح.