The Use of Infrared Laser Therapy In the Treatment of Venous Ulceration

M.E. Sugrue, FRCSI, J. Carolan, BSc, E.J. Leen, MB, T.M. Feeley, FRCSI, D.J. Moore, FRCSI, G.D. Shanik, FRCSI, *Dublin Ireland.*

Management of intractable venous ulceration remains an unrewarding task, which is increasingly delegated to the realm of the vascular surgeon. The purpose of this pilot study was to assess the ulcer-healing effects of the newest form of biostimilation—the low power laser. Twelve patients with chronic venous ulcers unresponsive to conservative measures were treated with infrared laser irradiation for twelve weeks. Two ulcers healed completely and there was a 27% ($p<0.01$) reduction in size of the remaining ulcers. Treatment resulted in a 44% ($p<0.01$) increase in ulcer floor area occupied by healthy granulation tissue. The most dramatic effect of laser treatment was the reduction in ulcer pain, from 7.5 to 3.5 (linear analogue scale) ($p<0.001$). Laser irradiation had no effect on $TcPO_2$, number of skin capillaries of pericapillary fibrin deposition in the lipodermatosclerotic area around the ulcer. The results of this pilot study are encouraging and a carefully controlled randomized study is indicated to compare low power laser irradiation to conventional treatment in the management of venous ulcers. (Ann Vasc Surg 1990;4:179-181).

KEY WORDS: veins, lasers, ulcers, venous

Venous leg ulcers remain a significant cause of patient morbidity and financial strain on the health services [1]. Outcome of venous ulcer treatment has changed little in the past twenty years and compression bandaging, local dressings, bed rest and limb elevation remain the mainstay of treatment. More aggressive treatment as with skin grafting and utilization of new treatment modalities such as hyperbaric oxygen, ultrasound and ultraviolet light have failed to significantly improve results. Laser energy is the newest form of biostimulation and there are a number of encouraging reports on the use of low power laser irradiation in the treatment of inflammatory musculoskeletal conditions [2]. The purpose of this study was to assess the clinical and histological effects of laser therapy on venous ulcers.

METHODS

Twelve patients with protracted venous ulcer disease entered the study. All patients had venous hypertension as confirmed by venous Doppler ultra-sound and ambulatory venous pressure studies. Only chronic ulcers were selected for treatment, all had been dressed at our veins clinic at least twice weekly for a minimum of six months. Patients with peripheral vascular disease (A/B index <1), diabetes mellitus, or collagen disorders were excluded from the study.

Two laser systems were used: four patients were treated with an infrared Endolaser 465* and eight were treated with an infrared Benson IR CEB-S+ mid laser (Table 1). Both lasers were used according to the manufacturers' instructions. The Endolaser is hand held and laser energy was delivered for 15 seconds per cm^2 while the Benson apparatus

has a four probe cluster on an adjustable arm and energy was delivered to the underlying ulcer for four minutes. The maximum treatment per session was 20 minutes regardless of ulcer size. Patients were treated three times per week on an outpatient basis until the ulcer healed, or for a maximum of 12 weeks.

Prior to study the ulcer was photographed, measured and percentage floor area consisting of granulation tissue was recorded. The depth of the ulcer was assessed subjectively as superficial, intermediate or deep.

A 4-mm2 punch biopsy was taken from the intact skin immediately adjacent to the ulcer before and after treatment. Control biopsies were taken from mid-thigh level in all patients. All biopsy specimens were examined by a pathologist without knowledge of the biopsy site. Sections were stained with Hematoxolin and Eosin and Martius scarlet blue and phosphotungstic acid to identify fibrin. The number of capillaries per mm^2 was assessed using the method described by Burnand and associates [3].

Transcutaneous oxygen levels ($TcPO^2$) were measured using a Kontron triple channel device. Probes were placed on the lipodermatosclerotic area adjacent to the ulcer and on the forefoot and thigh. The sites of the initial biopsy and $TcPO^2$ measurement were marked on the first tracing of the ulcer so that the repeat studies could be taken from adjacent areas at the end of the study.

Ulcer pain was recorded pretreatment and at four-weekly intervals using a visual analogue scale.

During the study period ulcers were maintained on the same treatment regimen as was in use prior to the study.

RESULTS

HEALING

Two ulcers healed completely, one from each group following eight weeks and 10 weeks treatment, respectively. Overall, there was a significant reduction in ulcer size following treatment (27%; $p<0.01$, Paired t-test). In the Endolaser group mean ulcer size was reduced from 12.2 cm^2 to 9.3 cm^2 and from 14.2 cm^2 to 8.8 cm^2 in the Benson group. All ulcers were classified as deep prior to treatment and all except one became superficial. In four of the 10 unhealed ulcers a layer of epidermis migrated across the ulcer and covered 10% to 50% of the surface. This was extremely thin and when biopsied revealed a normal epidermal pattern. This delicate epidermal layer was lost from one of the ulcers when treatment was discontinued.

Ulcer floors before treatment consisted chiefly of unhealthy, necrotic tissue and the remainder consisted of granulation tissue. Following treatment there was a significant increase in ulcer floor area occupied by granulation tissue (44%; $p< 0.01$, Paired t-test).

Granulation tissue floor occupancy increased from 63% to 85% in the Endolaser and 30% to 85% in the Benson group.

PAIN

Prior to treatment ulcers were reported by the patients as being moderately painful, scoring 7.5 (mean) on a visual analogue scale. The most dramatic finding in the study was the marked reduction in ulcer pain. This was usually volunteered by the patients following only one or two treatment sessions. Mean analogue score was reduced from 8 to 3 in the Endolaser group and from 7.2 to 3.9 in the Benson group. The overall reduction in pain score was from 7.5 to 3.5 ($p<0.001$, Paired t-test).

Transcutaneous oxygen levels

In only one patient was there a noteworthy increase in $TcPO^2$ from 14 to 56 mm/Hg. This patient's ulcer healed. In the remaining patients there was no change in $TcPO^2$ measurements.

Histology

Skin biopsies adjacent to the ulcer areas showed a significantly greater capillary density than controls. There was no change in the number of capillaries posttreatment. Pericapillary fibrin was identified in nine of the twelve patients, and was considered marked in four, and was seen in none of the controls. Fibrin levels were unchanged after treatment.

DISCUSSION

The plethora of therapeutic modalities used in the treatment of venous ulceration is, in itself, testimony to the lack of efficacy of any one measure. Recently, a number of reports have claimed various beneficial biological effects with the use of laser radiation [2]. Low power infrared lasers similar to those used in this study have been reported to promote wound contraction and healing [4,5]. This study set out, therefore, to assess the effect of laser irradiation on ulcer healing. As this was a pilot study, chronically indolent ulcers were selected and no attempt was made to compare laser treatment with any other treatment modality, nor did we attempt to compare results of the two laser systems.

New venous ulcers presenting to a clinic will heal in 40-80% of cases over a 12 week period with conservative compression bandaging combined with bed rest and elevation [6]. In order to rule out spontaneous ulcer healing we studied chronic ulcers which were resistant to standard therapy. The ulcers were invariably large and painful and 80% had been treated by skin grafting on one or more occasions. Apart from complete healing in two of 12 ulcers and a 30% reduction in ulcer size, there was a marked change in the

nature of the ulcer base where necrotic slough was replaced by healthy granulation tissue in 90% of ulcers.

As the group had undergone no change in dressing routine and no new therapy was used during the study, these improvements were almost certainly related to the laser therapy.

The marked improvement in ulcer healing is not unexpected. Animal studies [4] have shown acceleration of wound contraction associated with increased fibroblasts in the wound bed induced by helium-neon laser irradiation. In vitro studies have shown the helium-neon laser stimulates human fibroblast proliferation [7]. While several theories have been proposed to explain these laser effects, the mechanism of action is not clear but is though not to be related to heat production.

Perhaps the most dramatic effect of laser treatment in this study was the reduction in ulcer pain. As patients were aware they were receiving active therapy, it is impossible to rule out a placebo effect and a blind randomized trial would be necessary to accurately assess the laser analgesic effect. Low power laser has been shown to have an analgesic effect in the treatment of supraspinatus and bicipital tendonitis [8] and neuralgia and arthritis [9]. The mechanism of this laser-induced analgesic effect is not known but it is not thermally induced. It has been proposed that the analgesic effect is produced in a manner similar to that obtained with transcutaneous nerve stimulation (TENS) [9].

The marked increase in the number of skin capillaries in the ulcer bearing area compared to the normal skin and pericapillary fibrin deposition are similar to the findings of Burnand and colleagues [3, 10]. Laser treatment did not affect the number of skin capillaries or the pericapillary fibrin levels.

In only one patient did the $TcPO^2$ in the ulcer area increase dramatically; in this patient the ulcer healed. As the reduced $TcPO^2$ in the lipodermatosclerotic area seems to be due to pericapillary fibrin deposition, failure to improve $TcPO^2$ was not expected.

The results of this study suggest there may be a role for low power laser in the treatment of venous ulcers, particularly those which are painful and resistant to other measures. A blind, randomized study on carefully selected ulcers is essential to establish the role, if any, for the low power laser in the treatment of venous ulceration.

ACKNOWLEDGEMENT

We wish to thank The Benson Laser Corporation Limited for the use of the Space Mid IR CEB-S appliance, and for their advice and guidance. We also acknowledge the help of Catmar Ltd, Dublin, Ireland who kindly gave the Endolaser appliance for use in this study.

REFERENCES

1. O'DONNEL TF, BROWSE NL, BURNAND KG, et al.
   The socioeconomic effects of iliofemoral venous thrombosis.
   J Surg Res 1977;22: 483-487.
2. MESTER E, MESTER AF, MESTER A, The biomedical effects of laser application.
   Lasers in Surgery and Medicine 1985;5: 31-39.
3. BURNAND KG, WHIMSTER IW, CLEMENSON G, LEA THOMAS M, BROWSE NL. The relationship between the number of capillaries in the skin of the venous ulcer bearing area of the lower leg and the fall in foot vein pressure during excersize. *Br J Surg* 1981;68: 297-300.
4. DYSON M, YOUNG S. Effect of laser therapy on wound contraction and cellularity in mice. Lasers in Medical Science 1986; 1: 125-130.
5. MESTER E, JASZAGI-NAGY. The effect of laser irradiation on wound healing and collagen synthesis. *Studia Biophys* 1973;35: 227-234.
6. MOFFATT C, WRIGHT DD, CCCOLLUM CN, GREEHHALGH RM. Community venous ulcer clinics: A new approach to healing. *Proc the Venous Forum, Autumn Meeting* 1988;10.
7. BOULTON M, MARSHALL J. HeNe laser stimulation of human fibroblast proliferation and attachment in vitro. *Lasers in the Life Sciences* 1986;1: 125-134.
8. ENGLAND SM, COPPOCK JS, STRUTHERS GR, BACON PA. An observer blind trial of I.R. ceb mid-laser therapy in bicipital tendonitis and supraspinatus tendonitis. *Proc Int Cong Laser in Medicine and Surgery, Bologna*, 1985;413-414.
9. WALKER J. Relief from chronic pain by low power laser irradiation. *Neuroscience Letts* 1983;43: 339-344.
10. BURNAND KG, WHIMSTER I, NAIDOO A, BROWSE NL. Pericapillary fibrin in the ulcer-bearing skin of the leg: the cause of lipodermatosclerosis and venous ulceration. *Br Med J* 1982;285: 1071-1072.