Prospective, Single-blind, Randomized, Controlled Study to Assess the Efficacy of the 585-nm Flashlamp-Pumped Pulsed-Dye Laser and Silicone Gel Sheeting in Hypertrophic Scar Treatment

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Objective: To determine the efficacy of the 585-nm flashlamp-pumped pulsed-dye laser and silicone gel sheeting in the treatment of hypertrophic scars in lighter- and darker-skinned patients.

Design: Prospective, single-blind, randomized, internally controlled, comparison investigation.

Setting: Large academic dermatology department.

Patients: Twenty patients with hypertrophic scars (19 completed the laser treatments and 18 completed the silicone gel sheeting treatments).

Main Outcome Measures: Clinical measurements included hypertrophic scar blood flow, elasticity, and volume. Patients’ subjective complaints of pruritus, pain, and burning were also monitored. Histological assessment of fibrosis, number of telangiectasias, and number of mast cells was performed. Statistically significant improvements in clinical measurements and patients’ subjective complaints determined treatment success.

Results: Mean scar duration was 32 months (range, 4 months to 20 years). There was an overall reduction in blood flow, volume, and pruritus over time (P = .001, .02, and .005, respectively). However, no differences were detected among treatment and control groups. There was no reduction in pain or burning (0-40 weeks), elasticity (8-40 weeks), or fibrosis (0-40 weeks, n = 5 biopsies) in the treated or control sections of the scars. Unlike in a previous study, the number of mast cells in the scars was similar to the number of mast cells in healthy skin.

Conclusion: Clinical results demonstrate that the improvements in scar sections treated with silicone gel sheeting and pulsed-dye laser were no different than in control sections.

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ABNORMAL scarring was first described in the Smith papyrus between 2500 and 3000 BC.1 In 1817, Aliberti2 proposed the word “cheloide” (“keloid”) to differentiate these lesions from malignant neoplasms. Today, keloids and hypertrophic scars are recognized as unchecked proliferations of fibrous tissue after injury to the skin.3 Whereas hypertrophic scars remain within the confines of the original wound, keloids invade the surrounding skin.3

Keloids and hypertrophic scars are common (keloids alone develop in 5% to 15% of wounds4) and can cause functional and psychological morbidity. Many treatments such as use of intralesional corticosteroids, excision, pressure therapy, radiotherapy, cryotherapy, and carbon dioxide laser ablation have been tried with limited success.5-7 Results of more recent studies show the 585-nm flashlamp-pumped pulsed-dye laser (FLPDL)8-12 and silicone gel sheeting (SGS)13-26 to be effective in hypertrophic scar therapy. However, few studies were masked or well controlled. In addition, use of the FLPDL has not been studied on darker-skinned people because melanocytes compete with hemoglobin for laser absorption.27 The paucity of FLPDL studies on darker-skinned people is unfortunate because the risk of keloid formation is 2- to 14-fold higher in this patient population.28

The cause of hypertrophic scars and keloids is unknown. The abundant collagen accumulation and fibroplasia might result from either excessive synthesis of collagens, fibronectin, and proteoglycans or deficient matrix degradation and remodeling.5

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Note: This article is also available on our Web site: www.ama-assn.org/derm.
PATIENTS AND METHODS

PATIENTS

Twenty patients older than 18 years with uniform, linear hypertrophic scars and skin type I, II, III, IV, V, or VI were enrolled between December 1, 1996, and May 31, 1997. Each hypertrophic scar was secondary to surgical wounds. Women of childbearing potential required a negative pregnancy test result before enrollment and agreed to use birth control for the study duration. Exclusion criteria included treatment of the scar within the preceding 2 months, keloidal scarring, and scars less than 8 cm long. Informed written consent was obtained from all patients, and the study was approved by the institutional review board of the Henry Ford Health System, Detroit, Mich.

STUDY DESIGN

This was a prospective, single-blind, randomized, internally controlled study. Each scar was divided into 3 sections, and each section was either randomly assigned to 1 of 2 treatments (FLPDL or SGS) or designated as control using a computer-generated randomization list (see later herein). Measurements for blood flow and volume were taken at 0, 8, 16, 24, and 40 weeks (16 weeks after discontinuation of therapy). Measurements for elasticity were taken at 8, 16, 24, and 40 weeks (the elastometer was not available at week 0). At each visit, the scars were photographed and a questionnaire was completed addressing scar pain, pruritus, and burning. Patients who consented underwent punch biopsies at 0 and 40 weeks (the elastometer was not available at week 0). Half of each biopsy sample was evaluated for routine histological features, the other half for cytokine messenger RNA levels (G.P.W., G.M.S., and D.P.F., unpublished data, 1998).

At baseline, each scar was divided into 3 equal sections (see previous paragraph). The boundaries of the treatment and control sites were marked with ink, and mirror images of the scars were traced onto clear plastic 8 1/2 × 11" sheets and used as templates. One template was kept in the patient’s chart and referred to during each visit, and 1 template was given to the patient and used as a guide for home placement of the SGS. Patients were instructed to wear SGS (Cica Care; Smith and Nephew, Largo, Fla) on the designated site for at least 12 continuous hours per day and to wash the SGS and test site with soap and water before and after treatment. The SGS was held in place using a dressing retention sheet (Hypafix tape; Smith and Nephew). Patients (n = 18) were treated for 24 weeks with SGS. One section of each patient’s scar was treated with an FLPDL (model SPTL-1; Candela Laser Corp, Wayland, Mass) at a wavelength of 585 nm, a pulsed duration of 450 microseconds, fluence per pulse between 6.5 and 8.0 J/cm², and a spot size of 5 mm with a 10% to 20% overlap. The fluence used on each patient was determined by the threshold dose—the lowest dose that produced nonblanchable purpura filling the entire spot size. Patients were given the option of applying a topical anesthetic agent (2.5% lidocaine and 2.5% prilocaine cream) 2 hours before laser therapy. After laser treatments, the site was iced for 15 to 20 minutes. Patients (n = 19) received 4 laser treatments at 8-week intervals. One section of the scar was randomized to control and left untreated for the study duration.

Questionnaires

At each visit, patients rated their pain, burning, and pruritus based on a quartile scale from 1 (absent or minimal) to 4 (severe).

Photography

A camera (model N70; NikonUSA, Melville, NY) with a 60-mm macro lens (NikonUSA) and ringflash (model SB-17B; NikonUSA) was used to photograph the 3 sections of the scars using 100 ASA film (Kodak Ektachrome, lot number 1681; Eastman Kodak Co, Rochester, NY). The film was stored at −20°C and allowed to thaw at least 1 hour before use. Lens aperture, exposure time, subject distance, and room lighting were kept constant for each patient.

Blood Flow (Erythema)

Blood flow was evaluated using a laser doppler (Laserflo BPM²; Vasamedics Corp, St Paul, Minn), as previously described by Ehrlich and Kelley. 29 (The laser doppler measures erythema by determining blood flow [in milliliters per minute per 100 g of tissue] noninvasively 1 to 1.5 mm beneath the skin surface.) Patients were allowed to acclimate to room temperature for a minimum of 15 minutes before monitoring. Also, in patients who used the topical anesthetic agent (n = 7), the agent was removed 15 minutes before monitoring. In the center of each scar section, the laser probe was affixed to the skin with a double-sided adhesive disk and left in place for 60 seconds until a stable reading was obtained. This procedure was repeated in an area of adjacent healthy skin 3 cm from the scar. The measurements were taken by a masked observer at constant sites on the skin during each visit. Only the patient and the individual performing the treatments knew the treatment assignments.

Elasticity

Elasticity was evaluated with the handheld elastometer, as previously described by Bartell et al. 30 With the limbs of the elastometer fully apposed by manual pressure, the limbs were affixed to the skin with double-sided adhesive tape, leaving an intervening segment of skin exactly 10 mm long. The manual pressure was released, and after 3 seconds, a stable reading was recorded. One measurement was taken in the center of each scar section along its longitudinal or long axis. Measurements were taken by a masked observer at constant sites during each visit.

The elastometer uses a constant-tension spring and a strain gauge to distract 2 adjacent points of skin. The distance of distraction was measured and expressed as the percentage of stretch using the following formula:
To determine whether SGS and the FLPDL are effective in treating patients of all skin types with hypertrophic scars, a prospective, single-blind, randomized, internally controlled study was conducted comparing the 2 treatments in a diverse patient population. Unlike in previous FLPDL studies, 50% of our patients had darker skin types (V or VI); therefore, we used slightly higher FLPDL fluences (6.5-8.0 J/cm²) compared with previous studies. The interaction between treatment and time was statistically significant. An overall treatment effect was detected (P = .01); ie, the overall reduction in burning over time was statistically significant. Only the control sections showed significant improvement in burning scores from week 0 to week 40 (mean ± SD burning reduction score = 0.58 ± 0.77; P = .01). The differences for the other 2 treatments were not statistically significant (SGS, P = .13; FLPDL, P = .12).

**RESULTS**

**CHARACTERISTICS OF THE STUDY POPULATION**

Of approximately 150 patients who were screened, 20 (15 women and 5 men; mean age, 49 years) were enrolled in the study (Table). Fifty percent of the patients had skin type II, 40% had type VI, and 10% had type V. Mean scar duration was 32 months; however, 8 patients’ scars were younger than 9 months. Patient 10 did not use SGS because of skin irritation. Patient 6 dropped out of the study at week 24 because of pain during laser treatments. She received a total of 2 FLPDL treatments and 8 weeks of SGS treatment. Her final scar evaluation was 16 weeks after treatments were discontinued.

**TREATMENT RESULTS**

**Questionnaires**

**Pain.** The interaction between treatment and time was not statistically significant (P = .41). In other words, the 3 treatments followed similar trends over time. No overall treatment effect was detected (P = .18). Also, no overall time effect was detected (P = .61).

**Burning.** The interaction between treatment and time was not statistically significant (P = .74). A trend was detected in the overall treatment effect (P = .05). Pairwise comparisons of the 3 treatments (averaged over time) showed a difference between control and FLPDL treatment of borderline statistical significance (P = .02). Differences between control and SGS treatment and between SGS and FLPDL treatments were not statistically significant (P = .11 and .08, respectively). For pairwise comparisons, P < .01 was considered statistically significant and P ≤ .01 and P ≤ .05 was considered borderline statistically significant. An overall time effect was detected (P = .01); ie, the overall reduction in burning over time was statistically significant. Only the control sections showed significant improvement in burning scores from week 0 to week 40 (mean ± SD burning reduction score = 0.58 ± 0.77; P = .01). The differences for the other 2 treatments were not statistically significant (SGS, P = .13; FLPDL, P = .12).
were no differences detected among treatments for a change in burning scores from week 0 to week 40 (control vs SGS, P = .50; control vs FLPDL, P = .75; and SGS vs FLPDL, P > .99).

**Pruritus.** Figure 1 shows the mean ± SEM pruritus values for each of the 3 groups at the 5 measurement times.

The interaction between treatment and time was not statistically significant (P = .74). As with the pain and burning scores, the 3 treatments followed similar trends over time. No overall treatment effect was detected (P = .12). An overall time effect was detected (P = .05); ie, the overall reduction in pruritus over time was statistically significant. All 3 sections showed significant improvement in pruritus from week 0 to week 40 (control, P = .003; silicone gel sheeting (SGS), P = .05; and 595-nm flashlamp-pumped pulsed-dye laser (FLPDL), P = .02). No differences among groups: control vs SGS, P = .25; control vs FLPDL, P > .99; and SGS vs FLPDL, P > .99. Weeks 0 to 24: n = 20; week 40: n = 19.

**Blood Flow (Erythema)**

Figure 2 shows the mean ± SEM blood flow values for each of the 3 groups at the 5 measurement times.

The interaction between treatment and time was not statistically significant (P = .21). No overall treatment effect was detected (P = .15). An overall time effect was detected (P = .001). Pairwise comparisons of the measurement times (averaging the different treatments) showed that the differences between week 40 and weeks 0 (P < .002), 8 (P = .001), 16 (P = .006), and 24 (P = .003) were statistically significant. The differences between week 0 and weeks 8 (P = .02), 16 (P = .01), and 24 (P = .02) were of borderline statistical significance.

All 3 scar sections showed a significant improvement in erythema from week 0 to week 40 (control, P < .001; SGS, P = .02; and FLPDL, P = .002). However, no differences were detected in the change from week 0 to week 40 among groups (control vs SGS, P = .24; control vs FLPDL, P = .26; and SGS vs FLPDL, P = .73). Weeks 0 to 40: n = 20; week 40: n = 19.

**Figure 1.** Time course for hypertrophic scar pruritus. Data are shown as mean ± SEM (rated on a quartile scale: 1 indicates absent or minimal; 4, severe). Overall reduction in pruritus over time, P = .005. Reduction in pruritus between weeks 0 and 40: control, P = .003; silicone gel sheeting (SGS), P = .05; and 595-nm flashlamp-pumped pulsed-dye laser (FLPDL), P = .02. No differences among groups: control vs SGS, P = .25; control vs FLPDL, P > .99; and SGS vs FLPDL, P = .53. Weeks 0 to 24: n = 20; week 40: n = 19.

**Figure 2.** Time course for hypertrophic scar and healthy skin blood flow. Data are shown as mean ± SEM. Overall reduction in blood flow over time, P = .001. Reduction in blood flow between weeks 0 and 40: control, P < .001; silicone gel sheeting (SGS), P = .02; and 595-nm flashlamp-pumped pulsed-dye laser (FLPD), P = .002. No differences among groups: control vs SGS, P = .24; control vs FLPDL, P = .26; and SGS vs FLPDL, P = .73. Weeks 0 to 24: n = 20; week 40: n = 19.

**Elasticity**

Week 0 elasticity measurements were done in only 4 patients and therefore were excluded. The interaction between treatment and time was not statistically signifi-
control vs FLPDL, 0 to week 40 among the 3 groups (control vs SGS, 0 to week 40 and baseline showed a trend toward overall reduction in volume over time, \( P = .02 \). Reduction in volume between weeks 0 and 40: control, \( P = .007 \); silicone gel sheeting (SGS), \( P = .35 \); and 585-nm flashlamp-pumped pulsed-dye laser (FLPDL), \( P = .53 \). No differences among groups: control vs SGS, \( P = .09 \); control vs FLPDL, \( P = .13 \); and SGS vs FLPDL, \( P = .85 \). Weeks 0 to 24: \( n = 20 \); week 40: \( n = 19 \).

No overall treatment (\( P = .56 \)) or time (\( P = .41 \)) effects were detected.

**Volume**

Figure 3 shows the mean ± SEM volume values for each of the 3 groups at the 5 measurement times.

The interaction between treatment and time was not statistically significant (\( P = .51 \)). No overall treatment effect was detected (\( P = .23 \)). An overall time effect was detected (\( P = .02 \)). Pairwise comparison of the measurement times (averaging the different treatments) showed that the differences between weeks 40 and weeks 8 (\( P = .01 \)), 16 (\( P = .02 \)), and 24 (\( P = .01 \)) were of borderline statistical significance. Also, the difference between week 40 and baseline (week 0) showed a trend toward overall volume reduction (\( P = .06 \)).

Only the control showed a significant improvement in volume from week 0 to week 40 (\( P = .007 \)). No differences were detected when comparing the change from week 0 to week 40 among the 3 groups (control vs SGS, \( P = .09 \); control vs FLPDL, \( P = .13 \); and SGS vs FLPDL, \( P = .85 \)).

Figure 4 illustrates the overall improvement in a vertical suprapubic hypertrophic scar (patient 5) from week 0 to week 40.

**Histological Analysis**

Five of 20 patients consented to undergo biopsy examination, which showed no change in fibrosis, number of telangiectasias per 20× field, or number of mast cells per 40× high-power field from week 0 to week 40 in any section of scars (\( P > .30 \) for all comparisons). Average ± SD number of blood vessels per 20× field was appreciably higher in the scar sections compared with healthy skin (pretreatment: 13.8 ± 2.1 vs 2.6 ± 1.4, \( P < .001 \); posttreatment: 14.5 ± 3.4 vs 2.6 ± 1.4, \( P = .005 \)). Average ± SD number of mast cells was no different in the scar sections compared with healthy skin (pretreatment: 3.5 ± 2.0 vs 4.6 ± 2.7, \( P = .45 \); posttreatment: 3.5 ± 1.4 vs 4.1 ± 1.7, \( P = .64 \)). Each biopsy sample displayed acanthosis and loss of the rete ridges.

**COMMENT**

Hypertrophic scars are common and can present functional and psychological challenges to the patient. The FLPDL and SGS have been shown in clinical studies to be effective treatment for hypertrophic scars. However, most of these studies were neither controlled nor masked, lacked objective means to measure improvement, and had little or no follow-up. Our study was controlled, masked, and used quantifiable means for measurement; patients were also followed up for 4 months after discontinuing the treatments. Moreover, FLPDL treatment has not been studied on darker-skinned patients; 50% of our patients had skin type V or VI. Blood flow decreased from 0 to 40 weeks in each of the 3 scar sections, but no differences were detected among groups, ie, control vs SGS. Prolonged angiogenesis is a contributing factor to hypertrophic scar formation. Use of the FLPDL damages cutaneous blood vessels through selective photothermolysis: hemoglobin absorbs light of a specific wavelength, generating heat and leading to coagulation necrosis. Selective photothermolysis is the proposed mechanism by which the FLPDL controls hypertrophic scar growth. However, in the FLPDL-treated section of the scars, blood flow between 8 and 24 weeks actually increased and remained elevated until week 40. It is possible that use of the FLPDL may have caused dermal necrosis, resulting in compensatory neovascularization. Hohenleutner et al reported focal dermal coagulation with FLPDL treatment at 7.5 to 8.0 J/cm².
We used slightly higher fluences (6.5-8.0 J/cm²) than previous studies: Goldman and Fitzpatrick⁹ and Dierickx et al¹² used fluences between 6.0 and 7.5 J/cm², Alster and Williams¹⁰ used fluences between 6.5 and 7.25 J/cm² (mean, 7.0 J/cm²), and Alster¹¹ used fluences between 6.5 and 6.75 J/cm². Eleven of our patients received fluences of 7.5 J/cm², and 4 received fluences of 8.0 J/cm². Sixteen weeks after discontinuing laser treatment, there was a trend toward reduction in blood flow. If there was dermal damage, the 8-week treatment and evaluation intervals may not have been of sufficient length to allow full recovery.

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CAR VOLUME decreased only in the control section. In the FLPDL-treated section, there was a trend from week 8 to week 24 toward an increase in scar volume. It is possible that FLPDL treatment caused hypertrophic scar formation, leading to an increase in volume. Elasticity, pain, and burning did not change in any of the groups; these factors may be more resistant to change. Similarly, there was no change in scar fibrosis or in the number of telangiectasias.

Why did the FLPDL- and SGS-treated sections fail to demonstrate clinical improvement over the control sections? As mentioned, use of the FLPDL is thought to inhibit scar formation through selective photothermolysis.⁸,¹⁰ This mechanism makes FLPDL the treatment of choice for port-wine stains.¹³,¹⁵ Because the deepest target vessels are only 0.5 to 0.7 mm from the dermo-epidermal junction in children and only 1.0 mm in adults,¹⁶ Because hypertrophic scars are several millimeters thick, many of the target vessels are much deeper than in port-wine stains;¹⁷ vessel coagulation depth of the FLPDL has been reported to be between 0.4 and 1.2 mm.²⁰ Another factor is that 8 of our patients had skin type VI and 2 had type V. Melanin acts as a competing chromophore for the FLPDL.²⁵ We used higher fluences in our darker-pigmented patients to try to compensate for the effect of melanin. However, the depth of penetration of the laser was likely less in these patients. Additional analyses were performed separating type II patients from those type V and VI; the results did not significantly deviate from previous findings. Previous studies¹¹,¹² that used fluences between 6.0 and 7.5 J/cm² to treat hypertrophic scars found no significant difference in treatment outcome vs the fluence of the laser. However, darker-pigmented patients were not included in those studies.

The mechanism of action of SGS is unknown. Silicone gel was first reported in 1982 by Perkins et al²⁶ to be effective therapy in preventing burn scar contractures and hypertrophy. In contrast to compression garments used for burn scars, the mechanism of action for silicone gel was thought to be unrelated to pressure.²⁵ In 1985, Quinn et al²³ reported that silicone gel exerted much less than the 15 to 40 mm Hg required for hypertrophic scar pressure therapy; they also excluded temperature effects and differences in oxygen transmission as other possible mechanisms. They concluded that silicone gel treatment probably improves hypertrophic scars by releasing a low-molecular-weight silicone fluid and by hydrating the stratum corneum.²³ However, in 1989, Ahn et al²² found no histological evidence of silicone in biopsy specimens of SGS-treated scars. Results of more recent studies¹³,¹⁴,³⁹ show that occlusive dressings, with or without silicone, can be effective in treating hypertrophic scars and conclude that hydration, not silicone, is what modulates scar formation. A reduction in fibroblast production and collagen proliferation in hydrated or occluded wounds has also been reported.⁴⁰,⁴¹ Chen et al²⁸ found elevated levels of metalloproteinases in wound fluid collected under occlusive dressings. In normal wound healing, the proteases are important in degrading the extracellular matrix and thus, in controlling scar formation.²¹ If SGS treatment reduces hypertrophic scar formation, it is possibly through regulation of the cytokine network.²⁹

Three additional factors may have resulted in similar clinical improvements among the control and treated sections. First, because the treated areas were adjacent to the control sites, the SGSs or FLPDL may have had a “global” effect resulting in clinical improvement in the control areas. Second, despite every effort to ensure proper placement of the SGSs, patients may have inadvertently overlapped the control sites with the SGS. Third, approximately 50% of the hypertrophic scars, which often undergo at least partial spontaneous resolution, were younger than 1 year. Hypertrophic scars this young (n = 10) may have improved, with or without treatment; however, a subset analysis of scars older than 1 year (n = 10) showed similar improvements in the control and treated sections.

Hypertrophic scars younger than 1 year were included in our study for a few reasons. First, we hypothesized that if any hypertrophic scars were to improve with FLPDL treatment, it would be the younger, erythematous (more vascular) hypertrophic scars. Second, we hypothesized that early, interventional treatment would prevent hypertrophic scars from getting worse. Finally, several uncontrolled SGSs and FLPDL hypertrophic scar studies have included hypertrophic scars younger than 1 year.¹²,¹³,¹⁴,¹⁵,²²,²⁵,²⁶ In these articles, it is not clear whether the hypertrophic scars would have resolved without treatment. A control site was included in our study to account for the possibility of spontaneous hypertrophic scar resolution.

A potential limitation with this study was the reduced sample size and statistical power. It was initially designed to have 90% power with 30 patients. Because of time restrictions, only 20 patients were enrolled and of them, 19 completed the laser treatments and 18 completed the SGS treatments. With this sample size, the power to detect minimal differences of the magnitude given in the “Statistical Methods” section was reduced to 74%. Statistical power of 74% is still reasonable and should not be considered a major limitation of this study.

This is the first controlled hypertrophic scar study to demonstrate that reductions in blood flow, volume, and pruritus are no different in control and SGS- or FLPDL-treated sections of the scars. Several past studies that showed improvement with SGS and FLPDL treatment were not controlled. The improvements they ob-
served may have been secondary to normal hypertrophic scar regression. As such, further controlled studies of the effects of SGS and FLPDL treatment on hypertrophic scars need to be performed.

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REFERENCES


