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# Prolonged Clinical and Histologic Effects from CO<sub>2</sub> Laser Resurfacing of Atrophic Acne Scars

SUNILA WALIA, MD AND TINA S. ALSTER, MD

*Washington Institute of Dermatologic Laser Surgery, Washington, DC*

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**BACKGROUND.** The recent development of high-energy pulsed CO<sub>2</sub> lasers that minimize thermal injury to uninvolved adjacent structures has revolutionized the manner in which atrophic facial scars are recontoured. Significant improvement of atrophic scars with laser resurfacing has clearly been demonstrated; however, the exact timing for assessment of skin for further treatment has varied due to the unknown amount of time needed after laser scar resurfacing to effect maximal collagen formation and remodeling.

**OBJECTIVE.** The aim of this study was to determine the immediate and long-term (12–18 months) histologic and clinical effects of atrophic acne scars after CO<sub>2</sub> laser resurfacing in order to provide physician guidelines for postoperative clinical assessment for retreatment.

**METHODS.** Sixty patients (50 women, 10 men, mean age 38 years, skin types I–V) with moderate to severe atrophic facial scars were evaluated. Nineteen patients received regional cheek treatment and 41 patients received full-face resurfacing with a high-energy pulsed CO<sub>2</sub> laser. Independent clinical assessments

of treated scars were performed at 1, 6, 12, and 18 months and blinded histologic analyses were made of skin biopsies immediately prior to and after laser resurfacing, and at 1, 6, 12, and 18 months postoperatively in six patients.

**RESULTS.** Significant immediate and prolonged clinical improvement in skin tone, texture, and appearance of CO<sub>2</sub> laser-irradiated scars was seen in all patients. Average clinical improvement scores were 2.22 (69%) at 1 month, 2.1 (67%) at 6 months, 2.37 (73%) at 12 months, and 2.5 (75%) at 18 months. Continued collagenesis and subsequent dermal remodeling were observed on histologic examination of biopsied tissue up to 18 months after surgery.

**CONCLUSION.** Continued clinical improvement was observed as long as 18 months after CO<sub>2</sub> laser resurfacing of atrophic scars, with an 11% increase in improvement observed between 6 and 18 months postoperatively. We propose that a longer postoperative interval (12–18 months) prior to assessment for re-treatment be advocated in order to permit optimal tissue recovery and an opportunity for collagen remodeling.

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ATROPHIC FACIAL ACNE scars occur frequently, most often as a consequence of severe acneiform episodes during the teenage years. Many patients seek treatment for the resultant disfigurement not only because of the obvious variations in skin texture, but also for the limitations placed on social interaction, self-esteem, and daily activities. Historically acne scars have been notoriously difficult to treat owing to the extensive underlying collagen destruction and to the inherent limitations of available technology.

Treatments for atrophic facial acne scars have included a multitude of chemical, physical, and surgical options.<sup>1</sup> For decades, dermabrasion was the best and only viable option available for remodeling atrophic scars. Successful outcomes with this procedure are highly dependent on the surgeon's skill and experience and to a lesser degree on the patient's healing response and medical history. Intraoperative bleeding impedes visualization of the treatment site and increases the possibility of infection. Postoperative complications such

as dyspigmentation, fibrosis, and milia formation are common. Combining dermabrasion with punch grafting may improve clinical outcomes, but it also raises the level of surgical skill required to achieve a cosmetically acceptable result. Dermal injection of filler materials such as collagen, silicone, fat, and fibrin below pitted or atrophic scars offers a simpler and safer alternative to dermabrasion. However, as most of these materials will eventually resorb within 6 months, patients must commit to a series of ongoing treatments in order to maintain the desired clinical results.

The recent development of high-energy pulsed CO<sub>2</sub> lasers that minimize thermal injury to uninvolved adjacent structures has revolutionized the ongoing quest for recontouring atrophic facial scars. Although several studies have been published documenting the safety and efficacy of treating atrophic facial scars with high-energy pulsed CO<sub>2</sub> laser resurfacing,<sup>2–6</sup> a prospective analysis of clinical and histologic effects of cutaneous laser scar revision has never been conducted. While it is generally agreed that sufficient time is necessary after laser scar resurfacing to effect maximum collagen formation and remodeling, the exact timing for assessment of skin for further treatment has

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Address correspondence and reprint requests to: Tina S. Alster, MD, 2311 M St. NW, Suite 200, Washington, DC 20037, or e-mail: talster@skinlaser.com.

ranged from 2 months to 1 year. The aim of this study was to determine the immediate and long-term (12–18 months) histologic and clinical effects of atrophic acne scars after CO<sub>2</sub> laser resurfacing in order to provide physician guidelines for optimal timing of postoperative assessment and determination of treatment.

## Materials and Methods

Sixty consecutive patients (50 females, 10 males, ages 18–53 years, mean 38 years) with moderate to severe atrophic facial scars were evaluated. Skin phototypes I–V were included. No patients had received isotretinoin treatment within 2 years of entry into the study. Twenty-one patients (36%) had received prior dermabrasion to the involved areas. Eleven patients (19%) had collagen, silicone, or fat injections and 7 patients (12%) had undergone prior chemical peels (glycolic acid or trichloroacetic acid).

Nineteen patients (33%) received regional cheek treatment (extending from the nasolabial folds to the preauricular area and mandible) in an outpatient setting with a high-energy pulsed CO<sub>2</sub> laser. A 3 mm collimated spot was used to treat the areas in the ultra-pulse mode at 500 mJ energy and 5–7 W power. Forty-one patients (67%) received full-face CO<sub>2</sub> laser treatment at 300 mJ energy and 60 W power through an 8 mm square scanning handpiece. The entire face was treated with two laser passes at number 6 density, thoroughly removing partially desiccated tissue with saline-soaked gauze after each pass. Additional regional passes, as determined by the treating physician, were performed using 500 mJ and 5–7 W power with a 3 mm collimated handpiece. Laser pulses or scans were placed adjacent to one another without overlapping, thereby preventing char formation. Treatment end points were readily determined in the bloodless field by relative effacement of the scars or the appearance of yellowish discoloration within the laser-irradiated tissue.

Anesthesia was obtained with regional nerve blocks using 1% lidocaine with 1:200,000 epinephrine. In addition, for full-face procedures, intravenous anesthesia was administered by a certified nurse anesthetist using a combination of propofol, versed, fentanyl, and ketamine.

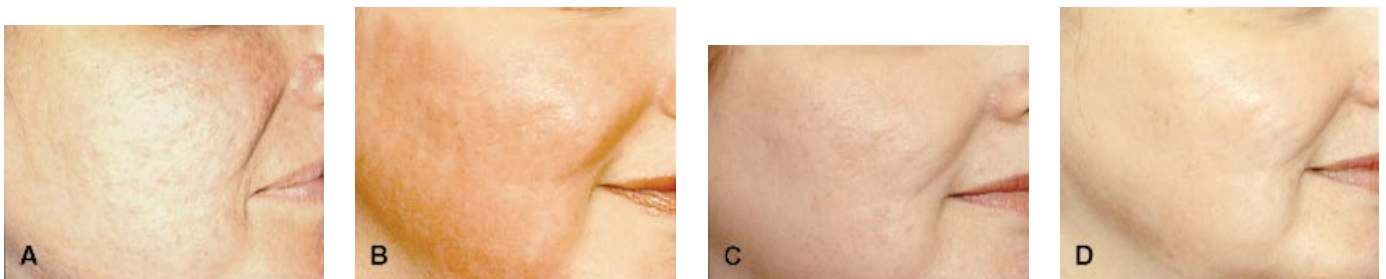
Immediately following treatment, Aquaphor ointment was applied to the irradiated skin. Each patient was instructed to perform gentle facial rinses with cool water several times daily, followed by liberal ointment application. Ice packs and round-the-clock acetaminophen were prescribed during the first 24–48 hours postoperatively to reduce swelling and discomfort. All patients were prescribed prophylactic antiviral treatment (acyclovir 400 mg three times a day or famciclovir or valacyclovir 250–500 mg twice a day). Patients were followed closely during the first postoperative week, during which time any residual coagulated debris was removed with gentle face steaming and acetic acid/hydrogen peroxide compresses. All patients were able to apply camouflage makeup with instruction by 7–10 days postoperatively.

Clinical documentation was obtained by sequential photographs using identical camera settings, lighting, and patient positioning. Clinical assessments of treated scars compared with baseline pretreatment photos were performed independently by two masked physician evaluators preoperatively, immediately postoperatively, and at 1, 6, 12, and 18-month intervals, using the following scale: 0, <25% improvement; 1, 25–50% improvement; 2, 51–75% improvement; 3, 76–90% improvement; 4, >90% improvement.

Skin punch biopsy specimens (3 mm diameter) were obtained from representative ice pick and atrophic scars prior to and immediately after laser resurfacing and at 1, 6, 12, and 18 months postoperatively in six patients. Skin samples were processed with hematoxylin and eosin and evaluated for epidermal and dermal changes, collagen/elastin content, and evidence of dermal remodeling by a board certified dermatopathologist in a blinded fashion.

## Results

Significant immediate and prolonged improvement in skin tone, texture, and appearance of CO<sub>2</sub> laser-irradiated scars was observed in all patients. Average clinical improvement scores were 2.22 at 1 month (mean improvement 69%), 2.1 (67%) at 6 months, 2.37 (73%) at 12 months, and 2.5 (75%) at 18 months (Figures 1–3). The slight worsening in clinical improvement seen between 1 and 6 months was attributed to



**Figure 1.** A) Preoperative appearance of patient with moderate to severe atrophic acne scarring. B) 1-month postoperative appearance demonstrating 70% scar improvement. C) 6-month postoperative appearance demonstrating a slight decrease in clinical improvement. D) 12-month postoperative appearance; patient exhibits a markedly improved complexion.



**Figure 2.** A) Severe, irregular, atrophic acne scars prior to surgery. B) 1-month postoperative appearance with good improvement. C) 6-month postoperative appearance; scars show relative worsening. D) 12-month postoperative appearance with significant improvement of all previous surface irregularities.

the resolution of postoperative edema with subsequent (temporary) return of skin surface irregularities. Erythema lasting an average of 3.5 months (range 6 weeks–6 months) was typical following laser treatment. The incidence of hyperpigmentation (36%) overall was slightly more prevalent in patients with darker skin tones, but was observed in all skin phototypes following laser irradiation. There was no incidence of infection, scarring, or delayed hypopigmentation.

Continued collagenesis and subsequent dermal remodeling were observed on histologic examination of biopsied tissue up to 18 months after surgery. In particular, 6-month postoperative specimens showed more densely packed collagen bundles. In addition, horizontal realignment of collagen fibers in the papillary dermis with increased collagen/elastin content (on elastin staining), normalization in polarity of keratinocytes, and a return of the rete ridges were demonstrated (Figure 4).

## Discussion

Clinical and histopathologic studies have previously demonstrated the efficacy of high-energy short-pulse CO<sub>2</sub> laser resurfacing in the improvement of facial atrophic acne scars, with 50–80% improvement typi-

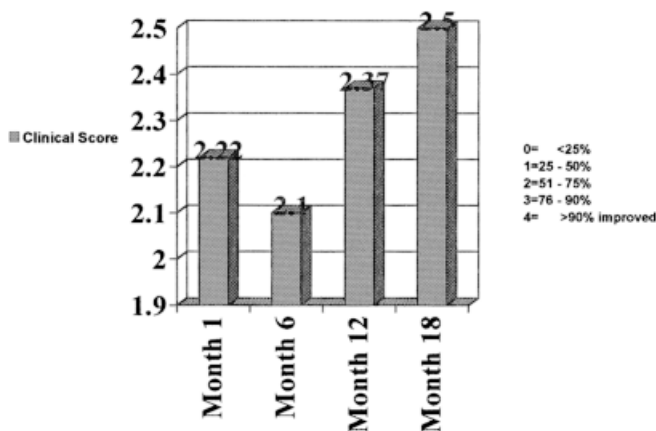
cally seen.<sup>2–8</sup> The differences in results reported with apparently similar laser techniques may be due to variations in the types of scars treated. Results may also vary due to differences in operative technique, as laser surgeons often use different laser systems, fluences, and end points for treatment. While some surgeons use the chamois-yellow color of the dermis as an indication of adequate treatment effect, others follow an observed shrinkage of dermal tissue as a clinical end point.

Cutaneous laser resurfacing involves four distinct mechanisms of wound creation and repair: controlled tissue vaporization, dermal heating with collagen shrinkage, neocollagenesis, and collagen remodeling. In other words, epidermal ablation and selected dermal injury occurs, permitting reepithelialization and wound repair over a contracted dermal bed. High-energy pulsed CO<sub>2</sub> laser irradiation optimally ablates tissue to a depth of 20–30  $\mu\text{m}$ , leaving a residual thermal damage zone of 50–150  $\mu\text{m}$  which augments skin tightening and collagen remodeling.<sup>9,10</sup> The laser-induced wound contracts secondary to tissue desiccation, with immediate collagen shrinkage effected through heating of the tissue to 55°C–60°C—a process that has been termed selective dermaplasty.<sup>11</sup> It is this partially denatured collagen that initially serves as a tighter (smaller) matrix for subsequent neocollagenesis.

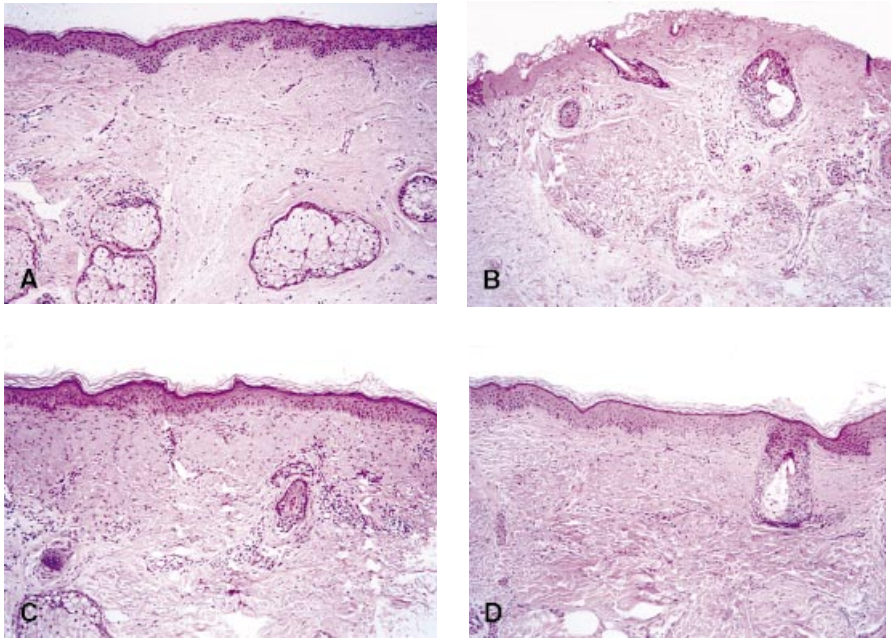
The final stage of CO<sub>2</sub> laser-induced wound healing involves an inflammation cascade, stimulating an increased production of myofibroblasts and matrix proteins (hyaluronic acid) and further ensuring a contracted scaffold for continued collagen deposition and remodeling.<sup>12</sup> Our investigation supports the importance of this last step (collagen remodeling) in providing prolonged clinical improvement after cutaneous laser resurfacing.

Histopathology findings paralleled the clinical improvement seen. Specifically, the prolonged and enhanced clinical improvement correlated directly with the degree of new collagen deposition and dermal remodeling observed histologically up to and beyond 12 months.

Thus our histopathology results mirrored the three phases of wound repair: the inflammatory phase, the



**Figure 3.** Clinical improvement scores.



**Figure 4.** A) Baseline (preoperative) biopsy specimen of atrophic acne scar showing dermal fibrosis. B) Immediately after CO<sub>2</sub> laser resurfacing, epidermal ablation is evident with residual thermal damage limited to the upper to mid-dermis. C) Six months postoperatively, chronic inflammation with new collagen formation is seen. D) Twelve months after laser resurfacing, horizontal realignment of collagen fibers, increased collagen/elastin content (on elastin stain), normalization of epidermal keratinocytes, and return of the rete ridge pattern are observed.

proliferative phase, and the remodeling phase.<sup>13</sup> There is little doubt that it is the third phase of wound repair—the remodeling phase—consisting of the deposition of matrix materials and their subsequent change over time, that is crucial for the continued improvement seen in laser scar revision. Dermal macromolecules such as fibronectin, hyaluronic acid, proteoglycans, and collagen are deposited during repair and serve as a scaffold for cellular migration and tissue support.<sup>14</sup> The deposition and remodeling of the extracellular matrix are dynamic processes. Long after the functional barrier of the skin is restored, events continue to occur that are related to wound injury and repair. The total amount of collagen increases early in the repair process, reaching a peak within 2–3 weeks after injury. Tensile strength, a functional assessment of collagen, increases by 40% one month after injury and may improve for as long as 1 year.

Changes in the types of collagen present and synthesized occur during the remodeling phase as well. While type III collagen predominates during initial wound repair, a return to a more stable preinjury phenotype, consisting largely of type I collagen, is seen after 1 year.<sup>13</sup> In addition, the composition of other matrix material within the wound changes as the amount of water and glycosaminoglycans decreases. The new connective tissue contains glycosaminoglycans and proteoglycans, the synthesis of which occurs concomitantly with the production of the new collagen. During this time, fibroblasts alter their phenotypic character to become myofibroblasts.<sup>13</sup> Demonstrating electron

microscopic characteristics of both smooth muscle cells and fibroblasts, these cells contain abundant rough endoplasmic reticulum needed for the production of large amounts of matrix proteins.<sup>12</sup> As a myofibroblast, the cell can also participate in wound contraction. Wound contraction is thought to be a result of the actin-rich myofibroblasts that align themselves along the lines of contraction. Stimulation by a number of mediators (angiotensin, epinephrine, norepinephrine, bradykinin) cause myofibroblasts to exhibit muscle-like contractions which are unified and require cell-cell and cell-matrix communication. Fibronectin also provides a provisional substratum for the migration and ingrowth of cells, a linkage for myofibroblasts to effect wound contraction, and a nidus for collagen fibrillogenesis.<sup>12</sup> Our investigation supports that this phenomenon is a continual process, lasting at least 12 months after CO<sub>2</sub> laser resurfacing.

We propose, therefore, that a longer postoperative interval be advocated in order to permit optimal tissue recovery and opportunity for collagen remodeling. It is this late phase that is so crucial for the significant improvement seen after CO<sub>2</sub> laser resurfacing of atrophic acne scars and, as such, it is beneficial to postpone a final clinical evaluation for at least 12–18 months before considering additional treatment.

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