# Combined therapy in the treatment of auricular keloids

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Abstract

Management of earlobe keloids is still controversial. Many different treatment modalities have been employed; however, no single approach has been completely successful. We used combination therapy that included compression therapy, laser excision, and serial steroid injection, which has improved our therapeutic outcomes for earlobe keloids.

## Introduction

Scar formation after cutaneous trauma, surgical or otherwise, encompasses a broad spectrum, ranging from normal scars to hypertrophic scars to keloids. The term keloid is derived from the Greek word cheloides, meaning "crab's claw," because of its lateral extensions, resembling the legs of a crab, growing into normal tissue.[1] Keloid scars outgrow the limits of the original wound as a result of excessive collagen deposition. Histologically, they are characterized by numerous fibroblasts and thick, glassy collagen bundles arranged haphazardly, with abundant mucinous ground substance. Often indistinct from hypertrophic scars histologically, keloids grow past the range of the wound scar (figure 1), whereas hypertrophic scars are a fibroblastic proliferation within the confines of the original wound scar.

Many theories exist to explain this haphazard collagen deposition in keloid scars. The scars essentially develop from an abnormality in the synthesis and degradation of collagen (which may have a genetic influence), incited by various tissue traumas, or may have an autoimmune or inflammatory component.

Earlobe keloids are more difficult to treat than those at other sites, with higher rates of failure and recurrence.[2] Numerous therapeutic modalities have been used to treat them, including intralesional steroid injection alone; surgical excision alone; surgical excision with skin grafting; surgery followed by radiation therapy; radiation therapy alone; systemic drug therapies, including vitamin E, penicillamine, colchicines, thiotepa, and tetrahydroxyquinone; massage; silicone gel sheeting; intralesional interferon; compression therapy; cryotherapy; laser excision; electrodessication; and topical medications such as 5-fluorouracil or bleomycin. Combination therapy appears to be the most effective treatment, although few studies have compared the various regimens.[3] No study has proven the effectiveness of one therapy over another.[4] Furthermore, no matter which form of treatment is instituted, the likelihood of keloid recurrence remains high.

We currently employ a combined approach, using the $CO_2$ laser for excision, followed by serial postoperative steroid injections. The goal of this study was to determine whether our combined approach is a viable treatment modality for earlobe keloids.

## Patients and methods

Patients. Our series includes 6 patients over a 2-year period (1999-2001). All 6 patients had a history of earlobe piercing. The age range of our patients was 15 to 33 years (mean, 25.6). Three patients had had no previous surgical excisions, 2 patients had had two previous excisions, and 1 patient had had one previous excision (none of these was performed at our institution). Three of the 6 patients, therefore, presented with a recurrent earlobe keloid after attempted surgical extirpation; the remaining 3 had primary keloids. All 3 patients with recurrent keloids had undergone "cold steel" surgical extirpation without adjunctive intralesional steroid injections.

Methods. We use a combination approach at our institution. We begin with a full-thickness excision that includes removal of the entire ear-piercing track from anterior to posterior ("dumbbell extirpation"), using the pulsed $CO_2$ laser (figure 2). After extirpation, anterior defects are closed primarily without tension, using 5/0 interrupted monofilament polypropylene sutures (Prolene; Ethicon,

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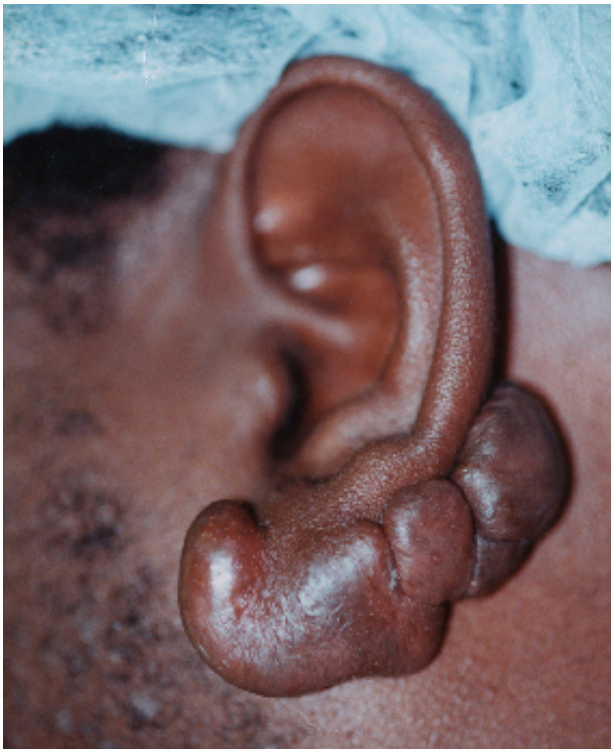

Figure 1. Photo shows a keloid of the left earlobe, preoperatively.

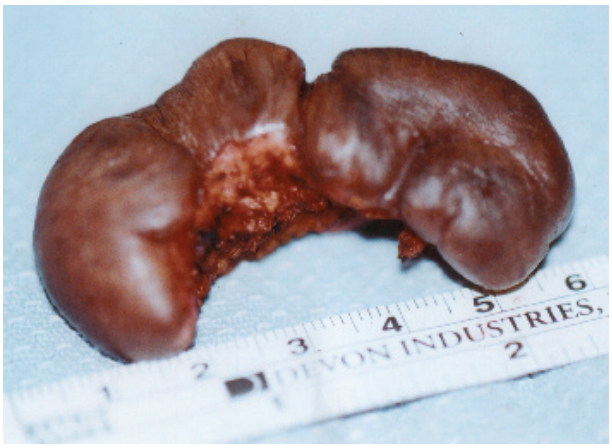


Figure 2. Photo shows the keloid excised from the left earlobe of the same patient shown in figure 1.

Inc.) only anteriorly; posterior defects are left to heal by secondary intention. Immediately after extirpation, approximately 0.5 to 1.0 ml of triamcinolone 40 mg/ml is injected subcutaneously at the time of surgery; it is then repeated every 2 weeks for a total of four injections, and then monthly for six additional injections. Sutures are removed on postoperative day 5. Patients initiate compression earring therapy daily once the posterior defect has demonstrated granulation.

## Results

To date, all 6 patients have completed the above surgical regimen and adjunctive modalities. Follow-up ranged from 4 to 6 years. All patients tolerated the procedure well, without complication. We have had no recurrences to date, and favorable cosmetic results (figure 3).

## Discussion

Treatment "success" needs to be better defined in terms of recurrence and latency from treatment. Keloids are known to recur after many years, and earlobe keloids have a higher recurrence rate than those at other anatomic sites.[5] It has therefore become generally accepted that keloid treatment control rates should be reported with a minimum 2- to 3-year follow-up, and actuarial statistical reporting of data is preferable.[6,7]

Prospective studies with keloids have been difficult, however, as many patients do not comply with postoperative follow-up visits.[3] In fact, only one randomized trial has been published for earlobe keloids; it suggested that treatment with a combination of surgery and radiotherapy is superior to the combination of surgery and intralesional steroid injection.[8]

Anecdotally, Parlette and Hendrix reported their best results with surgical excision preceded by a series of intralesional steroid injections (triamcinolone, 10 mg/ml every other week for four visits), followed by a similar series beginning 2 weeks after surgery.[9]

There is a reported recurrence rate of 80% for keloids treated with primary surgical excision alone.[10] Various techniques have been used, including core excision,[11] primary excision with local advancement flaps, wide excision with healing by secondary intention, and primary closure.

Early reports suggested that laser excision might have fewer recurrences. However, recent studies using $CO_2$ lasers, neodynium:YAG lasers, and argon lasers report a recurrence rate of 37.5 to 100%.[12,13]

Proponents of the $CO_2$ laser excision technique claim that the intrinsic properties of laser surgery, which slow fibroblast proliferation, may be responsible for delaying or preventing the recurrence of keloids. The $CO_2$ laser emits an invisible beam of light in the far-infrared spectrum at a wavelength of 10,600 nm. When the beam is defocused and a beam diameter of 2 mm occurs, soft tissue is vaporized, whereas in the focused mode, with a 0.1 to 0.2-mm beam, a "light scalpel" results, with a potential for bloodless excisional surgery. Ultrastructural studies have shown that the focused $CO_2$ laser produces precise destruction, since thermal necrosis is only 30 to 50 µm deep; thus, the potential for collateral thermal injury and subsequent keloid formation is minimized.[13]

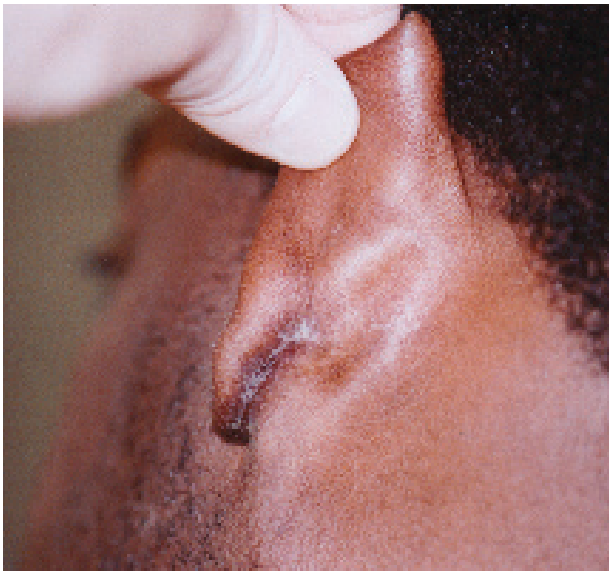

Figure 3. Photo shows the 18-month postoperative results for the patient in figures 1 and 2.

Kantor et al reported good results with the $CO_2$ laser excision of earlobe keloids in 16 patients during a postoperative follow-up interval ranging from 2 to 40 months.[13] However, hypertrophic scarring occurred in 4 of 16 patients (25%) in their study. Some authors suggest that these hypertrophic scars may, in fact, be early recurrent keloids.[14] Furthermore, a prospective study by Stern and Lucente[12] reported a 70% recurrence rate of earlobe keloids after $CO_2$ laser excision; however, the authors acknowledged that none of their patients was white (whites generally have a lower rate of collagen synthesis than blacks), and their study population was fairly noncompliant with postoperative adjunctive triamcinolone injections.

The combination of surgical excision, either cold steel or laser excision, followed by postoperative intralesional steroid therapy, has diminished the reported recurrence rate to less than 50%.[15] Therefore, although intralesional corticosteroids do not eradicate keloids, they can diminish pruritis and pain and can soften the lesions. They are believed to act by inhibiting fibroblast growth and decreasing alpha-2-macroglobulin levels, leading to collagen degradation. Various corticosteroid preparations have been used (from hydrocortisone to dexamethasone to triamcinolone), with fairly equal efficacy.[3]

Radiotherapy has also been used as an adjunctive treatment to surgical excision for keloids. Radiotherapy acts by destroying the proliferating fibroblast and neovascular buds, resulting in decreased collagen production. The timing of radiation administration (within 1 week after excision) and total dose given (700 to 1,500 rad) appear to be important variables in reducing keloid recurrence.[16] With adjuvant postoperative radiation therapy, control rates are 70 to 90%.[17]

In a randomized, prospective trial of earlobe keloids, Sclafani et al demonstrated that surgery followed by radiation showed a trend toward lower keloid recurrence rates (12.5%) than did surgery with postoperative steroid injections (33%).[8] Although no statistically significant difference was found between the two groups, the authors believed that postoperative radiation therapy was more effective than intralesional steroid injection as an adjunct to surgical extirpation, especially in the noncompliant patient.

Many physicians are reluctant to treat a benign disease such as keloids with radiotherapy, citing the potential risk of radiation-induced malignancy. However, several studies with long-term follow-up have not reported any patients with postradiation carcinogenesis.[18-20]

Various other adjunctive modalities have also been studied, such as silicone gel sheeting, topical therapies, systemic therapies, and compression therapy. Most of these are anecdotal reports.[21] Silicone gel sheeting has been shown to decrease pruritis and pain of scars while increasing scar pliability, theoretically by maintaining scar hydration and ultimately decreasing proinflammatory cytokine proliferation and collagen deposition; scar elevation and pigmentation usually remain unchanged, however. With this modality, silicone gel sheeting is applied for at least 12 hours daily. One study showed that 79% of revised keloid scars treated with silicone gel sheeting had had no recurrence by 6 months postsurgery.[21]

Compression therapy is another adjunctive therapy for earlobe keloid regression. It is believed that constant pressure greater than capillary pressure (24 mm Hg) decreases soft-tissue cellularity, increases interstitial space, and causes collagen bundles to be more widely dispersed.

Cryotherapy has also been reported as an adjunct to enhance the efficacy of the intralesional corticosteroids after surgical excision of keloids. This technique uses a refrigerant to induce cell and microcirculatory damage, leading to tissue necrosis and sloughing. It appears to improve retention of the corticosteroid in the keloid when performed immediately before injection, in addition to improving patient tolerance and overall keloid regression.

Regardless of the excisional technique employed, proper surgical technique during keloid excision is imperative and may reduce keloid recurrence. Collateral trauma to the wound bed must be minimized. Instruments that do not crush the tissue should be used. Tension-free closures are essential, and absorbable sutures should be avoided. Furthermore, although most surgeons recommend removing every vestige of the keloid, including the core in a dumbbell-shaped keloid, some believe that leaving a rim of keloid intact may reduce subsequent tissue response.[22]

In conclusion, our approach to treating earlobe keloids uses a combination of three treatment modalities: laser

surgical excision, steroid injection, and mechanical compression therapy. This technique targets the formation of the earlobe keloid at numerous sites in the proposed theoretic pathogenesis by minimizing collateral tissue trauma and targeting local cutaneous immune/inflammatory responses, as well as targeting collagen deposition in an organized fashion with mechanical pressure therapy. These three treatment modalities allow for synergism of this multimodality therapy.

Although our population size is quite small and has a follow-up latency of less than 6 years, we feel our combination of techniques for treating earlobe keloids demonstrates a lower recurrence rate than that previously reported in the literature, with cosmetically favorable results and limited side effects or complications.

We realize that keloids can recur after many years, and that most plastic surgeons are now reporting keloid treatment control rates after a follow-up of 3 years minimum. The transient nature of our patient population limits extensive, long-term follow-up; however, our series to date certainly conforms to this standard.

Long-term follow-up and prospective, controlled studies are necessary to adequately compare the efficacy of our treatment approach with others for this difficult-to-treat clinical entity.

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