Case Letter

Hypertrophic Scar Treatment With Intralesional Triamcinolone Acetonide and Pulsed Dye Laser Results in Necrosis

To the Editor:

Intralesional corticosteroids and pulsed dye laser (PDL) (585–595 nm), either as monotherapy or combination therapy, are commonly used to treat hypertrophic scars.¹ We describe an unusual adverse effect of protracted necrosis following combination therapy with intralesional triamcinolone acetonide and PDL for hypertrophic scar revision.

A 29-year-old healthy man presented after sustaining a contaminated crush trauma to the medial aspect of the left foot (Figure, A), requiring staged reconstruction with split-thickness skin grafting. The site healed with a hypertrophic scar (Figure, B), and treatment was pursued 18 months later with a community dermatologist with laser experience. Intralesional triamcinolone acetonide from a new vial diluted with 2% lidocaine was injected into the hypertrophic portions of the scar (20 mg/mL; 1.5 cc injected). The scar was then treated with a PDL at settings of 595 nm, 7-mm spot size, 10 J/cm², pulse duration of 1.5 milliseconds, 30-millisecond spray with a 20-millisecond delay of cryogen spray, and doublestacked pulses with a 2- to 3-second delay between pulses. The patient reported that purpura was present in treated areas immediately after treatment.

At 6 days posttreatment, the first evidence of possible necrosis with tissue depression in mid posterior portions of the scar appeared (Figure, C). At day 14, deep ulcerations became evident (Figure, D), and by day 21, an exudative, yellow, fibrinopurulent membrane appeared (Figure, E). By 7 weeks posttreatment, ulceration size was only slightly reduced (Figure, F).

The necrosis was treated with debridement and local wound care using collagen matrix dressings (protease modulating matrix). Seven months after attempted scar revision, a deep 4-mm ulcer with a narrow tunnel-like connection to the skin surface remained anteriorly, disconnected from earlier mid posterior ulcerations, which was indicative of delayed onset of necrosis (Figure, G).

Intralesional corticosteroids have been associated with several side effects including pain, dermal atrophy, infection, pigmentation changes, and telangiectases.² Pulsed dye laser therapy is generally described as safe with mild and transient side effects including purpura (resolves in 7–10 days), edema (diminishes in 48 hours), hyperpigmentation, and pain.³ More serious complications such as ulceration and scarring were initially described with PDL for purely vascular cutaneous lesions,⁴ and reports of necrosis and scarring have followed with PDL treatment utilizing higher fluences, shorter pulse durations, and stacking of pulses.⁵

Combination therapy for scar revision with intralesional triamcinolone acetonide and PDL reduces the corticosteroid dose and has been shown to be more efficacious than either modality alone with minimal to no complications.⁶ In our case, we believe the therapeutic combination resulted in several effects including bulk heating, vascular compromise, and inhibition of wound healing that together contributed to protracted necrosis. Triamcinolone acetonide was injected first; the corticosteroid fluid bolus likely conducted heat energy from the PDL spot leading to tissue damage, which corresponded clinically to the deep, delayed-onset necrosis. The PDL's destructive effect on the vasculature after stacked pulses likely led to the initial necrosis, with wound healing time potentially extended by the anti-inflammatory effect of the corticosteroids. The location on the lower extremity, an area with limited vasculature and subcutaneous tissue, may have further contributed to impaired wound healing. Finally, the underlying scar tissue was by nature devoid of adnexal structures to expedite wound healing. Although the combination of intralesional corticosteroids and PDL is widely used and considered safe, we suggest that dermatologists carefully choose PDL parameters and pulse delivery methods when utilizing these modalities for scar revision.

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