Successful treatment of corticosteroid-induced cutaneous atrophy and dyspigmentation with intralesional saline in the setting of keloids

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INTRODUCTION

Intralesional corticosteroids are used to treat various dermatologic conditions through direct delivery of medication to the target tissue. Although intralesional corticosteroids are generally well tolerated, their side effects include atrophy, dyspigmentation, and, infrequently, infection, hemorrhage, and hypersensitivity.[1-3] Corticosteroid injection site atrophy and dyspigmentation due to dermatologic use have been estimated to have an incidence of 0.5%.[1-3] Dyspigmentation is noted more prominently in patients with skin of color and generally resolves within weeks to months.[2] Cutaneous atrophy usually begins within 2-3 months of an injection and resolves in 1-2 years, although it has been reported to persist beyond 5 years.[1-3] Previous studies have demonstrated improvement in steroid-induced lipoatrophy of the face, buttocks, and extremities following intralesional saline injections.[1,4-7] We present a case of severe steroid-induced hypopigmentation and cutaneous atrophy due to intralesional triamcinolone treatment of a supraumbilical keloid treated successfully with serial intralesional saline injections.

CASE REPORT

A 24-year-old woman with Fitzpatrick skin type V presented with a linear keloid scar on the abdomen following a piercing of the umbilicus 8 years prior (Fig 1). The keloid was treated with 0.5 and 0.7 mL of intralesional triamcinolone at 40 mg/mL for a total of 2 injections, with 2 months between the injections. The patient experienced mild discomfort but no atrophy or dyspigmentation after her first intralesional triamcinolone injection. Seven months following the second injection, the patient presented with progressively worsening pigmentary changes, sensitivity, and increased skin fragility, with bleeding at the site despite no additional interventions by her medical team since her last injection. The symptoms began 2 months after her last triamcinolone injection, with gradual progression since onset. Physical examination demonstrated significant epidermal atrophy, lipoatrophy, hypopigmentation, and overlying telangiectasias of the supraumbilical region (Fig 2). Focal areas demonstrated white dermal papules corresponding to steroid crystal aggregates. After a discussion of management options, the decision was made to proceed with intralesional saline injections. Intradermal and subcutaneous injections with bacteriostatic 0.9% sodium were performed at the location

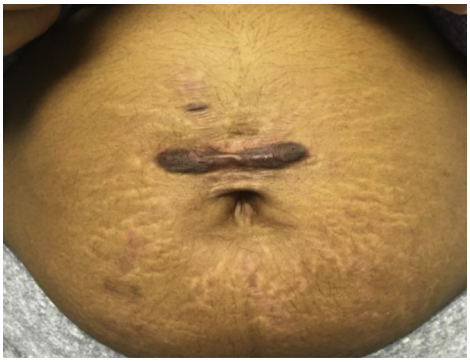

Fig 1. The image shows the initial keloid presentation.

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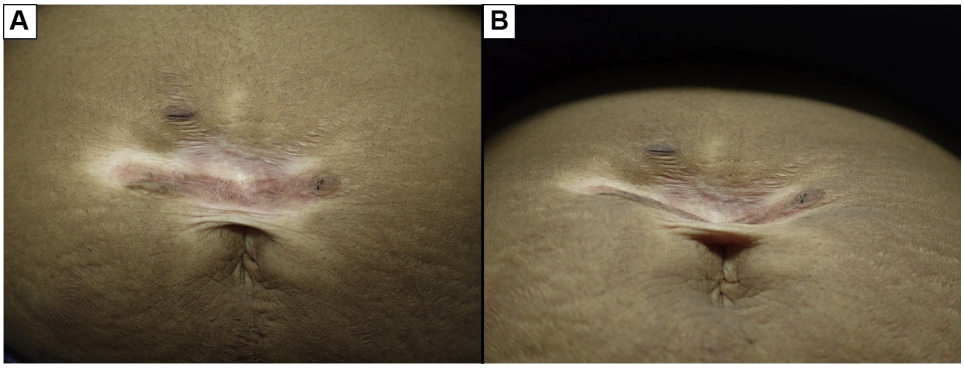

Fig 2. The image depicts cutaneous atrophy and hypopigmentation following an intralesional triamcinolone injection.

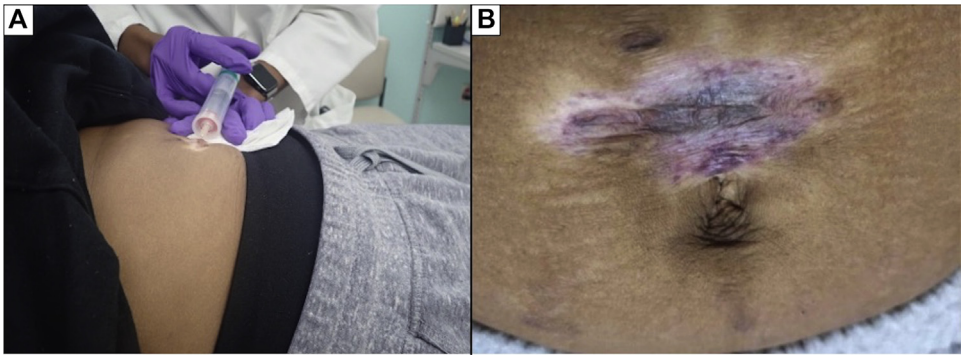

Fig 3. The image shows the postintralesional saline cutaneous findings. **A**, Saline was injected until the skin was no longer depressed and relatively taut. **B**, Ecchymoses between the intralesional saline injections.

of the keloidal scar and surrounding hypopigmented patch until the skin was no longer depressed and relatively taut (Fig 3, *A*). The injections were administered every 2-4 weeks for a total of 6 treatments. Between the injections, the patient reported significant bruising and discomfort (Fig 3, *B*), which subsequently resolved. Following treatment completion, she noted marked improvement in hypopigmentation and atrophy. At a 6-month follow-up, atrophy and pigmentary alterations had completely resolved (Fig 4).

DISCUSSION

Although intralesional corticosteroids are frequently well tolerated and effective in treating various dermatologic conditions, there is risk of potential side effects.[1-3] The appearance of cutaneous atrophy and dyspigmentation can cause significant patient distress, especially in patients with skin of color, in whom hypopigmentation can be more noticeable, given the difference between a natural phototype and hypopigmented lesions. Their clinical appearance, severity, and time course depend on corticosteroid solubility, concentration, dose, injection depth, and the location of injection site.[1-3] Potential pigmentary changes at the site of the corticosteroid injection are more apparent in patients of skin of color and include hypopigmentation or hyperpigmentation.[2,3,8,9] The pigmentary alterations tend to self-resolve over a course of weeks to months,[2,8,9] but we hypothesized that the prolonged and progressive course of hypopigmentation in the patient presented here might have been secondary to the persistent presence of crystalline steroid deposits at the injection sites. The underlying mechanism of corticosteroid-induced dyspigmentation remains unknown.[8] Histologic examination of hypopigmented areas has demonstrated intact melanocytes along the dermal-epidermal junction, with decreased melanin pigment present; consequently, it has been proposed that corticosteroids affect melanocyte function or melanin production.[2,8,9] The potential for dyspigmentation appears to depend on the location of injection site as well as particle size, aggregation propensity, and the density of the steroid.[2,9]

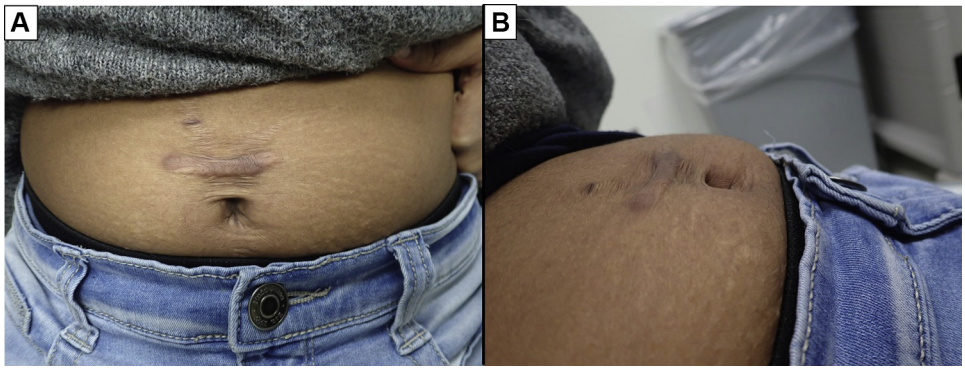

Fig 4. The image shows the abdomen 6 months after the completion of serial intralesional saline injections. Examination demonstrated persistent resolution of cutaneous atrophy and dyspigmentation but with early keloid recurrence.

Although the mechanism of atrophy following a steroid injection is not completely understood, it has been hypothesized to involve decreased fibroblasts, reduced glycosaminoglycan production, and collagen degeneration.[1-3,6] Histologic findings of cutaneous atrophy are variable and can be characterized by epidermal atrophy, collagen homogenization, decreased elastin, degenerative changes of adnexa, including pilosebaceous structures and eccrine glands, and involution of subcutaneous fat lobules.[1-3] In addition, the presence of granular basophilic material in the dermis, consisting of ground substance with polarizable corticosteroid crystals, is commonly observed.[1-3] Cutaneous atrophy resolution, ranging from months to a few years, parallels the disappearance of crystalline deposits from the affected tissue.[1-3]

Numerous methods have been proposed for treating cutaneous atrophy, including autologous fat injection, fat grafting, hyaluronic acid filler, and intralesional saline. Bacteriostatic saline is a relatively safe, nonallergenic solvent that is comparably inexpensive and readily available.[5] Intralesional saline has been hypothesized to resuspend crystalline steroid deposits, allowing them to be presented as foreign particles to macrophages, which then remove them from the site.[7,10] Studies have demonstrated clinical improvement in steroid-induced cutaneous atrophy of the face, extremities, and trunk.[1,4-7] Injection depth, needle bore size, saline volume, injection intervals (range, 1-4 weeks), and the total number of injections (range, 1-9 sessions) vary widely, and there is currently no accepted standard.[1,4-7]

Shiffman[7] demonstrated resolution of steroid-induced atrophy, secondary to treatment of hypertrophic scarring of the breasts, 1 month following intralesional saline administration.[7] However, to date, intralesional saline for the treatment of steroid-induced atrophy secondary to keloid treatment and its use on the abdomen has not been reported; furthermore, its effect on dyspigmentation has not been highlighted. Our case highlights the safety, tolerance, and efficacy of the use of intralesional saline for the treatment of corticosteroid-induced epidermal atrophy, lipoatrophy, and dyspigmentation. Further studies could evaluate the ideal injection technique, volume of saline, frequency of injections, and total treatments required to effectively reverse cutaneous atrophy and dyspigmentation.

Conflicts of interest

None disclosed.

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