OPEN

PRS GLOBAL OPEN

CASE REPORT
Reconstructive

Dexamethasone Mesotherapy: An Alternative for Keloid Treatment in Hispanic Skin

Jenny Carvajal, MD*
Melissa Carvajal, MD†

Summary: The incidence of keloids in individuals with skin of color is as high as 16%. Intralesional steroid injection is recommended as a first-line treatment, even though the outcomes are often suboptimal. Histologically, the keloid epidermal layer is thicker than in normal skin, and the vascular density is higher in the marginal area at subepidermal level due to the elevated expression of vascular endothelial growth factor. Dexamethasone significantly suppresses this proangiogenic cytokine compared with Triamcinolone. We report the case of a 32-year-old phototype VI man with a 6-month-history of a keloid on the dorsum of his right hand that caused functional and cosmetic morbidity. We performed an intralesional injection of dexamethasone using a mesotherapy technique, that led to significant shrinking and complete recovery of range of motion after two sessions, with no regrowth at the 1-year follow-up. Mesotherapy is a safe and easy technique used in cosmetic medicine, which allows for a slower diffusion of dexamethasone and prolongs its pharmacological action, reducing the risk of local side effects. This technique has the potential to be standardized, but its main drawback is the need for proper sedation. Randomized clinical trials are required to further evaluate the clinical efficacy of dexamethasone mesotherapy. *(Plast Reconstr Surg Glob Open 2024; 12:e5612; doi: 10.1097/GOX.0000000000005612; Published online 13 February 2024.)*

Ethnicity has a significant influence on keloid formation, as individuals with skin of color develop keloids 15 times more frequently, particularly, those of African, Asian, and Hispanic descent,[1] with an incidence as high as 16%.[2]

Cryotherapy, interferon, and verapamil have been described for the treatment of keloids but have inconsistent results in skin of color. The same happens with lasers, silicone gel, 5-fluorouracil, radiotherapy, bleomycin, imiquimod, photodynamic therapy, electrical stimulation, intralesional steroid injections, and surgery.[1] Nowadays, intralesional steroid injection is recommended as a first-line treatment for keloids,[3] even though the outcomes are often suboptimal.

Teng et al have shown that the mean thickness of the keloid epidermal layer is 391.4 ± 2.3 µm (0.4 mm), which is significantly thicker compared with the normal skin.[4] Histologically, keloids present hyalinized collagen bundles, nodular fibroplasia, and an accumulation of myofibroblasts. The central area expresses more HIF-1a (the major transcription factor in response to hypoxia). The vascular density in the marginal regions is higher because angiogenesis is presumed to be activated, as keloids grow outside the original lesion, which explains the elevated expression of the proangiogenic cytokine vascular endothelial growth factor (VEGF) in this area.[4]

Although keloids are caused by chronic inflammation in the reticular dermis[5] and the original structure is lost, studies have identified the superficial, middle, and deep layers in their dermis, which display different features.[6] The superficial dermis presents active fibroblasts, lymphocytes, and the "keloid subepidermal vascular network" at a depth of 150–400 µm. The randomly distributed vessels run parallel to the epidermis with several vertical branches, which increases blood perfusion across the subepidermal area[4,6]

Despite clinical, histological, and in vitro observations, the mechanisms underlying steroid therapy remain unclear; however, analytical results have demonstrated that dexamethasone significantly suppresses VEGF expression compared with triamcinolone and induces its regression in vivo.[7]

With the improved understanding of keloids and their growth, we focused on developing an alternative technique in which we could effectively block VEGF.

*From *Private Practice, Carvajal MD, Plastic Surgery, Medellín, Colombia; and †General Practitioner,Carvajal MD, Plastic Surgery, Medellín, Colombia.*

Received for publication October 24, 2023; accepted December 28, 2023.

DOI: 10.1097/GOX.0000000000005612

Disclosure statements are at the end of this article, following the correspondence information.

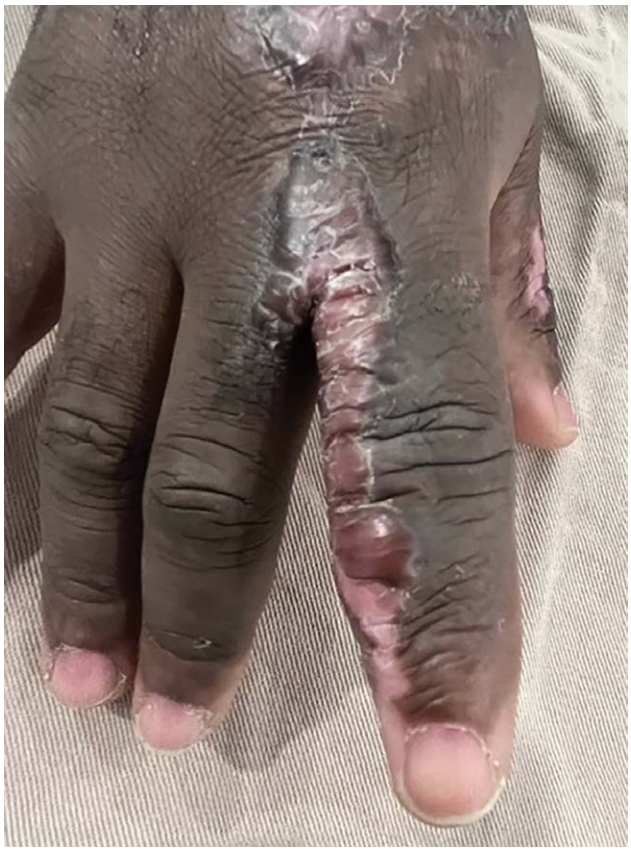

Fig. 1. A 32-year-old phototype VI man with a 6-month history of a friction burn involving the dorsum of his right hand, the ulnar side of his second finger, and the second interdigital fold, who developed a keloid of approximately 8 cm × 1.5 cm in these areas, limiting the abduction and the flexion of his index finger.

Therefore, we chose to inject dexamethasone using a mesotherapy technique to stop expansion and achieve regression.

CASE REPORT

A 32-year-old phototype VI man with a 6-month-history of a friction burn involving the dorsum of his right hand, the ulnar side of his second finger, and the second interdigital fold was referred to us due to an unaesthetic rigid scar in these areas, limiting the abduction and the flexion of his index finger. Clinical evaluation showed a keloid of approximately 8 cm × 1.5 cm size with irregular margins (Figure 1). We decided on intralesional infiltration with steroids, using a mesotherapy technique.

TECHNIQUE

Sedation was performed with an intravenous infusion of propofol, fentanyl, midazolam, and ketamine. Before surgical antisepsis, we demarcated the keloid perimeter and designed a 0.5 cm × 0.5 cm grid on the surface. We used a 1 mL insulin syringe with a 30 G, 13-mm needle to prepare a mix of 1 mL of bupivacaine 0.5% solution

Fig. 2. Marking the keloid perimeter and designing a 0.5 cm × 0.5 cm grid on the keloid surface to puncture each vertex at a 15-degree angle and a depth of 2–3 mm. A bupivacaine 0.5% with 2 mL of Duo-decadron 16 mg + 4 mg/2 mL injectable solution was used.

for every 2 mL of Duo-decadron 16 mg + 4 mg/2 mL solution, which contains dexamethasone acetate and dexamethasone sodium phosphate. We punctured each vertex at a 15-degree angle and a depth of 2–3 mm to inject 0.05 mL, creating a small papule. We performed a total of 64 microinjections, which required 3.2 mL of the mixture (1.4 mg dexamethasone acetate + 0.35 mg dexamethasone sodium phosphate per cm^2; Fig. 2).

RESULTS

There was significant softening of the keloid after a week, with recovery of abduction and flexion of the second finger. It shrunk approximately 50% in the following 4 weeks (Fig. 3). We decided on a second steroid infiltration using this technique after 2 months. The keloid shrunk 80% by the fourth week, with complete recovery of range of motion. No additional interventions were prescribed. Patient was happy and did not desire additional procedures. The 1-year follow-up shows no regrowth (Fig. 4).

DISCUSSION

Keloids can produce cosmetic and functional morbidity, which is challenging. Our patient was not able to perform a clamp with his dominant hand, affecting his daily activities.

Fig. 3. After 4 weeks, shrinkage of 50% and significant softening of the keloid was observed with recovery of abduction and flexion of the second finger.

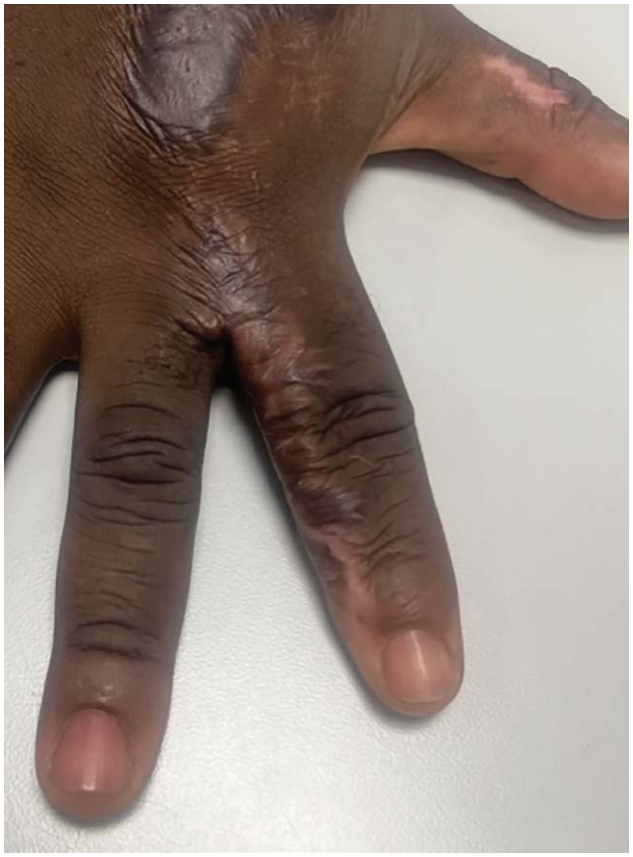

Fig. 4. The keloid shrunk 80% after the second dexamethasone mesotherapy injection with complete recovery of range of motion. The 1-year follow-up shows no regrowth.

Management of keloids in skin of color is difficult, and there is no level I evidence. Surgical excision could have resulted in a worse functional and aesthetic outcome. We chose intralesional corticosteroid administration even though 63% of patients develop side effects, including skin necrosis and ulceration, dermal and subcutaneous fat atrophy, and telangiectasia.[8] The risk increases when surrounding tissue is injected.[8]

Currently, there is not a standardized protocol for this procedure,[2] and the technique is highly operator dependent. There is great variability in the number of treatment sessions, ranging from one to eight, with an average of four. The treatment intervals go from weekly to monthly, with 4 weeks being the most common. The syringe and needle size also changes;[9] the plane of injection is not specified, or it is vaguely described. Triamcinolone is used 97% of the time, but the volume and concentration are highly variable, with maximum doses of 80 mg per session.[2]

As we mentioned before, dexamethasone induces keloid regression via interaction with glucocorticoid receptors and suppresses endogenous VEGF and fibroblast proliferation,[7] but it must be administered to the most angiogenic parts of the keloid: the marginal areas at subepidermal level. This was our motivation to try mesotherapy (local intradermal therapy).

Mesotherapy is a safe and easy technique used in cosmetic medicine, which uses a 13-mm (30–32 guage) needle appropriately inclined to perform multiple microdermal deposits of a drug, in our case dexamethasone. Fragmenting the dose modifies the normal kinetics of absorption, allowing for a slower diffusion and a longer pharmacological action.[10] This could minimize the risk of side effects.

We believe that this technique proved to be successful and has the potential to be standardized. Its main drawback is that it requires adequate sedation to accurately deposit dexamethasone in the superficial dermal plane, which could also be painful.

CONCLUSIONS

We propose the use of dexamethasone mesotherapy technique as a safe alternative to induce regression of keloids in patients with skin of color. Randomized controlled trials are needed to further evaluate the clinical efficacy of dexamethasone mesotherapy.

Jenny Carvajal, MD
Carvajal MD Plastic Surgery
Medellín, Antioquia
Colombia
E-mail: yennycar@hotmail.com

DISCLOSURES

The authors have no financial interest to declare in relation to the content of this article.

REFERENCES

1. Ud-Din S, Bayat A. Strategic management of keloid disease in ethnic skin: a structured approach supported by the emerging literature. *Br J Dermatol.* 2013;169:71–81.
2. Yin Q, Louter JMI, Niessen FB, et al. Intralesional corticosteroid administration in the treatment of keloids: a scoping review on injection methods. *Dermatology.* 2023;239:462–477.
3. Naik PP. Novel targets and therapies for keloid. *Clin Exp Dermatol.* 2022;47:507–515.
4. Touchi R, Ueda K, Kurokawa N, et al. Central regions of keloids are severely ischaemic. *J Plast Reconstr Aesthet Surg.* 2016;69:e35–e41.
5. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids: a 2020 update of the algorithms published 10 years ago. *Plast Reconstr Surg.* 2022;149:79e–94e.
6. Teng Y, Hao Y, Liu H, et al. Histology and vascular architecture study of keloid tissue to outline the possible terminology of keloid skin flaps. *Aesthetic Plast Surg.* 2022;46:985–994.
7. Wu WS, Wang FS, Yang KD, et al. Dexamethasone induction of keloid regression through effective suppression of VEGF expression and keloid fibroblast proliferation. *J Invest Dermatol.* 2006;126:1264–1271.
8. McGoldrick RB, Theodorakopoulou E, Azzopardi EA, et al. Lasers and ancillary treatments for scar management Part 2: keloid, hypertrophic, pigmented and acne scars. *Scars Burn Heal.* 2017;3:2059513116689805.
9. Yin Q, Niessen FB, Gibbs S, et al. Intralesional corticosteroid administration in the treatment of keloids: a survey among Dutch dermatologists and plastic surgeons. *J Dermatolog Treat.* 2023;34:2159308.
10. Mammucari M, Maggiori E, Russo D, et al. Mesotherapy: from historical notes to scientific evidence and future prospects. *Scientific World J.* 2020;2020:3542848.