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Stellate Hypopigmentation in a Pediatric Patient After Treatment with Intralesionally-Injected Corticosteroid

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 3-year-old
Final Diagnosis: Steroid-induced hypopigmentation
Symptoms: Depigmentation of skin
Medication: —
Clinical Procedure: —
Specialty: Dermatology • Pediatrics and Neonatology • Pharmacology and Pharmacy

Objective: Unusual clinical course

Background: Several factors contribute to keloids in post-operative patients, including skin mechanics, genetics, and inflammatory processes. One of the most widely used treatment modalities for keloidal scars involves the intralesional injection of corticosteroids, such as triamcinolone acetonide (TAC). TAC is a first-line treatment option for keloids due to its proven efficacy and effectiveness in reducing collagen synthesis, glycosaminoglycan synthesis, inflammatory processes, and proliferation of fibroblasts. Some common adverse effects of intralesional corticosteroid injection include localized hypopigmentation, depigmentation, skin atrophy, and lipoatrophy.

Case Report: In this report, we describe the case of a 3-year-old African American male patient who presented for dermatologic evaluation of a diffused stellate hypopigmentation attributed to intralesional corticosteroid injection following a keloid removal. Specifically, we summarize this case's clinical features, diagnosis, and outcomes.

Conclusions: The case illustrates self-limiting hypopigmentation that repigmented successfully without clinical intervention. Although previous reports of corticosteroid injections' adverse effects resulting in hypopigmentation have been published, this condition is uncommon or poorly reported in pediatric patients. This report aims to contribute to our understanding of the effects of administering corticosteroids in pediatric patients by virtue of diversifying the cases reported in the currently available literature.

Keywords: Adrenal Cortex Hormones • Hypopigmentation • Pigmentation Disorders

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/935898

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Background

Keloids and hypertrophic scars are known complications of any intrusion or injury to the skin. These injuries to the skin include surgical incisions, which generally cause traumatic damage to the skin layers, including the reticular dermis, and typically lead to scarification due to immune system activity in the healing process [1]. These scars, including keloidal scars, can be effectively treated with intralesional corticosteroid injections [1,2]. Corticosteroids such as triamcinolone acetonide (TAC) have proven efficacy and efficiency and are commonly used as a first-line treatment option in treating keloids [3]. However, the administration of intralesional corticosteroid injection can confer localized adverse effects, including hypopigmentation and atrophy [1,4]. TAC at a 40 mg/ml dosage is associated with a considerable increase in the risk of dermal atrophy [5]. Improving our understanding of the factors contributing to these adverse effects can be instrumental in improving clinical guidelines surrounding the use of injectable corticosteroids, especially in the under-studied pediatric population.

When injected into the skin, TAC's long-term absorption mechanism minimizes systemic absorption and maximizes localized effect [3,5]. Response rates from injected TAC vary from 50% to 100%, with a keloid recurrence rate of 9% to 50%, depending on the location of the keloid on the body [5]. In some cases, TAC injection treatment is supplemented with topical adjuncts to improve clinical outcomes [3]. These injections and topical medications can cause hypopigmentation, skin atrophy, subcutaneous fat atrophy, telangiectasias, rebound effects, alopecia, infection, ulceration, and localized dystrophic calcification [1]. It is understood that hypopigmentation and depigmentation processes involve the functional alteration of pigment-producing melanocytes; however, there is no clear understanding of the underlying processes, particularly in maturing and developing pediatric skin. This case report documenting the clinical course of a pediatric patient who developed diffused hypopigmentation following treatment with intralesional corticosteroid injection aims to contribute to our understanding of this condition.

Case Report

Our patient was a 3-year-old African American male with Fitzpatrick type V skin, a medical history of trisomy 21, and no family history of keloids or vitiligo. The patient developed a peristomal keloid after gastrostomy tube placement 7 months prior to this clinic visit (**Figure 1**). The keloid treatment consisted of surgical excision and an intralesional administration of a single dose of 0.4 ml of triamcinolone (40 mg/ml). This treatment successfully repressed the recurrence of the keloid;

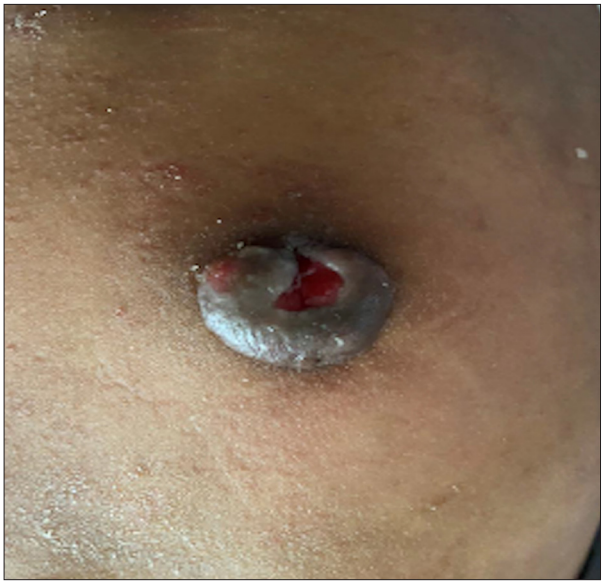

Figure 1. Keloid developed from the incision after gastrostomy tube placement in this Fitzpatrick type V pediatric patient.

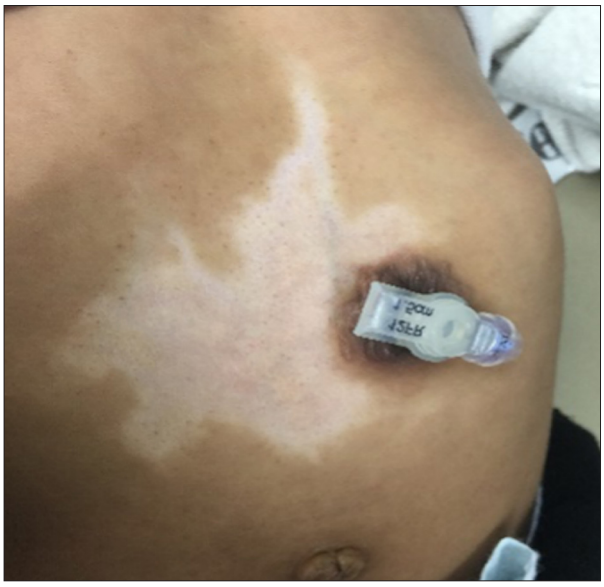

Figure 2. Initial presentation of leucoderma with mild atrophy. Well-demarcated 7.5×7 cm hypopigmented minimally atrophic patch located on the periumbilical skin noted at 5 months following surgery and intralesional triamcinolone treatment.

however, about 3 months after treatment, the family sought a dermatology referral for an asymmetrical but well-demarcated diffused hypopigmentation adjacent to the treatment site on the periumbilical skin measuring 7.5×7 cm (at greatest vertical borders) (**Figure 2**). Palpation of the center of the affected area suggested mild dermal atrophy. Given that the hypopigmentation followed the intralesional corticosteroid injection,

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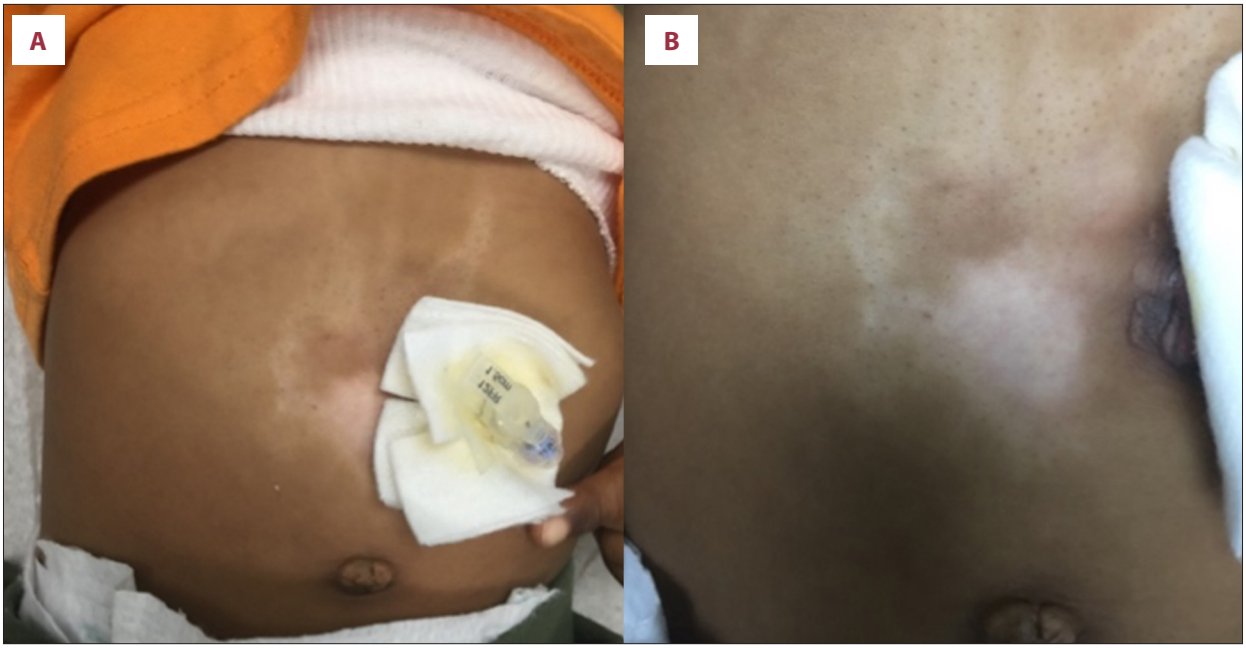

Figure 3. (**A, B**) Spontaneous improvement of the lipoatrophy and hypopigmentation on the periumbilical skin at the follow-up visit (11 months following intralesional triamcinolone treatment).

the most likely diagnosis pointed to a steroid-induced adverse effect instead of other conditions on the differential, ie, vitiligo. The presence of dermal atrophy also fit best with steroid-induced process. Considering the self-limiting nature of this condition, observation without intervention was the best course of action. The patient's caregiver was educated on the mechanism and adverse effects of long-term steroids treatment (including hypopigmentation, skin atrophy, and lipoatrophy) and was advised to continue monitoring changes at the affected site. In the 5-month follow-up of this case, the affected area had significantly recovered pigmentation (**Figure 3**), with a promising outlook for full recovery. Of note, the dermal atrophy appeared much smaller on the follow-up exam.

Discussion

Hypopigmentation and atrophy are the most commonly occurring local adverse effects from the intralesional injection sites and are postulated to involve the spread of corticosteroid crystals along cutaneous lymphatic vessels [4]. These perilesional/perilymphatic leucodermas diffuse from the treatment site in patterns such as linear radiation and other streaky iterations [2]. The observed hypopigmentation, in this case, has been hypothesized to result from reduced melanocyte function [6] rather than T-cell-mediated melanocyte destruction, as seen in depigmenting conditions like vitiligo [7]. Hypopigmentation and gradual auto-repigmentation of the affected area in our case is consistent with loss of melanocyte function. Accordingly, this report contributes data that can be factored in future analyses of a collection of similar pediatric cases for meaningful insight and recommendation.

Case reports on different preparations of steroids inducing hypopigmentation in the pediatric population are relatively rare, but this could result from underreporting. Nonetheless, the existing examples have documented areas of pigmentary change that do not appear to mimic an intrinsic disease process. An example of this was a case study reporting intradermal steroid administration causing hypopigmentation to spread up to 2 feet from the injection site [8]. Other examples of self-limiting fat atrophy and depigmentation side effects around a triamcinolone injection site have also been reported [9-11]. These limited examples demonstrate that different preparations may have different levels of risk for adverse effects, but most importantly, they point to a scarcity of reported pediatric cases, which may affect clinical guidelines on corticosteroid use. For example, a series of 24 patients with corticosteroid-induced atrophy and depigmentation was recently reported, yet only 1 child (a 4-year-old) was listed [6].

In general, the adverse effects of steroids are self-limiting and resolve in a few months or even years; however, there are also ways to correct them cosmetically. Some corrective measures involve micro-needling, saline and platelet-rich plasma injections, narrowband ultraviolet B, 308-nm laser and 10 600 nm laser phototherapy, and melanocyte-keratinocyte transplantation, and fat grafting are also other options to correct atrophy and hypopigmentation [2,4,8]. The risks and benefits of these treatments, however effective, would have to be weighed carefully in

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a case such as we are reporting due to the involvement on abdominal skin over a more conspicuous location such as the face.

Conclusions

Based on the available information, the benefits from the effectiveness of intralesional corticosteroid injections outweigh cosmetic adverse effects like hypopigmentation and lipoatrophy. The use of less potent and shorter-acting steroid preparations may help prevent some of these adverse effects, but these approaches and appropriate concentrations need to be studied further in the pediatric population. Analysis and inclusion of varied pediatric patient profiles would help us better understand the interaction of corticosteroids and factors affecting the development and adaptations of the skin.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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