

Hypopigmentation on the Ear

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A 20-year-old black woman underwent multiple intralesional corticosteroid injections for treatment of a keloid on the superior aspect of the left helix and subsequently presented with a streak of atrophy and hypopigmentation in the postauricular region of unknown duration due to the lesion location.

WHAT'S THE DIAGNOSIS?

- corticosteroid-induced hypopigmentation
- hypopigmented mycosis fungoides
- hypopigmented sarcoidosis
- morphea
- vitiligo

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The authors report no conflict of interest.

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THE DIAGNOSIS:

Corticosteroid-Induced Hypopigmentation

This patient received several intralesional injections of triamcinolone acetonide once monthly for treatment of the keloid scar on the left ear at an outside institution. There was improvement in the size of the keloid over time. On physical examination during the most recent visit there was a prominent streak of hypopigmentation and atrophy near the corticosteroid injection site with extension to the postauricular region. There also was telangiectasia noted within the area of hypopigmentation. Intralesional triamcinolone injections were discontinued and the patient was advised to return for monitoring.

Intra-articular and intralesional corticosteroid injections frequently are used by clinicians. Cutaneous complications associated with these injections include atrophy, pigmentary changes, hypersensitivity reactions, flushing, cellulitis, and necrotizing fasciitis. Tendon rupture also has been reported.¹

There are several case reports in the literature describing hypopigmentation and/or subcutaneous atrophy after intralesional or intra-articular corticosteroid injections. A variety of underlying conditions were treated including alopecia areata, keloids, rheumatoid arthritis, de Quervain tendonitis, and psoriasis.²⁻⁶ The lesions typically are described as linear rays of atrophy and hypopigmentation at or near the injection site, with some cases noting extension along lymph channels and proximal veins.^{4,6} There usually is no associated pruritus or pain.³ This phenomenon can be seen after single or multiple injections.^{4,6}

Extension of hypopigmentation from the site of injection has been postulated to be due to venous or lymphatic uptake.^{2,4-6} The mechanism of hypopigmentation is not known. Biopsy of a previously described case showed intact melanocytes along the dermoepidermal junction.² Biopsy from another case revealed a decrease in melanin staining, which suggests a decrease in number or activity of melanocytes.⁴ It was proposed that hypopigmentation was secondary to loss of melanocyte function instead of loss of melanocytes.² Spontaneous improvement or resolution of the hypopigmentation were noted in some cases

ranging from 1 month to 1 year after initial presentation, but the hypopigmentation also can be persistent.³⁻⁶

Hypopigmented sarcoidosis and hypopigmented mycosis fungoides, both often present on dark-skinned individuals, are included in the differential diagnosis. Hypopigmented sarcoidosis presents with hypopigmented macules or patches, some with central papules, and hypopigmented mycosis fungoides presents with hypopigmented patches or plaques with fine scale and onset often in childhood or adolescence.^{7,8} Morphea can present with an initial inflammatory stage that develops into a sclerotic firm plaque or nodule with hyperpigmentation or hypopigmentation.⁹ Vitiligo usually presents with depigmented macules or patches and depigmented hair within the lesion.¹⁰

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