Linear Hypopigmentation on the Right Arm

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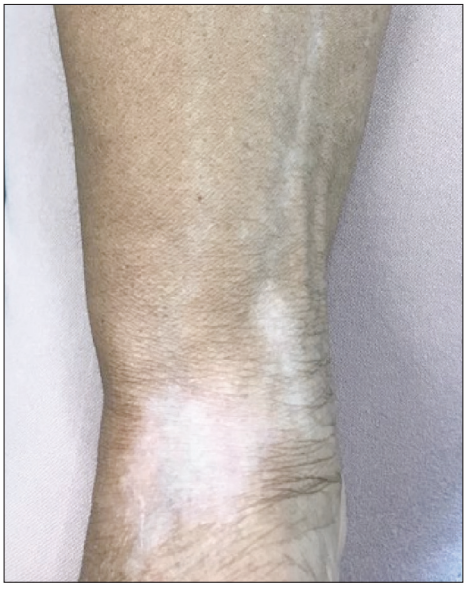

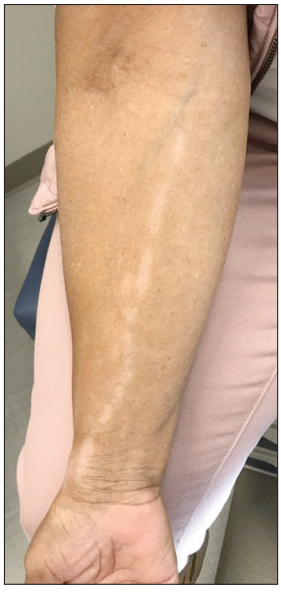

A 73-year-old woman presented to the dermatology clinic with hypopigmentation along the right arm. Her medical history was notable for prior treatment with intralesional triamcinolone injections for De Quervain tenosynovitis. Two months after receiving the steroid injections she noted progressive spreading of an asymptomatic white discoloration originating on the right wrist. Physical examination revealed a hypopigmented atrophic patch on the medial aspect of the right wrist (left) with linear hypopigmented patches extending proximally up the forearm (right).

WHAT'S YOUR **DIAGNOSIS?**

a. chemical leukoderma
b. hypomelanosis secondary to lichen striatus
c. postinflammatory hypopigmentation
d. progressive macular hypomelanosis
e. segmental vitiligo

PLEASE TURN TO PAGE 130 FOR THE DIAGNOSIS

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The authors report no conflict of interest.

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THE **DIAGNOSIS:** Chemical Leukoderma

A clinical diagnosis of chemical leukoderma was made. In our patient, the observed linear hypopigmentation likely resulted from the prior treatment for De Quervain tenosynovitis in which an intralesional corticosteroid entered the lymphatic channel causing a linear distribution of chemical leukoderma. The hypopigmentation self-resolved at 6-month follow-up, and the patient was counseled to continue steroid injections if indicated.

Chemical leukoderma is an acquired depigmenting dermatosis that displays vitiligolike patterning. Detailed personal and family history in addition to complete physical examination are crucial given the inability to distinguish chemical leukoderma from vitiligo on histopathology. A set of clinical criteria proposed by Ghosh and Mukhopadhyay[1] includes the presence of acquired depigmented macules and patches resembling vitiligo, history of repeat exposure to certain chemical substances, hypopigmentation at the site of exposure, and/or confettilike white macules. Three of these 4 clinical findings must be present to establish a diagnosis of chemical leukoderma. The extent of disease involvement may be graded as follows: Stage I is defined as leukoderma only at the site of contact to the offending agent. Stage II involvement is characterized by local spread beyond the exposure site via the lymphatic system. Stages IIIA and IIIB leukoderma entail hematogenous spread distant to the site of chemical exposure. Although stage IIIA leukoderma is limited to cutaneous involvement, stage IIIB findings are marked by systemic organ involvement. Stage IV disease is defined by the distant spread of hypopigmented macules and patches that continues following 1 year of strict avoidance of the causative agent.[1]

The pathogenesis behind chemical leukoderma is not completely understood. Studies have suggested that individuals with certain genetic susceptibilities are predisposed to developing the condition after being exposed to chemicals with melanocytotoxic properties.[2,3] It has been proposed that the chemicals accelerate pre-existing cellular stress cascades within melanocytes to levels higher than what healthy cells can tolerate. Genetic factors can increase an individual's total melanocytic stress or establish a lower cellular threshold for stress than what the immune system can manage.[4] These influences culminate in an inflammatory response that results in melanocytic destruction and subsequent cutaneous hypopigmentation.

The most well-known offending chemical agents are phenol and catechol derivatives, such as hydroquinone, which is used in topical bleaching agents to treat diseases of hyperpigmentation, including melasma.[2] Potent topical or intralesional corticosteroids also may precipitate chemical leukoderma, most notably in individuals with darker skin tones. Hypomelanosis induced by intralesional steroids frequently occurs weeks to months after administration and commonly is observed in a stellate or linear pattern with an irregular outline.[5] Other offending chemical agents include sulfhydryls, mercurials, arsenic, benzoyl peroxide, azelaic acid, imiquimod, chloroquine, and tyrosine kinase inhibitors.[2,5]

Segmental vitiligo is characterized by unilateral hypopigmentation in a linear or blocklike distribution that does not cross the midline. However, onset of segmental vitiligo classically occurs prior to 30 years of age and frequently is related with early leukotrichia.[6] Additionally, the hypomelanosis associated with segmental vitiligo more often presents as broad bands or patches that occasionally have a blaschkoid distribution and most commonly appear on the face.[5] Lichen striatus is a lichenoid dermatosis that presents as asymptomatic pink or hypopigmented papules that follow the Blaschko lines, often favoring the extremities. Postinflammatory hypopigmentation also may occur as an associated sequela of resolved lichen striatus. Although the disease onset of lichen striatus may occur in adulthood, it typically appears in childhood and is triggered by factors such as trauma, hypersensitivity reactions, viral infections, and medications. Physical injuries such as trauma following surgical procedures also can lead to hypomelanosis; however, our patient denied any relevant surgical history. Progressive macular hypomelanosis is a skin condition presenting as ill-defined, nummular, hypopigmented macules or patches that commonly affects women with darker skin tones with an ethnic background from a tropical location or residing in a tropical environment.[5] Lesions frequently appear on the trunk and rarely progress to the proximal extremities, making it an unlikely diagnosis for our patient.

In most cases of chemical leukoderma, spontaneous repigmentation often occurs within 12 months after the elimination of the offending substance; however, hypopigmented lesions may persist or continue to develop at sites distant from the initial site despite discontinuing the causative agent.[1] Therapies for vitiligo, such as topical corticosteroids, topical immunosuppressants, narrowband UVB phototherapy, and psoralen plus UVA photochemotherapy, may be utilized for chemical leukoderma that does not self-resolve.

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