

Correspondence

Spreading of pre-existing segmental vitiligo after immunotherapy with house dust mite in a patient with atopic dermatitis

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A 10-year-old Korean girl presented with a 3-year history of linearly arranged, hypopigmented patches on her left abdomen, and she was diagnosed with segmental vitiligo (SV) based on Wood light examination and the segmental distribution of the lesion. She also had a 7-year history of atopic dermatitis (AD). She underwent a 2-year course of excimer laser treatment for the SV, but the lesions did not respond well. The AD lesions were stable during the excimer laser treatment, but flared suddenly during treatment without progression of SV. The AD lesions were aggravated

even by conventional treatments for AD such as topical steroid and calcineurin inhibitors. Therefore, immunotherapy using house dust mite allergens was tried, as the patient was highly allergic to house dust mite by Multiple Allergen Simultaneous Test (MAST; Hitachi Chemical Diagnostics, Mountain view, CA, USA) allergy test.

The patient received weekly injections of house dust mite allergen (Tyrosine-s; Allergy Therapeutics Ltd, Worthing, West Sussex, UK). Three months after immunotherapy, the AD lesions had improved, but the hypopigmented patches on the patient's left abdomen started to increase in size along the ipsilateral dermatome (Fig. 1a,b), and new hypopigmented patches developed on the dorsum of her left foot (Fig. 1c,d).

There have been a few previous reports of coexisting vitiligo and AD. A recent study showed a strong association

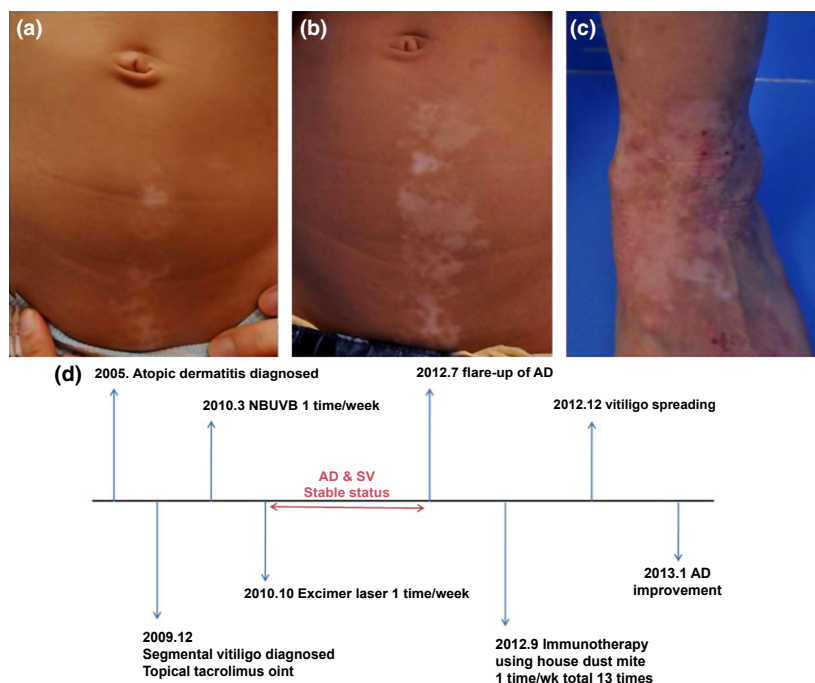


Figure 1 (a) Linearly arranged, hypopigmented patches on the left abdomen before immunotherapy. (b) Hypopigmented patches expanding from pre-existing segmental vitiligo (SV) along the ipsilateral segment after immunotherapy. (c) Coexistence of atopic dermatitis (AD) and newly developed hypopigmented patches on the dorsum of the foot in the same direction as the pre-existing abdominal vitiligo lesions. (d) Clinical course of AD and SV during excimer laser therapy and immunotherapy.

between prepubertal nonsegmental vitiligo (NSV) and AD, and a possible shared mechanism for both.¹ However, the association of AD and SV has not been clearly established.

To our knowledge, this is the first case report of spreading of SV after immunotherapy in a patient with AD. Such spreading is not thought to be associated with an autoimmune process, although SV has recently been reported to share an autoimmune mechanism with NSV.^{2,3} Although SV is usually explained by the neural hypothesis, this case supports a recent hypothesis that SV has a similar immunological mechanism to NSV in its development.³ A CD8+ melanocyte-specific T cell-mediated immune response is known to play a role in SV with associated halo naevi, similar to NSV.⁴ It has been reported that dysfunction of humoral immunity may play a role in the progression of SV, whereas cellular immunity seems to be involved in NSV.⁵

T helper (Th)1/Th17 cellular immunity is thought to be involved in the pathogenesis of vitiligo, whereas AD is regarded as a Th2-skewed allergic skin disease. The mechanism of immunotherapy in AD is to downregulate Th2 response and to upregulate the Th1 pathway through allergen sensitization. Thus, our case shows that immunotherapy in AD could aggravate vitiligo via an immunotherapy-induced Th1 immune response.

The presence of AD lesions over the dorsum of the patient's left foot might indicate development of vitiligo lesions through koebnerization. However, this does not explain the spreading of vitiligo lesions on the left abdomen, which was not accompanied by AD. Furthermore, the AD lesions were bilateral and the vitiligo lesions were unilateral.

In conclusion, our case suggests that immunotherapy for AD treatment could initiate spreading of SV, and that

SV might have an overlapping immunological mechanism with NSV.

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Conflict of interest: the authors declare that they have no conflicts of interest.

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