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VITILIGINOUS LESIONS DURING CONTACT IMMUNOTHERAPY FOR ALOPECIA IN A PATIENT WITH AUTOIMMUNE THYROIDITIS

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Sir,

Squaric acid dibutylester (SADBE) is frequently used for the treatment of alopecia, but sometimes unwanted side effects occur. Herein we report a case which developed vitiliginous lesions induced by topical SADBE application in a patient with autoimmune thyroiditis.

A 60-year-old female visited our department, complaining of diffuse alopecia of the scalp. She was suffering from chronic autoimmune thyroiditis over several years, and taking thyradin (90mg per day). After obtaining written informed consent, topical application of SADBE solution was started. Two months later, a sufficient effect was obtained for hair regrowth; however, when she was treated with 0.05% SADBE, depigmentation appeared and rapidly spread on the scalp. On physical examination, diffuse depigmentation along with gray hairs was found on the scalp, forehead, and nape (Fig. 1). Laboratory examination showed normal liver and renal function, anti-nuclear antibody (1:160, speckled), anti-thyroid antibody (1:100), anti-microsome antibody (1:6400), anti-thyroid peroxidase antibody (139.6 IU/ml, normal <16), but anti-thyroid stimulating hormone receptor antibody, free T3 and T4 levels were normal. A skin biopsy showed decrease of melanocytes and melanin deposition in the epidermis (Fig. 2A). Results of immunohistochemistry revealed a predominant CD8-positive T-cell infiltration (Fig. 2B). SADBE therapy was stopped, and betamethasone butyrate propionate and carpronium chrolide lotion were applied, which showed a dramatical effect within 9 months (Fig. 3).

Adverse effects of topical immunomodulatory therapy include local irritation, blister formation, persistent dermatitis, lymphadenopathy, generalized eczema, and urticarial reaction. Mechanism of contact immunotherapy is suggested to modulate cytokine gene expression balance, and interferon-γ expression was reduced while interleukin-2 (IL-2), IL-8, IL-10 and tumor necrosis factor-α levels were increased in the lesional skin [1]. Further, recent studies demonstrate that Th17 cells are involved in the pathogenesis of contact hypersensitivity. Skin biopsies of hypersensitivity reactions to contact sensitizers demonstrate the presence of CD8+ T-cells, Th1 CD4+ effector cells and regulatory T-cells [2]. T-cells play an important role in the pathogenesis of vitiligo, and IL-10 is supposed to play a role by inhibiting T-cells. Also, active Th17 cells are increased in vitiligo skin [3], and thus those mediators may play a role in the autoimmune mechanisms of vitiligo.

Our case developed vitiliginous lesions not only on the scalp, but the forehead and nape were also involved beyond the application areas. Three months later from the start of immunotherapy, depigmentation was suddenly induced without prior contact dermatitis, which suggests that our case was not a result of contact leucoderma.

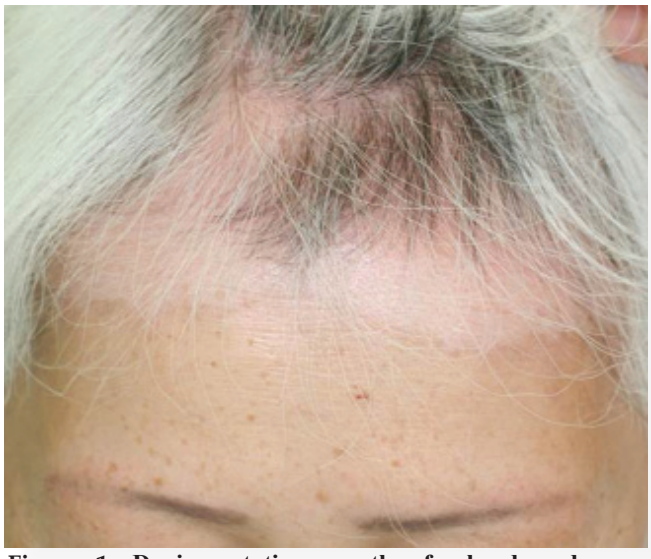

Figure 1. Depigmentation on the forehead and nape developed during contact immunotherapy with SADBE.

However, it is difficult to differentiate contact leukoderma, which arises on the site of application as a consequence of eczematous reaction, or even at the distant areas. Nine months later, depigmentation was recovered by topical corticosteroid ointment. To date, several cases of vitiligo induction during topical immunotherapy have been reported [4-8].

The mechanisms are speculated as a direct cytotoxic effect of SADBE on the melanocytes, or as a result of Köbner phenomenon. Thyroiditis is a representative disorder associated with not only alopecia but also vitiligo. In our case, autoimmune condition may be associated with the development of vitiligo, and SADBE may have a triggering role. Careful attention should be paid when we carry out contact immunotherapies for patients with autoimmune thyroiditis.

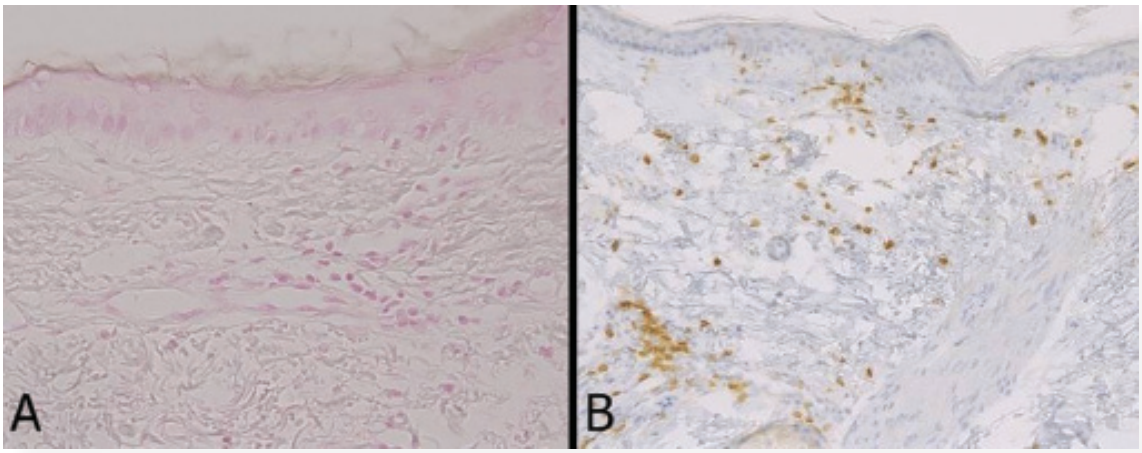

Figure 2. (a) Histology showing decrease of melanocytes in the epidermis (Fontana-Masson stain, ×200) (b) Cellular infiltrates were positive for CD8 (×100).

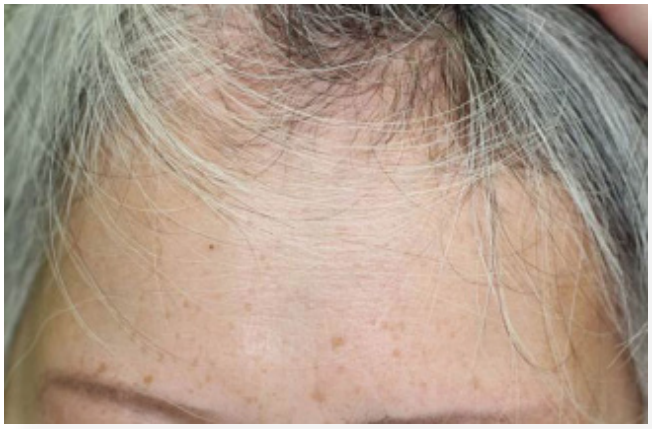

Figure 3. Depigmentation was much improved by topical steroid ointment 9 months later.

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