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Letter to the Editor

Successful treatment of refractory alopecia universalis by persuading a patient not to sleep with her dog

Dear Editor,

Alopecia areata (AA) is a common disorder that generically involves the sudden loss of circular patches of scalp hair. The mechanism of AA is gradually being clarified, and currently available evidence suggests that AA can be considered as a T-cell-mediated autoimmune disease.¹ A history of atopic disposition and autoimmune disease is associated with an increased risk of AA or alopecia universalis (AU), which is a severe form of AA, and the prognosis looks less favorable.^{2,3} The severer AA is at onset, the worse the prognosis.⁴

A 57-year-old woman who had mild atopic dermatitis was referred to our hospital for treatment of scalp hair loss that had rapidly expanded to the whole body (Fig. 1a). A skin biopsy taken from her scalp revealed a hair follicle infiltration in the peribulbar area (Fig. 1b), and we diagnosed the patient's condition as AU in 2009. Laboratory investigations showed that serum IgE was increased (6908 IU/mL; normal, <380 IU/mL), with no other abnormal findings. She was treated with a topical corticosteroid, an oral anti-histamine, and cepharanthine during one year at the former hospital. Furthermore, we performed semi-pulse corticosteroid therapy (500 mg/daily for three days) and cryotherapy for 14 months in addition to those treatments. Since 2011, we treated the patient with excimer light therapy (4.2 J/cm² total dose) at two-week intervals for 24 sessions. Although hair growth was partially achieved during the treatment with cryotherapy and excimer light therapy, alopecia reoccurred. Therefore, the patient opted for only conservative medical treatments such as an oral anti-histamine. In 2012, we belatedly discovered that she had kept a dog since 2002 and slept with her dog next to her every day. Laboratory tests revealed that serum value of the specific IgE antibody against dog's dander was very high (2460 UA/mL; normal, <0.34 IU/mL). We persuaded the patient not to sleep next to her dog, and she followed the advice from May 2012. After three months of follow-up, the alopecia gradually improved (Fig. 1c, d). The hair growth was slow, but favorable (Fig. 1e, f). Additionally, her atopic dermatitis also got much better as the SCORAD score decreased from 21.0 to 5.3. Finally, two years and seven months later, her alopecia was completely in remission, including body hair (Fig. 1g, h). Laboratory tests showed that the specific IgE antibody of the dog's dander was clearly lower than that at the first time (894 UA/mL in 2014).



Fig. 1. (a) She had a few scalp hairs at the initial visit. (b) A skin biopsy obtained from scalp showed moderate peribulbar lymphocytic inflammation (hematoxylin-eosin [HE], original magnification $\times 100$). (c) (d) Three months after we persuaded her not to sleep with her dog, hairs of parietal region was recognized. (e) (f) Her terminal hair grew about 85% in November 2013 (after 18 months of follow up). (g) (h) Her scalp and body hair grew completely in December 2014 (after 2 years 7 months of follow up).

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Atopic disposition is well known as a risk factor of AU,^{2,3} however, pathogenetic relationship between AU and allergy is not clearly understood. In the present case, avoiding contact with dog resulted in significant improvement of hair growth in the patient of refractory AU with the high specific IgE antibody levels of the dog's dander. This observation suggests that hypersensitive reaction induced by allergen might be involved in the pathogenesis of alopecia and could be a clue to determine the mechanism of AU in atopic patients and lead to a new approach to treatment.

Conflict of interest

The authors have no conflict of interest to declare.

Takahiro Arita ^a, Tomoko Nomiya ^{b,*}, Jun Asai ^a, Norito Katoh ^a

^a Department of Dermatology, Graduate School of Medical Science, Kyoto Prefectural University of Medicine, Kyoto, Japan

^b Shouwa Skin Clinic, Hokkaido, Japan

* Corresponding author. Department of Dermatology, Graduate School of Medical Science, Kyoto Prefectural University of Medicine, 465 Kajii-cho, Kawaramachi-Hirokoji, Kamigyo-Ku, Kyoto 602-8566, Japan.

E-mail address: tomoko-k@koto.kpu-m.ac.jp (T. Nomiya).

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