

LETTER TO THE EDITOR

Case of intractable ophiasis type of alopecia areata presumably improved by fexofenadine

Dear Editor,

Alopecia areata (AA), the most common cause of inflammation-induced hair loss, is a T-cell-mediated autoimmune disease restricted to hair follicles. Disease onset is associated with the collapse of hair follicle immune privilege both in humans and in animal models. Ophiasis type AA is one of the severe types of AA, and is characterized by a turban- or snake-like pattern of hair loss, affecting a band-shaped area in the occipital and temporal scalp regions. Ophiasis type AA as well as AA totalis in general results in the worst outcomes with lower frequency of spontaneous remission and poorer responses to conventional therapy than other clinical types of AA.¹ Despite our understanding of the pathophysiology of AA, evidence-based treatments are still limited, especially for patients with severe types of AA. Previous clinical studies have suggested that antihistamines, including fexofenadine and ebastine, have favorable effect in severe AA patients when used concurrently with topical immunotherapy.² However, the efficacy of antihistamine agents against ophiasis type AA has not been discussed in the published work in detail. Here, we present a case of ophiasis type AA remarkably improved after administration of fexofenadine. Although it is a single case observation, the present case indicates that fexofenadine might be a potential treatment for refractory AA including ophiasis type AA, especially in patients with atopic dermatitis.

A 19-year-old woman with a past medical history of atopic dermatitis in childhood presented at our clinic with ophiasis type AA (Fig. 1a). She had noticed hair loss in a small round area starting 5 months before her first presentation, and it had worsened rapidly to a snake-like pattern of hair loss at a band-shaped area of the scalp. We initially treated her with very strong class topical corticosteroids and p.o. administration of 6 mg/day cepharanthine, but no significant improvement was observed over the next 4 months. We then stopped cepharanthine and began 120 mg/day fexofenadine, a histamine H1 receptor (H1R) antagonist, after obtaining informed consent. Three months later, her hair regrowth had become apparent (Fig. 1b); 8 months later, cosmetically satisfactory regrowth was achieved (Fig. 1c). Fexofenadine treatment was well tolerated with no apparent side-effects.

It remains unclear how fexofenadine improved ophiasis type AA. It has been reported that fexofenadine enhances the efficacy of contact immunotherapy for extensive AA in patients with atopic backgrounds.³ Interferon (IFN)- γ induces the expression of major histocompatibility complex (MHC) class I and II, intercellular adhesion molecule (ICAM)-1 and human leukocyte antigen (HLA)-DR on the hair epithelium and dermal papilla in AA.



Figure 1. Clinical presentation. Clinical pictures of the patient: before (a), 3 months after (b) and 8 months after (c) starting treatment with fexofenadine.

On the other hand, degranulating mast cells are observed adjacent to affected hair follicles in AA and histamine enhances the IFN- γ -induced expression of ICAM-1 and MHC class I on keratinocytes. Collectively, antihistamines are expected to be effective for AA treatment with an atopic background. Consistently, previous reports indicated the possibility of fexofenadine as an alternative drug after contact immunotherapy for AA.^{3,4} In the present case, ophiasis type AA without history of contact immunotherapy was improved after administration of fexofenadine. Although it should be noted that we cannot exclude the possibility of a course of natural regression of AA from a single case observation, the present case suggested that fexofenadine

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might be a potential treatment for refractory AA including ophiasis type AA, especially in patients with atopic backgrounds. Further studies will be needed to establish the efficacy of fexofenadine therapy on severe AA including ophiasis type AA with or without atopic background.

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