A case of atopic dermatitis with alopecia universalis in a patient treated with abrocitinib

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Key words: alopecia universalis; atopic dermatitis; comorbidity; Janus kinase inhibitor.

INTRODUCTION

Atopic dermatitis (AD) is sometimes accompanied by alopecia areata (AA) or, in severe cases, alopecia universalis (AU). Preclinical studies have shown that type 1 and type 2 cytokines are involved in AD and AA.[1,2] Janus kinase (JAK) inhibitors are effective blockers of JAK/signal transducer and activator of transcription-mediated inflammatory signaling pathways, which regulate multiple cytokines, such as interleukins and interferons.[3] A recent case report showed hair regrowth after simultaneous treatment of AU and AD with tofacitinib, a selective inhibitor of JAK 1 and JAK 3.[4] Here, we report for the first time, to our knowledge, the clinical use of oral abrocitinib (a specific JAK 1 inhibitor) in the management of both AD and AU.

Abbreviations used:

AA: alopecia areata
AD: atopic dermatitis
AU: alopecia universalis
JAK: Janus kinase

CASE REPORT

We present the case of a 14-year-old girl with a history of AD since childhood, a 3-year history of AU, and allergic rhinitis. According to her parents, she developed a rash combined with gradual full-body hair loss 3 years previously, over the 4 months after receiving a measles-mumps-rubella vaccination injection (Fig 1, *A-C*). Routine laboratory tests revealed an increasing immunoglobulin E level (>5000 kU/L) and an elevated lactate dehydrogenase level (>300 U/L). Physical examination showed generalized symmetrical red patches and plaques on the limbs and trunk, with several ulcers and scabs. She was diagnosed with severe AD (Investigator's Global Assessment = 4). Prior to consultation, she had experienced no significant changes with the use of topical steroids, oral antihistamines, and Chinese acupuncture treatments. Therefore, the patient was treated with abrocitinib (200 mg), administered orally once every day. After 12 weeks of treatment, she noted patchy hair regrowth on all affected body parts, which indicated an effective therapy. Fifty-two weeks after the initiation of therapy, thick regrowth of terminal hairs was noted on her scalp, eyebrows, limbs, and axillae (Fig 1, *D* and *E*). Meanwhile, the AD lesions subsided (Investigator's Global Assessment = 1) after abrocitinib administration (Fig 1, *F*). At present, which is more than 2 years after therapy initiation, the patient's AU symptoms are completely relieved, and AD lesions relapsed mildly (Investigator's Global Assessment $\leq$ 2) after the follow-up period.

DISCUSSION

AD is a high-risk factor for developing AA/AU, and it is often associated with the burden of mental health conditions. Recent population-based studies have shown a bidirectional association between AA/AU and AD,[5,6] suggesting that these 2 diseases may share underlying mechanisms. In this representative case,

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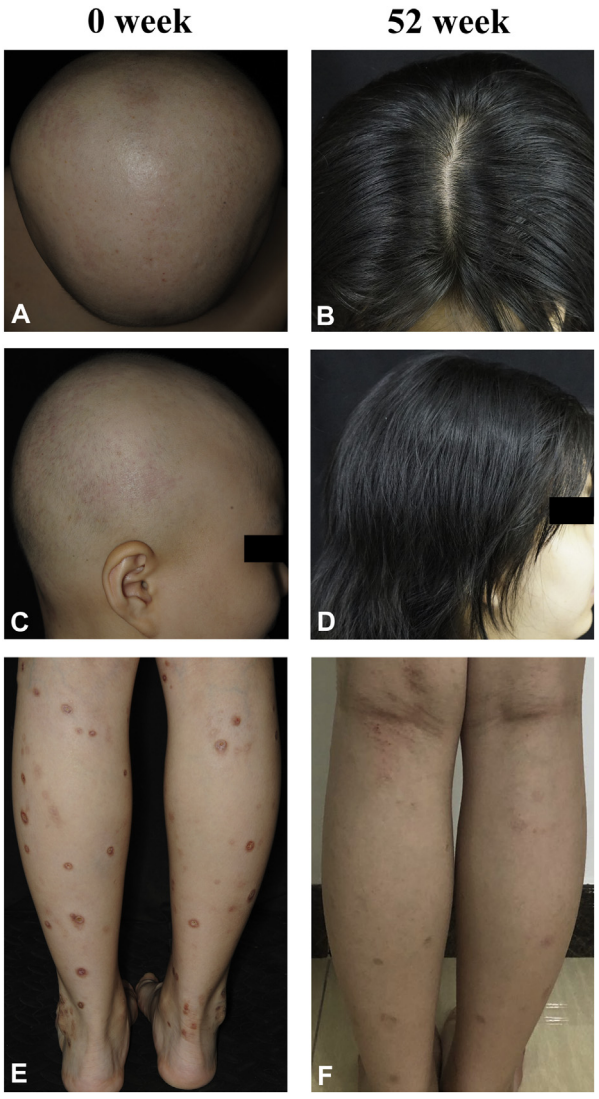

Fig 1. Therapeutic assessment of individual with atopic dermatitis accompanied by alopecia universalis. **A,** Superior cranial view of the scalp before abrocitinib treatment. **B,** Superior cranial view of the scalp after abrocitinib treatment. **C,** Right temporal view of the scalp before abrocitinib treatment. **D,** Right temporal view of the scalp after abrocitinib treatment. **E,** Posterior view of the lower limbs before abrocitinib treatment. **F,** Posterior view of the lower limbs after abrocitinib treatment.

the injection of a measles-mumps-rubella vaccination appears to have triggered the development of AU. A similar case of vaccine-induced AA/AU has also been reported after COVID-19 vaccination.[7] The exact mechanisms driving such rare events are not yet fully understood, but vaccine-induced antibodies may intensify autoimmune disorders through JAK-signal transducer and activator of transcription activation. Despite a mild relapse of AD, the patient remains satisfied with the therapy outcome. The possible side effects include infection, abnormal transaminase, cardiotoxicity, and skeletal dysplasia; however, none of these were observed at the time of this study. Therefore, our results demonstrate that abrocitinib can be an effective therapeutic option for patients with AD accompanied by refractory AU.

Conflicts of interest

None disclosed.

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