Linear alopecia areata



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Key words: alopecia; alopecia areata; linear alopecia.

INTRODUCTION

Alopecia areata (AA) is a common, immunemediated, nonscarring hair loss disorder. It typically presents with sharply demarcated round patches of hair loss and can be found in any age. Severe hair loss forms include alopecia totalis and alopecia universalis. AA may significantly affect a patient's quality of life. Here, we report a case of AA presenting a linear form of alopecia.

CASE REPORT

A 28-year-old man presented to our outpatient department with a history of repeated hair loss on the scalp in a linear pattern for the last 4 years. He had scalp hair regrowth after treatment with minoxidil 5% solution but the hair loss usually came back. The affected area was 10 cm long and 2 cm wide traversing the parietal scalp (Fig 1, A). The skin of the alopecic area was smooth and normal in color and consistency without any other skin changes such as erythema, atrophy, or scales. The hair pull test and the perilesional trichogram were normal. Besides the alopecia, the man was otherwise healthy without other skin and nail changes. There was no family history of alopecia or autoimmune diseases. A trichoscopic evaluation found short vellus hairs in the alopecic area (Fig 1, B). Laboratory examinations including complete blood counts, liver and renal functions, urinalysis, syphilis (rapid plasma reagin and treponemal pallidum partical agglutination), and C3 and C4 were normal, whereas a weakly positive antinuclear antibody with a titer of 1:40 was found. Histopathologic findings of the lesional skin included increased catagen and telogen follicles and negative staining for mucin on special staining with alcian blue (Fig 2, A and B). A diagnosis of AA was considered. He was treated with intramuscular injection of betamethasone 17α , 21-dipropionate and Abbreviations used:

AA: alopecia areata

EP: lupus erythematosus profundus





Fig 1. A, The clinical manifestation. **B**, The trichoscopic evaluation.

21-disodium phosphate monthly along with topical minoxidil 5% solution.

DISCUSSION

AA over the scalp shows various shapes and extents of hair loss, the most common pattern is a small annular or patchy bald lesion that can progress to total loss of scalp hair. Dermatoses in linear

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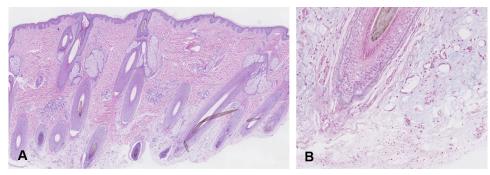


Fig 2. A, Hematoxylin-eosin staining (×40). **B**, Alcian blue staining (×100).

distribution are not uncommon, such as linear lichen planus, linear scleroderma, and linear epidermal nevi, but alopecia in a linear form is rare; only a few cases are reported in the medical literature. Eight cases of linear lupus erythematosus profundus (LEP) in the scalp presented as alopecia, as they simulated AA clinically but were proven otherwise by histopathology.²⁻⁸ Only 1 case of linear alopecia areata was reported in 2016.9 The patient, a 35-year-old man, presented with a linear bald patch on the scalp for 3 years. AA was finally diagnosed with histopathologic findings. Nonscarring patchy alopecia associated with LEP is sometimes misdiagnosed as AA. Clinical manifestations, scalp dermoscopic signs, and histopathologic findings of the nonscarring alopecia can help to differentiate between the 2 diagnoses. Decreased ratio of anagen follicles to catagen and telogen follicles, increased number of vellus follicles, and a swarm of bees-like presentation of lymphocytic inflammation around anagen follicles are the histopathologic characteristics of AA. The phenomenon of the swarm of bees may be absent in the chronic stage of AA. Deep dermal perivascular lymphoid infiltrates and abundant mucin deposition with patches in the subcutaneous fat tissue are considered the features of LEP. In this case, the patient had repeated hair loss and regrowth after treatment with minoxidil. Increased catagen and telogen follicles and short vellus hairs in the alopecic area were consistent with the features of AA. Lack of mucin deposition in the subcutaneous

tissue ruled out LEP. When nonscarring alopecia appears, the potential for an LEP disease flare-induced hair loss should be considered rather than a diagnosis of AA. It is difficult to explain why AA presented in a linear form.

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