Severe and Unresponsive HIV-associated Alopecia Areata Successfully Treated with Thalidomide

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Sir,

Alopecia areata (AA) is a non-scarring recurrent cause of hair loss afflicting approximately 1–2% of the general population. Up to 15% of patients progress to a severe form of the disease, and develop total scalp (alopecia totalis) or scalp and body hair loss (alopecia universalis). At this time spontaneous remission occurs rarely and the treatment is usually difficult. Thalidomide has turned out to be an extremely interesting drug, not only for inflammatory skin conditions. We describe here a patient with severe AA, resistant to several treatments, who showed a dramatic response to thalidomide.

CASE REPORT

A 26-year-old man with HIV infection classified as clinical category A3 (according to the current CDC classification) with a CD4 lymphocyte count of 105 cells/μl was seen at our hospital with alopecia. The patient had no family or personal history of AA or atopy, or other hair disease. He had developed a patchy alopecia of the scalp after 4 months of highly active antiretroviral therapy (HAART) with stavudine, didanosine and nevirapine. Potent topical corticosteroids with occlusion for 12 weeks were prescribed with no beneficial effect and several discrete alopecia patches became confluent. Then, a 12-week taper of prednisone was prescribed, with an initial dose of 1.5 mg/kg/day, but this systemic glucocorticoid therapy neither induced hair regrowth nor prevented the spread of alopecia, which extended to the eyebrows. Physical examination confirmed the confluence of alopecia patches in scalp and eyebrows with no signs of hair growth. No nail changes were observed.

For his HIV infection, we decided to prescribe topical diphencyprone as immunotherapy for his AA. After 36 weeks of this therapy no terminal hair growth was observed and the AA progressed to an almost total loss of the scalp hair (Fig. 1a). At this time, a punch biopsy specimen was obtained, which demonstrated melanin incontinence, a moderate lympho-histiocytic cell infiltrate, as well as small ball-shaped protrusions from the outer root sheath of telogen hair follicles and enlarged sebaceous glands, findings in accordance with the diagnosis of long-standing AA.

As different cytokines, mainly tumour necrosis factor-α (TNF-α), are involved in the pathogenesis of AA (1, 2), we decided to prescribe thalidomide to this patient, a drug that inhibits the synthesis of this pro-inflammatory and pro-apoptotic cytokines, and that is used with safety

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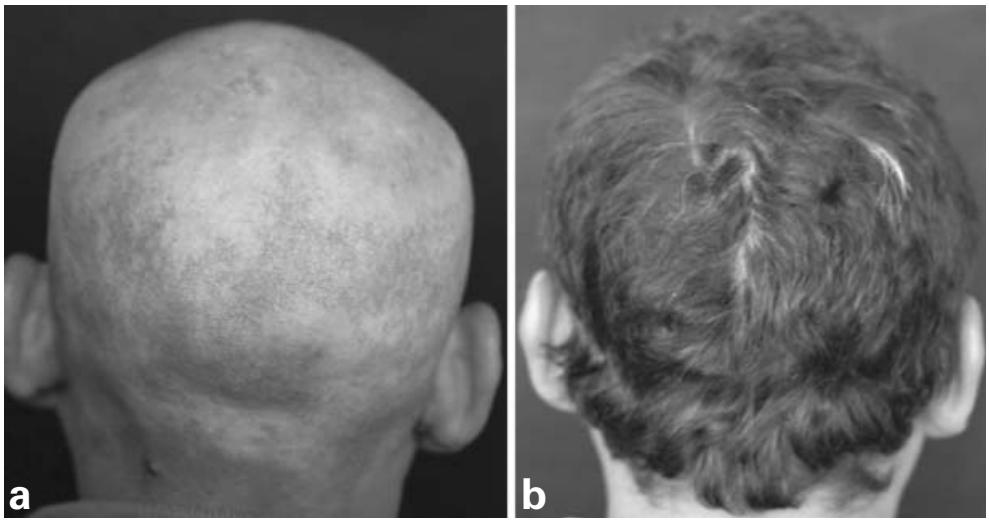

Fig. 1. (a) Extensive and confluent areas of alopecia areata on the scalp. (b) Four months after beginning of thalidomide therapy. The patient's scalp is fully covered by terminal hair.

in patients infected with HIV (3). A trial of oral thalidomide was started with a dose of 100 mg b.i.d. following 4 weeks of wash off. At 30 days after the beginning of treatment, we observed terminal hair growth in the parietal area. This treatment was administered for 15 additional days, then the thalidomide dose was tapered to 100 mg once daily for 30 days, one 50-mg tablet daily for 15 days, 25 mg every day for 15 days, 25 mg every other day for 15 days, and then the treatment was stopped. A remarkable improvement was observed, with hair regrowth in different zones of the scalp (Fig. 1b). After 4 months of thalidomide therapy the patient was fully recovered, and no evidence of hair loss has been found during a follow-up period of 18 months. Routine neurological examination and nerve conduction studies, performed before and regularly during thalidomide therapy, ruled out the presence of peripheral neuropathy, a frequent side effect of this drug (4). After treatment, we observed an increase in CD4 counts to 237/μl. Medications used to treat HIV infection were not modified during treatment of the hair disease.

DISCUSSION

We report here for the first time the efficacy of thalidomide in the treatment of severe AA associated with HIV infection. We found a dramatic response in this patient in a short period of time, and with no apparent adverse effects. It is very likely that the ability of thalidomide to suppress TNF-α synthesis and expression of different adhesion molecules (5) is causally related to the clinical response seen in this patient. However, the activity of this drug on the synthesis of transforming growth factor-β, a cytokine involved in the modulation of hair growth (6), could also contribute to the observed effect. In light of this result, we consider that further studies should be undertaken in more patients with AA in order to support the use of thalidomide as a therapeutic agent in these cases.

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