Alteration in Hair Texture Following Regrowth in Alopecia Areata

A Case Report

Whitney Valins, BS; Janelle Vega, MD; Sadegh Amini, MD; Heather Woolery-Lloyd, MD; Lawrence Schachner, MD

Background: Alopecia areata is a common cause of hair loss seen in 3.8% of patients in dermatology clinics and in 0.2% to 2.0% of the general US population. The pathology of the disease remains poorly understood. Hair loss in alopecia areata can range from a single patch to 100% loss of body hair. When hair regrowth occurs in alopecia areata, the new hair may demonstrate pigment alterations, but a change in hair texture (ie, curly or straight) has rarely been reported as a consequence of alopecia areata.

Observations: We report a case of an alteration in hair texture following regrowth in a male adolescent patient with alopecia areata. The new hair recapitulated his hair shape from early childhood.

Conclusions: The precipitating factor for a change in hair texture in alopecia areata may be a result of treatment, pathophysiologic changes, or a combination of both. Whether the change is triggered at the level of stem cell differentiation, by cytokine or hormonal influences, gene expression during hair follicle development, a combination of all of these, or an unknown cause is a question that remains to be answered.


Alopecia areata (AA) is a common cause of hair loss seen in 3.8% of patients in dermatology clinics and 0.2% to 2.0% of the general US population. Usually up to 50% of patients recover within 1 year of treatment, but spontaneous remissions and frequent relapses have been reported in up to 90% of cases. Initially during regrowth, the new hair may have a smaller shaft size and may show pigment alterations ranging from total achromia to mild hypopigmentation, with a tendency toward total repigmentation over time. A review of the literature has shown that the shape (ie, curly or straight) of the new hair after regrowth is usually similar to the shape of the hair prior to the AA episode, although rare reports of hair shape changes have been documented. Herein, we report a case of an alteration in hair shape following regrowth in a male adolescent patient with AA, where the regrowth resembles the patient’s hair shape from early childhood.

A 13-year-old African American boy presented with “a few months of hair loss.” His medical history included seborrheic dermatitis limited to the scalp, self-treated with an over-the-counter medicated shampoo; and seasonal allergic rhinitis and hypersensitivity to cat allergens, both treated with a mometasone furoate monohydrate nasal spray as needed. His family history was significant for hypothyroidism diagnosed in his mother and maternal grandmother, and negative for AA.

Physical examination revealed a 5 × 3-cm patch of hair loss on the right supratemporal hairline and a 3.5 × 2.0-cm patch of hair loss on the vertex of the scalp, with evidence of partial regrowth. There was no evidence of cicatricial alopecia or skin lesions suggestive of discoid lupus erythematosus. In addition, nail pitting was not seen. Relevant laboratory findings included mild elevation in the serum level of free thyroxine.
Hair shape, or curliness, is a spectrum ranging from straight hair to tightly curled (frizzy) hair. Prior classifications of hair shape included 3 categories based on ethnicity: African, Asian, and European. Recently, several morphologic parameters have been used to broaden the hair shape classification to 8 types, independent of ethnicity. These parameters include the curve diameter, the curl index (which measures the ratio of the stretched length of the hair related to its length at rest), the highest number of waves in the hair when it is pulled to four-fifths of its length, and the number of twists detected along the fiber.

After the clinical diagnosis of AA was made, the patient was treated with clobetasol propionate foam, 0.05%, once daily; triamcinolone acetonide cream, 0.1%, twice daily Monday through Friday; and clobetasol propionate cream, 0.05%, twice daily Saturday through Sunday.

After 3 months, the patient returned for a follow-up visit and showed evidence of substantial hair regrowth in both areas of prior alopecia. In addition, the newly growing hair in the 2 areas of prior alopecia displayed a different shape (ie, straight), in contrast to the curly shape seen on the rest of the patient's scalp (Figure). The patient's mother confirmed that the hair shape displayed in those specific areas of prior AA was similar to the hair shape seen in the patient during childhood. At a 1-year follow-up visit, the patient had not developed any new alopecic patches, and the hair growing in the areas of prior AA continued to display a straight shape, in contrast to the rest of the hair on his scalp.

**COMMENT**

Acquired straightening of the hair has been described in patients with human immunodeficiency virus (HIV) as well as in patients with chronic malnutrition and has been ascribed to mineral deficiencies and hormonal dysfunction. Kinking of the hair (in addition to pigmentation changes) has also been reported with the use of acitretin and etretinate, which are thought to be associated with a change in keratinization patterns within the inner root sheath.

To our knowledge, this is the first report of a patient with AA who experienced a change in hair structure that recapitulated the patient's hair texture as a child. The precipitating factor for this change may have been a result of treatment of AA, pathophysiologic changes associated with the AA, or a combination of both.

The pathogenesis underlying AA is yet to be fully understood. It is clear that immune dysregulation consisting of cytokines, hormones, and T cells cause the hair cycle to become dysfunctional and come to a halt. When the hair follicle reenters anagen, the same signaling molecules active during morphogenesis (ie, WNT and SHH) trigger hair growth. In their work on hair follicle development, Legue et al suggest that each hair cycle is a distinct morphogenetic event during which hair stem cells, under the influence of growth factors and cytokines, migrate from the bulge to promote new follicle growth, mostly maintaining their omnipotent properties.

In our patient, hair regrowth was achieved, but the pathologic changes induced by inflammation and/or treatment caused an alteration of hair texture. Whether the change was triggered at the level of stem cell differentiation, by cytokine or hormonal influences, by gene expression during hair follicle development, a combination of these, or an unknown cause is a question that remains to be answered. Elucidating triggers in hair texture change could help us further understand AA. Furthermore, it could have a major impact on the world of hair grooming, where countless hours are spent by people attempting to change their hair texture.

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Correspondence: Lawrence Schachner, MD, Department of Dermatology and Cutaneous Surgery, University of Miami, Miller School of Medicine, 1600 NW 10th Ave, RMSB, Room 2023A (R250), Miami, FL 33136 (lschachn@med.miami.edu).

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REFERENCES