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Case Report

Combination of light-emitting diode with minoxidil 2%, topical corticosteroid and oral immunomodulator induced hair regrowth in a pediatric alopecia areata

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Abstract Alopecia areata (AA) is a form of non-scarring hair loss caused by an autoimmune disease. Symptoms range from a total or severe to an only mild patchy loss of hair. Spontaneous remission may occur. Even though AA is the third most frequent reason for pediatric dermatology consultations, treating alopecia areata in children is still difficult. A 4-year-old, healthy girl, presented with non-scarring multiple patchy alopecias on the scalp, and the hair pull test was positive. Trichoscopy demonstrated exclamation marks, black dots, short vellus hairs, and telangiectasia. Routine laboratory tests were unremarkable. The patient was clinically diagnosed with alopecia areata. Dermoscopy may confirm the diagnosis. Therapies are designed to alleviate symptoms and signs. More pediatric data are needed to evaluate therapies' safeness and recurrence rates in such treatment approaches. Topical treatment using high potency topical corticosteroid and minoxidil 2% combined with light-emitting-diode (LED) and oral immunomodulator (inosine pranobex) showed good response and well tolerated in pediatric alopecia areata.

Key words
Alopecia areata, diode, immunomodulator, pediatric.

Introduction

Alopecia areata (AA) may occur on any or all parts of the body as a result of the complicated immunologic and multigenic inflammatory disorder that appears as a non-scarring hair loss.[1] Its most prevalent subtype is patchy AA. It is regarded as a hair follicle-specific autoimmune disorder caused by environmental triggers in genetically predisposed individuals.[2] The disease affects 0.63% of the pediatric population and 0.2% of the general population.[3] The lifetime risk of developing AA is estimated at 1.7%.[4] All ethnicities and genders appear to be afflicted similarly. Although the majority of patients experience it in their first three decades of life, it might be present at any age.[2] As the third most frequent skin disorder among children, AA is linked to a poorer health-associated life quality for both parents and the patients.[5] There are many treatment options available for AA including topical and systemic modalities, but none have been clinically proven to be consistently effective.[6]

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Case report

A 4-year-old, healthy female, presented with alopecia on the scalp. She had no history of asthma, atopic conditions, or any systemic diseases, and no other relevant family history. Two weeks prior to the hair loss, she had just recovered from tonsillitis. A physical examination revealed patchy nonscarring

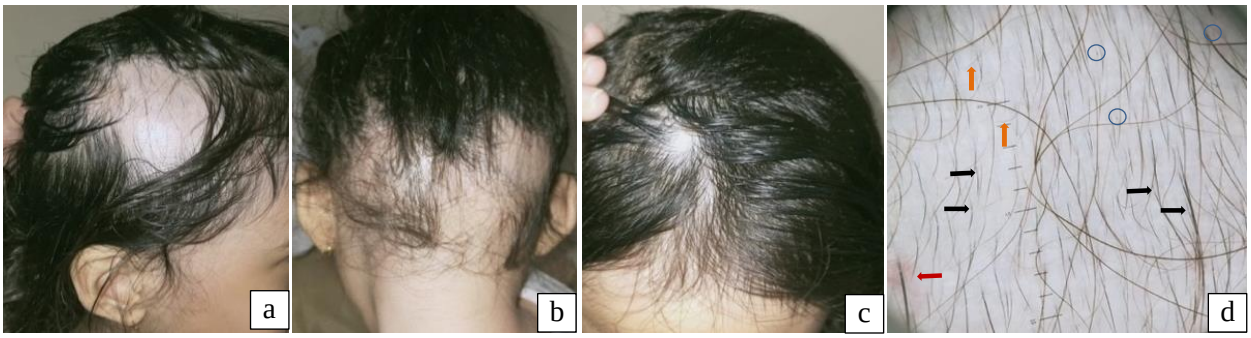

Figure 1 Initial lesions of AA in a 4-year-old girl appear as patchy hair loss on the right parietal (a), nuchal (b), and mid-scalp (c) regions. Vellus hair (green arrow), exclamation mark (black arrow), black dots (blue circle) and telangiectasia (red arrow) were found in her scalp (d).

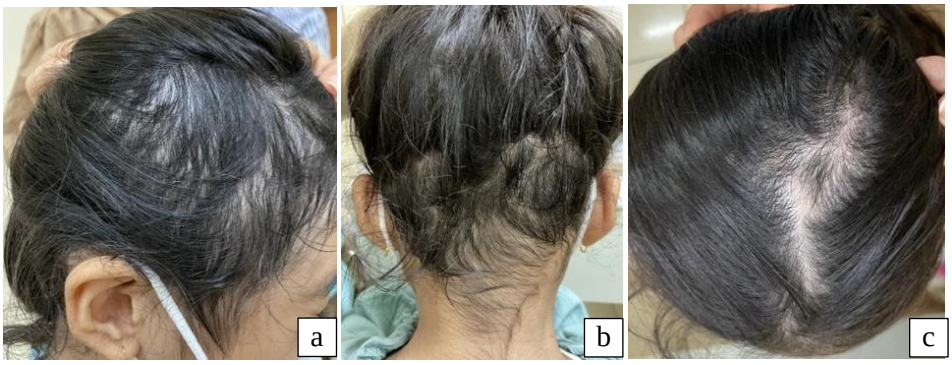

Figure 2 Clinical improvement on the right parietal (a), nuchal (b), and mid-scalp (c) regions, achieved after 3 months of treatment including 5 sessions of LED.

alopecia on the right parietal, mid-scalp, and nuchal regions of the scalp. The hair pull test was positive. Trichoscopy demonstrated exclamation marks, black dots, short vellus hairs, and telangiectasia **(Figure 1)**. She had no nail involvement. Evaluation of complete blood count, thyroid function, vitamin D, and blood glucose level were within normal limits TORCH and *Mycoplasma* screening showed high anti-CMV IgG antibody titers.

The patient had already been treated by another dermatologist a month earlier with topical superpotent corticosteroid and salicylic acid 1% without improvement but the appearance of new lesions. We initiated topical mometasone furoate lotion 0.1%, minoxidil 2% solution, oral inosine pranobex 3X170 mg twice a week, and photobiomodulation using light-emitting diodes (LED). Two months after the treatment with 4 sessions of LED, hair regrowth was noted on her right parietal and nuchal areas. However, her mid-scalp area continued to lose hair. The treatment continued without any additional therapy. The patient visited our clinic a month later (due to the pandemic situation) with a cosmetically satisfying result **(Figure 2)**.

Discussion

The patient was clinically diagnosed as AA due to its characteristic lesion; patchy non-scarring alopecia. Empty follicular openings and short vellus hairs are the most prevalent trichoscopic findings of pediatric AA.[3] She had exclamation marks, vellus hairs, and black dots. She also had serological evidence of past CMV infection. A case-control study reported all AA cases had a high anti-CMV IgM antibody titer indicating a significant association between CMV and AA.[7]

The response of AA to treatment is unpredictable.[1] Even during a course of successful treatment, relapse rates are high.[4]

Topical corticosteroids are most commonly used in children.[6] Its response rate in monotherapy is 81% (35/43) and its relapse rate is 53% (9/17). Topical corticosteroid adverse reactions were hardly seen in pediatric AA.[1]

The amount of research conducted on the efficacy of minoxidil in pediatric AA is low. Minoxidil used in pediatric AA with concentration varied between 1-5 %.[1] An RCT of pediatric AA evaluated 2% topical minoxidil after discontinuing 6-week course of prednisolone. Hair regrowth was found in 86% of the minoxidil group versus 17% of the placebo at 12 weeks.[8] Also known as isoprinosine or methisoprinol, inosine pranobex is an immunomodulator that enhances the activity of phagocytic cells and T-cell lymphocytes. A placebo-controlled study found significant complete hair regrowth in 33.3% of patients (16-48 years old) in the oral inosine pranobex group.[9] Therapy with LED is FDA-approved and considered to be safe.[9] Treatment of AA with LED every 1 or 2 weeks for up to 5 months showed hair regrowth in 46.7% of the treated areas, 1.6 months preceding the non-irradiated areas in patients above 18 years old ($p=0.003$).[10] The patient had AA before puberty, which commonly has a worse prognosis than AA among adults. Negative prognostic factors of AA include nail involvement, family history, atopy, and the presence of other autoimmune diseases.[1] Certain immunological and metabolic problems are more common in children with AA compared to the general pediatric population. Although our case had no comorbidities, clinicians should be conscious of the elevated risk and recommend additional testing if metabolic or immunologic diseases are presumed.[5]

Conclusion

Treatment using high potency topical corticosteroid and minoxidil 2% combined with LED and oral inosine pranobex showed good response and was well tolerated in a 4-year-old AA patient.

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