SKIN

BRIEF ARTICLE

A Rare Presentation of Concomitant Alopecia Areata and Vitiligo in a Teenager

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ABSTRACT

Introduction: Alopecia areata (AA) and vitiligo are two chronic, autoimmune skin diseases. While these two conditions are common, their co-existence is rare.

Case Presentation: A 13-year-old Caucasian female presented to the dermatology clinic with a 1-2-month history of hair loss on her occipital scalp. The patient was diagnosed with AA and prescribed topical mometasone 1% lotion twice daily and pulse-dose prednisolone solution 12 mL 1 day per month for 3 months. At her 3-month follow-up, the patient reported new-onset hair depigmentation. On physical examination, the patient's occipital scalp and right eyelash demonstrated a depigmented patch of hair, concerning for vitiligo. The patient was diagnosed with concomitant disease and topical tofacitinib was added to her treatment regimen.

Discussion/Conclusion: The colocalization of AA and vitiligo is rare, and the presentation suggests an underlying pathogenic link between the two skin diseases. While the definitive immunologic pathway remains unknown, researchers have narrowed down inflammatory markers involved in the development of both conditions, including CD8+ cytotoxic T-cells, interferon gamma (IFN-ɣ), and IFN-ɣ-induced chemokines. We present a 13-year-old female patient who presented to the clinic with scalp alopecia and later developed overlying scalp and eyelash vitiligo. The rare nature of the patient's presentation makes concomitant disease a therapeutic challenge and can impose significant psychological distress to a pediatric patient.

INTRODUCTION

Alopecia areata (AA) and vitiligo are two chronic, immune-mediated skin conditions. The clinical presentation of AA is inflammatory, patchy hair loss. In contrast, vitiligo presents as circumscribed, depigmented macules or patches of skin in a sporadic or segmental distribution. The two conditions are common; the lifetime incidence of AA and vitiligo are 2% and 1%, respectively.[1,2] However, coexistent disease is rare. While their clinical presentations differ, their pathogenesis is believed to be similar. We present a 13-year-old female who presented to the dermatology clinic with the classic presentation of AA. The patient then developed depigmented patches of hair on her scalp and right eyelash, concerning for AA-vitiligo disease overlap

CASE PRESENTATION

A 13-year-old Caucasian female with no pertinent past medical history presented to the dermatology clinic for evaluation of bald

spots on her scalp. She said she first noticed hair loss 1-2 months ago, and she had not used a new scalp or hair treatment. The patient denied weight changes, dry skin, heat/cold intolerance, trichotillomania, pruritus, pain, and bleeding from the site. The patient's family history is pertinent for a paternal grandmother and paternal aunt with thyroid disease and psoriasis. On physical examination, the patient demonstrated patchy, non-scarring hair loss on the right side of the scalp and scalp vertex. She was diagnosed with alopecia areata and offered both intralesional and topical treatment. At this time, she declined steroid injections and was prescribed mometasone 0.1% lotion twice daily to affected areas and pulse-dose prednisolone solution 12 mL daily for 1 day of the month for 3 months. At her 1-month follow-up, the patient reported new areas of hair loss on the left and occipital scalp and diffuse scaling, despite consistent use of mometasone. She was encouraged to continue her treatment regimen at this visit. At her 3-month follow-up, the patient reported hair growth since her last visit. On physical examination, a patch of depigmented hair was discovered on her occipital scalp and right eyelash (Fig 1A, 1B). The patch of depigmentation was suspicious for vitiligo, and the patient was instructed to add crisaborole 2% ointment to the white areas daily. The patient was unable to afford the ointment, and she was instead prescribed topical tofacitinib for the white areas. She was scheduled for a follow-up in 4-6 weeks.

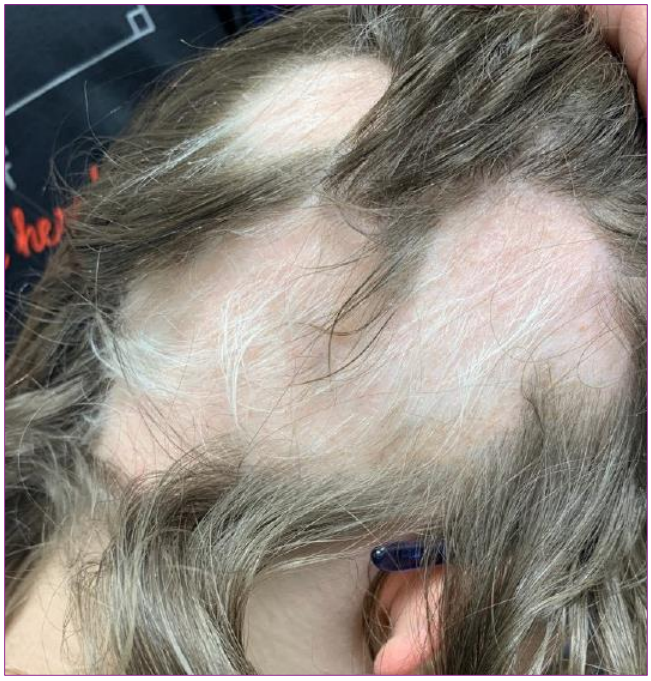

Figure 1A. Alopecia areata with concomitant vitiligo on occipital scalp. The patient demonstrated patchy hair loss with overlying depigmented patches of skin and hair on her posterior scalp concerning for vitiligo.

Figure 1B. Vitiligo on right eyelash. The patient demonstrated depigmented hair growth in her right eyelash

DISCUSSION

Alopecia areata (AA) is an organ-specific, inflammatory, and non-scarring form of hair loss. The condition is caused by CD4+ and CD8+ T-cell-mediated destruction of anagen-phase hair follicles.[3] AA often presents in patients aged 30-40 and there is no sex predominance; the degree of disease ranges from mild, circumscribed patches of alopecia to complete scalp and body hair loss.[3] The etiology and pathogenesis of the disease is unknown, but it is theorized that there is an intermingling between genetic,

environmental (e.g., stress, trauma, viral illnesses), hormonal, and autoimmune factors that predispose patients to the chronic disorder. In contrast, vitiligo is a chronic, immune-mediated depigmenting skin condition; it is due to CD8+ destruction of epidermal melanocytes. This condition often presents in the pediatric population, with over half of cases reported before age 20.[2] Vitiligo and AA both fall under the spectrum of autoimmune diseases. Patients with either disease are inclined to suffer from other autoimmune cutaneous and systemic diseases, including psoriasis, lichen planus, atopic dermatitis, systemic lupus erythematous, autoimmune thyroiditis, and diabetes mellitus.[3]

The concomitant presentation of both AA and vitiligo is uncommon. However, the co-localization of AA and vitiligo is rarer, and its unique presentation has piqued dermatologists' interest in the clinical and immunologic commonalities between the two diseases. The clinical presentation of AA has been theorized to be interconnected with melanocytes and pigmentation. In AA, autoimmune destruction of hair follicles is specific to pigmented hair; white hair remains unscathed. In addition, after AA has been treated, depigmented hair grows first.[4] In vitiligo, the active re-pigmentation process first begins at the follicular and peri-follicular region of hair shafts.[4,5] These clinical manifestations support evidence of an underlying link between the two diseases. Therefore, investigations that revolve around the identification of a common pathogenic pathway are underway.

It is well-elucidated that CD8+ cytotoxic T-cells have a role in the destruction of hair follicles and melanocytes in AA and vitiligo, respectively; however, the two diseases have more striking pathogenic similarities.[6] Rork *et. al* suggest that the initiating trigger for both diseases is increased reactive oxygen species and cellular stress levels. In addition, mice models have demonstrated that interferon-gamma (IFN-ɣ) and IFN-ɣ-induced cytokines are additional drivers in disease pathogenesis.[7] Kumar *et. al* also proposed the notion that localized, pro-inflammatory Th1 cell reactions or the inactivation of an immune suppressor mechanism leads to concomitant damage to the melanocytes and hair follicles, explaining the regional overlap of the disease in our patient.[4] To date, the exact pathogenic link has not been determined.

The management of AA includes topical and intra-lesional corticosteroids, topical calcineurin inhibitors, and narrow-band ultraviolet B phototherapy (NB-UVB). The treatment of vitiligo is similar. However, the straightforward management of each disease becomes challenging when they present concurrently. In literature, there are few details regarding treatment and follow-up for patients with concomitant disease. Harris *et. al* reported success with the use of ruxolitinib, a Janus kinase (JAK) inhibitor, in a patient with coexistent AA and vitiligo. JAK inhibitors interfere with IFN-ɣ signaling and IFN-ɣ-induced chemokines, which could explain its success in the treatment of both conditions.[8] In our case, the patient presented a therapeutic challenge because of her concomitant disease. Her vitiligo was present on her right eyelash – a location which led us to choose a steroid-sparing agent such as crisaborole 2% ointment as a first-line treatment option for her vitiligo. While the FDA has approved crisaborole for atopic dermatitis in adults and children, case reports have noted its success in the treatment of vitiligo.[9] To our knowledge however, no studies have reported improvement of alopecia areata with crisaborole. While other steroid-sparing agents such as JAK inhibitors can be used

for the initial management of concomitant disease, the patient's insurance coverage prevented us from considering these agents as first-line therapy. Additional studies should be completed to determine the best management for this rare presentation.

CONCLUSION

We present a 13-year-old female who presented to the dermatology clinic for patchy hair loss and subsequent development of overlying scalp and eyelash vitiligo. The colocalization of the two diseases in her scalp is rare, and studies have proposed a common immunologic mechanism that drives the regional presentation of her disease. However, the exact pathway has not been elucidated. This presentation remains a therapeutic challenge because of limited research on adequate treatment regimens and its ability to cause significant psychosocial distress to the patient.

Conflict of Interest Disclosures: None

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