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Post-COVID-19 Alopecia Universalis: Autoimmune Hair Loss and the Challenge of Relapse Management

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ABSTRACT

Background: Alopecia universalis (AU) is the most severe form of alopecia areata (AA), characterized by complete scalp and body hair loss. While post-COVID-19 hair loss is often attributed to telogen effluvium (TE), emerging evidence suggests that COVID-19 may also trigger AU through immune dysregulation, particularly via interferon-gamma (IFN-γ)-mediated inflammation. The chronic and relapsing nature of AU raises challenges in long-term disease management, particularly regarding treatment duration and relapse prevention.

Case Presentation: We present a 79-year-old woman with no personal or family history of hair loss who developed sudden-onset AU one month after recovering from COVID-19. Initial treatment with topical and intralesional corticosteroids failed, prompting systemic therapy initiation. Over 10 months of treatment, she achieved significant hair regrowth. However, 3 months after discontinuation, hair loss recurred, reinforcing the relapsing nature of post-COVID-19 AU.

Conclusion: This case uniquely demonstrates the rapid onset and relapsing nature of post-COVID-19 AU, distinguishing it from idiopathic AU and emphasizing the need for modified long-term treatment strategies. While Janus kinase (JAK) inhibitors and other immunomodulatory therapies show promise, the risk of relapse upon treatment discontinuation highlights the need for further research into long-term management strategies and maintenance therapy in post-viral AU.

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INTRODUCTION

COVID-19 has been linked to several post-viral dermatologic sequelae, most notably telogen effluvium (TE). However, emerging evidence suggests that SARS-CoV-2 may also trigger autoimmune-mediated hair loss, including alopecia areata (AA) and its most severe form, alopecia universalis (AU).[1,2,3] UnlikeTE, which is transient and non-inflammatory, AU is characterized by auto-immuneT-cell infiltration targeting hair follicles, leading to loss of immune privilege.[2,3] Histopathologic studies of AU have demonstrated peribulbar lymphocytic infiltration, supporting the role of immune dysregulation in follicular destruction.[3] Given COVID-19's association with an elevated IFN-γ response, its role in triggering AU warrants further investigation.[4,5]

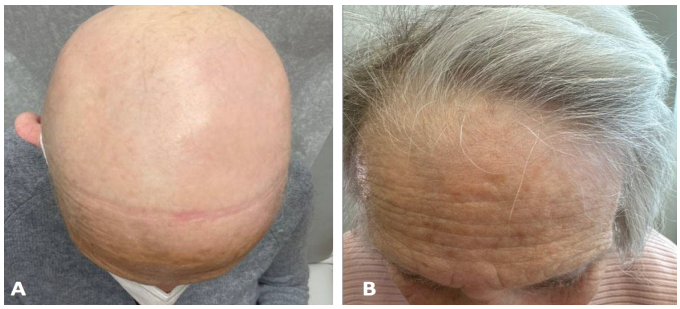
Recent studies suggest that SARS-CoV-2 may act as a viral trigger for AU via IFN-γ-driven immune dysregulation. The virus-induced prolonged inflammatory response may cause dysregulated immune privilege of hair follicles, potentially resulting in AU onset or exacerbation.[5,6,7] Additionally, AU cases have been reported following both COVID-19 infection and vaccination, suggesting that immune hyperactivation may be a key pathogenic factor.[6,8]

Although idiopathic AU is chronic and relapsing, post-COVID-19 AU may follow a distinct course due to persistent post-viral immune activation.[4,9] Given the increasing reports of post-COVID-19 AU, understanding its unique disease course and long-term management is critical. Here, we present a case of post-COVID-19 AU with relapse after treatment discontinuation, reinforcing the need for continued investigation into optimal treatment strategies.

Case Report

A 79-year-old woman with a history of hypothyroidism and thrombocytopenia (both stable and well-controlled) developed sudden, extensive, non-scarring hair loss one month after recovering from COVID-19 in January 2023. She had no personal or family history of hair loss. By February 2023, she exhibited total scalp and body hair loss (Figure 1A), consistent with AU. Given the temporal association with COVID-19 and the absence of other known triggers, post-COVID-19 immune dysregulation was suspected as the underlying mechanism.[4,9]

FIGURE 1. (A) Diffuse hair loss involving the entire scalp and eyebrows prior to 4 mg baricitinib treatment. (B) Significant hair regrowth involving the scalp and eyebrows after 10 months of 4 mg baricitinib treatment.



Initial treatments with topical clobetasol, fluocinolone solution, and intralesional triamcinolone injections over 3 months yielded no improvement. In April 2023, systemic baricitinib 4 mg daily therapy was initiated after a lack of response to topical therapy. Throughout the treatment course, she demonstrated strict adherence to the regimen. By February 2024, she had significant regrowth of scalp and eyebrow hair growth after 10 months of therapy (Figure 1B). However, 3 months after discontinuation in February 2024, diffuse hair loss recurred.

DISCUSSION

This case underscores post-COVID-19 AU as a distinct autoimmune sequela, differentiating it from idiopathic AU and post-viral telogen effluvium. Unlike TE, which resolves spontaneously, AU is chronic, immune mediated, and often relapsing.[1,2,10] Its rapid onset following COVID-19 and relapse after therapy discontinuation support the hypothesis that post-COVID-19 immune dysregulation contributes to AU pathogenesis and may require long-term treatment approaches.[4,9,10]

One of the key challenges in managing post-COVID-19 AU is disease relapse after treatment discontinuation. While JAK inhibitors and other immunomodulatory agents show promise in treating AU,[11-15] the recurrence of hair loss upon discontinuation raises concerns about long-term disease control. Post-COVID-19 AU may exhibit a unique disease course compared to idiopathic AU, possibly requiring longer treatment durations due to persistent post-viral immune activation.[10,15]

CONCLUSION

This case highlights post-COVID-19 AU as an emerging autoimmune phenomenon, distinct from idiopathic AU and requiring unique considerations for management. The high relapse rate post-treatment suggests that COVID-19 may induce prolonged immune dysregulation affecting hair follicle immune privilege. Given the growing recognition of post-viral AU, defining optimal treatment protocols, comparing relapse rates in idiopathic vs. post-viral AU, and evaluating the necessity of maintenance therapy will be crucial for guiding long-term clinical management.

DISCLOSURES

The authors have no conflicts of interest to disclose.

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