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New-onset alopecia areata during calcitonin gene-related peptide inhibitor therapy: first reported case

Luca Valtellini,^{1,2} Mauro Barbareschi,² Silvia Mariel Ferrucci,¹ Angelo Valerio Marzano,^{1,2} Andrea Sechi¹

¹Dermatology Unit, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan;

²Pathophysiology and Transplantation Department, University of Milan, Italy

Correspondence: Andrea Sechi, MD, Dermatology Unit, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Via Pace 9, 20122 Milan, Italy. E-mail: andrea.sechi@policlinico.mi.it; Tel.: +390255031

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Ethics approval and consent to participate: this retrospective review of patient data did not require ethical approval in accordance with local guidelines. Informed consent was obtained from the patient included in this study.

Consent for publication: the patient gave her written consent to use her personal data for the publication of this case report and any accompanying images.

Availability of data and materials: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Dear Editor,

Calcitonin gene-related peptide (CGRP) is a neuropeptide with potent vasodilatory effects and a key role in trigeminovascular signaling, underlying the pathogenesis of migraine. Consequently, the inhibition of CGRP activity has been demonstrated to reduce the incidence of acute migraine attacks. Based on this evidence, monoclonal antibodies against CGRP or its receptor have been approved by the European Medicines Agency for prevention of both episodic and chronic migraine. Among CGRP inhibitors, galcanezumab has also been authorized for the treatment of episodic cluster headache.¹ Since the advent of CGRP inhibitors, several cases of drug-induced telogen effluvium have been reported, with incidence rates ranging from 1.10% to 7.23%.^{1,2} However, to the best of our knowledge, alopecia areata (AA) associated with CGRP inhibition remains undescribed in the literature.

Herein, we present the first reported case of new-onset AA during CGRP inhibitor therapy.

In March 2025, a 54-year-old woman was referred to our dermatology department with alopecia universalis (Figure 1). AA developed in May 2024 and was unresponsive to both systemic and topical corticosteroid therapy. The patient had no personal or family history of AA or other autoimmune diseases. She had started treatment with galcanezumab for chronic migraine approximately 4 months prior to the onset of hair loss. The drug was stopped in July 2024 due to the development of alopecia; however, no clinical improvement was observed with discontinuation. Trichoscopic examination revealed diffuse yellow dots on the scalp, consistent with a diagnosis of chronic-phase AA. The patient was subsequently started on ritlecitinib 50 mg once daily.

Although most literature on CGRP inhibitor-associated hair loss focuses on telogen effluvium, evidence supports a potential link between CGRP receptor antagonism and AA. CGRP is proposed to influence the hair growth cycle by upregulating insulin-like growth factor 1, a key regulator of hair follicle development through its role in cellular proliferation and migration.³ Moreover, CGRP has also been involved in maintenance of hair follicle immune privilege (IP), particularly during the anagen phase, when follicles are most susceptible to immune-mediated damage. CGRP has been reported to prevent interferon-gamma-induced IP collapse by downregulating the expression of major histocompatibility complex class I and II molecules and inhibiting perifollicular mast cell degranulation.⁴ Reduced CGRP levels have been observed in intracutaneous sensory nerve fibers of lesional AA skin and in the serum of AA patients.^{4,5} These findings are not epiphenomenal but may reflect impaired immunoregulatory function of CGRP in AA patients.⁴ Notably, CGRP appears capable of preventing, but not reversing, experimentally induced

follicular IP collapse.⁴ This may potentially explain the lack of therapeutic response following galcanezumab discontinuation in our patient.

Further research is needed to elucidate the exact mechanisms by which pharmacologic CGRP inhibition may trigger AA and to establish appropriate management strategies for affected patients.

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Figure 1. a, b, d, e) Clinical and d, f) trichoscopic presentation of alopecia areata in the reported patient.

