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Review Article

Alopecia Areata Associated with Dupilumab: A Case Report and Literature Review

Sehar Nadeem[1], Humaira Nfn[2], Jodi Yanking Li[3], Vignesh Ramachandran[4,5*]

[1]Department of Psychology, Brooklyn College, Brooklyn, NY, USA
[2]Department of Biology, Macaulay Honors College, Brooklyn College, Brooklyn, NY, USA
[3]Department of Psychology, New York University, New York, NY, USA
[4]Skin Institute of New York, New York, NY, USA
[5]Ronald O. Perelman Department of Dermatology, New York University, New York, NY, USA
***Correspondence author:** Vignesh Ramachandran, MD, Department of Dermatology, New York University, Skin Institute of New York, USA; Email: vig.ramachandran@gmail.com

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Abstract

Alopecia Areata (AA), an autoimmune condition characterized by non-scarring hair loss and Atopic Dermatitis (AD) share T-helper cell type 2 (Th2)-mediated inflammatory pathways. Dupilumab, a monoclonal antibody targeting IL-4 and IL-13 signaling, is effective for AD but has been paradoxically associated with AA onset. We report a case of a 47-year-old female with AD, asthma and environmental allergies who developed AA (SALT score 45%) after six months of dupilumab therapy. Histopathology confirmed AA, likely triggered by dupilumab, which was discontinued. Treatment with pulse-dose dexamethasone, baricitinib, topical clobetasol and intralesional kenalog led to complete hair regrowth within 12 weeks. This case highlights a rare adverse effect of dupilumab, possibly due to disrupted follicular homeostasis or enhanced Th1/Th17 pathways. Clinicians should monitor for AA in patients on dupilumab, particularly those with atopic or autoimmune predispositions. Further research is needed to elucidate mechanisms and identify risk factors.

Keywords: Alopecia Areata; Dupilumab; Atopic Dermatitis; Adverse Effect

Introduction

Alopecia Areata (AA) is a common autoimmune condition characterized by non-scarring hair loss, often presenting as discrete, smooth patches on the scalp or other hair-bearing areas. It is driven by a complex interplay of immune dysregulation, predominantly involving T-helper cell type 2 (Th2)-mediated inflammation, with Interleukin (IL)-4 and IL-13 playing critical roles in its pathogenesis [1]. Atopic Dermatitis (AD), another Th2-driven disorder, can coexists with AA, perhaps due to their sharing similar immunological pathways [2]. Dupilumab, a monoclonal antibody that inhibits IL-4 and IL-13 signaling by targeting the IL-4 receptor alpha subunit, has revolutionized the management of moderate-to-severe AD by effectively reducing Th2-mediated inflammation [3]. This treatment has been markedly successful in dermatology and AD is one of the most commonly treated conditions by dermatologists. Furthermore, dupilumab is also indicated in the management of other Th-2-mediated pathologies, such as chronic rhinosinusitis, eosinophilic esophagitis, nasal polyps, chronic spontaneous urticaria and prurigo nodularis. As such, its use in medicine has continued to expand and additional new indications are expected for conditions such as bullous pemphigus. Given the shared Th2-driven mechanisms, dupilumab has been hypothesized to benefit AA, with case reports documenting its potential to induce hair regrowth in some patients with AA, including those with concurrent AD [4-6]. However, in our outpatient dermatology clinic, we were presented with a patient who we believe had AA triggered by dupilumab. We present this case herein, its implications, postulate mechanisms of action and future directions for research.

Methods and Materials

A 47-year-old healthy female with a history of eyelid swelling was presented to our dermatology clinic with chief complaint of acute hair loss. Initial history revealed that she was told by her primary care physician that her years-long history of eyelid swelling may be part of her atopic diathesis, which included her diagnosis of asthma and environmental allergies. After failing months-long treatments with hydrocortisone 2.5% cream, betamethasone dipropionate 0.05% cream and oral prednisone (40 mg for 1 week), she was started on dupilumab (600 mg subcutaneous loading dose followed by 300 mg every 14 days). However, her eyelid symptoms did not improve despite 6 months of every two-week injection of dupilumab. Her asthma symptoms and environmental allergies did markedly improve. At month 6, she began noticing sudden shedding of hair in "clumps". She had initially attributed this to weather changes, but when the rapidity and extent of hair loss progressed, she stopped the dupilumab and came to our dermatology clinic as a referral from her primary care physician. On evaluation, she was noted to have large, smooth, alopecic patches. Her SALT score was approximately 45% with a positive hair pull test (Fig. 1). A punch biopsy from the right superior parietal scalp was performed, revealing an infiltrate of lymphocytes at the base of catagen and telogen hair follicles. A PAS-D stain fails to reveal fungal organisms. This confirmed a diagnosis of Alopecia Areata (AA), likely triggered by dupilumab. Our patient was managed with multimodal therapy. First, dupilumab was held as it was not improving her eyelid swelling symptoms. She was referred to ophthalmology for this condition. She was started on pulse dose oral dexamethasone twice weekly at a dose of 0.1 mg/kg/day for 8 weeks, 4 mg daily of baricitinib daily, topical clobetasol 0.05% solution twice daily and intralesional kenalog (2.0 mg/cc concentration) every other week. After 12 weeks of treatment, she had marked improvement with complete resolution of her AA (Fig. 2) and baricitinib was stopped. At this time, clobetasol 0.05% solution was decreased to every other day and intralesional kenalog treatments were also stopped.

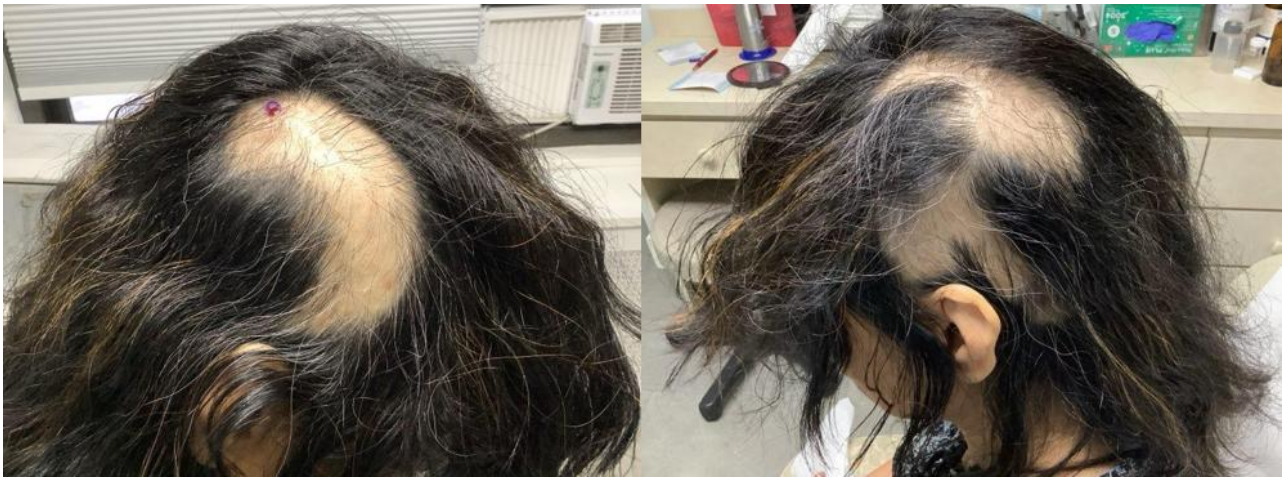

Figure 1: A-B: Discrete alopecic patches were noted on initial examination of approximately 45% SALT score.

Figure 2: Marked improvement with complete regrowth of hair.

Case (year)	Age/Sex	Site	Dupilumab Indication	Comorbidities	Onset	Treatment	Management/Outcome
Flanagan, et al., (2019) [6]	27 years old/ male	AA on scalp (vertex and temporal region)	Moderate AD	N/A	18 weeks	Loading dose of 600 mg, dupilumab, 300 mg subcutaneousl AA - intralesional triamcinolone and clobetasol	Complete regrowth 2 months after Dupilumab discontinued
Gallo. (2020) [7]	24 years old/ male	Entire scalp	Severe AD	Ulcerative colitis	8 weeks	Loading dose of 600 mg, dupilumab, 300 mg subcutaneously AA - dupilumab was discontinued. Topical clobetasol cyclosporine, 3 mg/kg/day	Partial hair regrowth within 4 weeks. After 3 months, hair regrowth was complete
Kulkarni, et al., (2022) [8]	22 years old/ male	Patchy AA on vertex scalp	Moderate AD	N/A	3-4 months	Initially 300 mg Q2W, later switched to 300 mg Q4W AA - oral/topical steroids + mycophenolate	Complete regrowth 5-6 months after Dupilumab discontinued. No relapse of AA after Dupilumab reintroduced with lower dose
Mitchell & Levitt (2018) [9]	29 years old/ male	Patchy AA on scalp	Chronic AD	N/A	5 weeks	300 mg Q2W AA - intralesional triamcinolone (10 mg/mL)	Partial regrowth
Carnicle, et al., (2021) [10]	42 years old/ female	Reactivation of AA (diffuse, androgenetic-like pattern)	Severe AD	AA in remission for >5 years	4 months	Not specified AA - 1 dose IM triamcinolone	Complete regrowth 2 months after Dupilumab discontinued
Chromy, et al., (2023) [11]	36 years old/ male	AA of beard	Chronic rhinosinusitis with nasal polyps (No AD)	N/A	25 weeks	Not specified	N/A
Barroso-Garcia, et al., (2018) [12] Salgüero-Fernández, et al., (2018) [13]	31 years old/ male	AA in patches on the anterior scalp	Severe AD	N/A	6 weeks	Initially 600 mg followed by 300 mg every 2 weeks AA - intralesional triamcinolone	N/A
Yazdanyar S, et al., (2019) [14]	33 years old/ male	Diffuse AA in the frontal and occipital region + beard	Severe AD	N/A	7 weeks	Initially 600 mg followed by 300 mg every 2 weeks AA - topical mometasone 0.1%	Complete hair regrowth after 3 months

Barbarin C, et al., (2019) [15]	24 years old/ male	AA in patches	AD (unspecified severity)	N/A	1 week	Initially 600 mg followed by 300 mg every 2 weeks AA - ketoconazole 3% shampoo	Partial regrowth after 3 weeks
Kanda N, et al., (2019) [16]	23 years old/ female	AA in patches in the frontal, vertex and occipital areas	Chronic AD	N/A	48 hours	Initially single 600-mg dose, stopped after 8 weeks of treatment AA - topical minoxidil and clobetasol propionate	Complete regrowth after 6 months
Stander, S et al., (2020) [17]	35 years old/ male	AA in patches, mostly in the parietal, occipital and frontal regions	Severe AD	N/A	6 weeks	Initially 600 mg followed by 300 mg every 2 weeks AA - methylprednisolone (0.5g/day) for 3 days	Partial regrowth (78%) after 4 months
Beaziz J, et al., (2021) [18]	53 years old/ male	AA in patches	Recurrent AD	N/A	1 year	Initially 600 mg followed by 300 mg every 2 weeks AA - dupilumab was discontinued and cyclosporine 200 mg daily	Complete regrowth after 4 months
Chung J, et al., (2019) [19]	45 years old/ female	AA in oval patches in the occipital region + two small patches in the temporal region	Severe AD	asthma and allergic rhinitis	1 year	Initially 600 mg followed by 300 mg every 2 weeks AA - clobetasol propionate 0.05%	Complete hair regrowth after 2 months
Chung J, et al., (2019) [19]	51 years old/ female	AA in patches	Chronic AD	At 26 month, pt noted generalized thinning of hair, suspected for iron deficiency-induced telogen effluvium (no improvement after successful iron supplementation)	28 months (AA patches first appeared)	24 months of open-label dosing in clinical trials of dupilumab , then transitioned to commercial dosing of dupilumab, 300 mg every 2 weeks	At 30 month, hair loss progressed to AU (alopecia universalis) At 34 month, pt resumed dupilumab monthly shot, 90% regrowth of scalp hair at 44 month.
Zhu. (2020) [20]	25 years old/ male	Alopecia universalis, loss of his few remaining patches of scalp hair	Chronic AD	N/A	6-8 weeks	AA - Dupilumab was discontinued. Cyclosporine, tofacitinib	Patchy improvement of alopecia totalis within 6 weeks of treatment

Zhu. (2020) [20]	37 years old/ male	Right jawline localized non- scarring alopecic patch	AD (unspecified severity)	N/A	5 weeks	Dupilumab 300 mg Q2W AA - Topical calcineurin inhibitors	No follow-up visit
	37 years old/ female	Scalp, occipital localized alopecic patch	AD (unspecified severity)	Pre-existing Alopecia areata	N/A (Prior resolved alopecia, recurrent on dupilumab)	300 mg Q2W AA-topical steroids	Continued Dupilumab, stable disease

Table 1: Literature review and summary of cases illustrating alopecia areata as an adverse event in the setting of dupilumab use.

Discussion

The acute onset of AA in our patient following dupilumab therapy for AD illuminates a rare but significant adverse effect that complicates the use of this monoclonal antibody in Th2-driven diseases. Dupilumab's efficacy in AD stems from its inhibition of IL-4 and IL-13 signaling, which mitigates Th2-mediated inflammation [3]. However, our case suggests that this intervention may, in certain patients, disrupt immune or follicular homeostasis, precipitating AA. This paradoxical outcome highlights the intricate interplay of cytokine networks in atopic and autoimmune conditions and underscores the need for heightened clinical awareness when prescribing dupilumab.

Our patient's presentation, marked by a SALT score of 45%, a positive hair pull test and histopathologic confirmation of AA, aligns with a small but growing body of reports describing dupilumab-associated AA (Table 1) [7-9]. The temporal association between dupilumab initiation and hair loss, with symptoms emerging after 6 months of therapy, suggests a drug-induced etiology. Our literature review noted in Table 1 highlights the wide-ranging time course for onset of AA in the setting of dupilumab initiation, highlighting the need for heightened clinical awareness of this possible etiology. The underlying mechanism remains speculative but may involve dupilumab's impact on sebaceous gland function, as IL-4 and IL-13 regulate lipid production critical for hair follicle health [8]. By blocking these cytokines, dupilumab could disrupt the follicular microenvironment, leading to non-scarring hair loss. Alternatively, the suppression of Th2 pathways may enhance Th1 or Th17-driven inflammation, which is implicated in some studies on the pathogenesis of AA [1]. These hypotheses merit further exploration to clarify the immunological and follicular dynamics at play.

Clinically, this case has several implications. First, dermatologists and allergists should consider AA as a potential adverse effect when prescribing dupilumab, particularly for patients with a history of atopy or autoimmune predispositions. This is particularly noteworthy given the proliferation of the use of dupilumab in clinical medicine and AA not being listed as a side effect on the drug label. Furthermore, ongoing clinical trials are attempting to assess the efficacy of dupilumab for AA (NCT05551793). This highlights the need to ensure the medical literature reflects the growing body of evidence of AA being induced by dupilumab itself.

Routine scalp examinations and patient education about hair loss symptoms could facilitate early detection. Second, our patient's marked improvement following dupilumab discontinuation and multimodal therapy—pulse-dose dexamethasone, baricitinib, topical clobetasol and intralesional Kenalog-demonstrates the potential for reversibility. Nevertheless, the medication was held not only in our patient's case but also in several reports in the literature. A comprehensive risk and benefit discussion should be had with patients who develop AA in the setting of dupilumab use given the myriad of treatment options available in the AA landscape. The rapid regrowth, with complete scalp coverage after 12 weeks, suggests that prompt intervention can restore hair follicle function, offering reassurance to clinicians managing similar cases. Notably, the lack of response to dupilumab for eyelid swelling prompted referral to ophthalmology, highlighting the importance of addressing treatment failures holistically.

This case also raises questions about patient selection for dupilumab therapy. While dupilumab has shown promise in AA treatment for some patients, as evidenced by clinical trials and case reports, our findings emphasize the need to identify risk factors for adverse outcomes [4-6]. Factors such as baseline cytokine profiles, genetic predispositions or concurrent autoimmune

conditions may influence susceptibility to dupilumab-induced AA. Future research should prioritize biomarker studies to stratify patients and mechanistic investigations, such as *ex-vivo* scalp tissue analysis, to elucidate how dupilumab alters hair follicle biology. Additionally, comparative studies evaluating alternative biologics or JAK inhibitors in patients with AD and AA could guide therapeutic strategies.

Conclusion

In conclusion, this case underscores the dual nature of dupilumab as a transformative therapy for AD and a potential trigger of AA in susceptible individuals. By advocating for vigilant monitoring, tailored treatment approaches and targeted research, clinicians and researchers can better navigate the complexities of dupilumab therapy, ensuring optimal outcomes for patients with atopic and autoimmune conditions.

Conflicts of Interest

The authors declare no conflict of interest in this paper.

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