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LETTER |  Full Access

Dupilumab-induced psoriasis in a patient with atopic dermatitis and alopecia totalis: A case report and literature review

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This manuscript is original and has not been published before nor is it under consideration for publication elsewhere.

Dear Editor,

Dupilumab, a monoclonal antibody that targets IL-4 and IL-13 receptors, downregulates the T-helper-2 cell (Th2) inflammatory response. Atopic dermatitis (AD) is Th2-mediated with IL-4 and IL-13 predominance.¹ Dupilumab is approved for moderate-to-severe adult AD.

While dupilumab clinical trials had few adverse events, recent literature has reported dupilumab-induced psoriasis, primarily in AD patients.²⁻¹² Herein, we present a unique case of dupilumab-induced psoriasis in a patient with both AD and alopecia totalis (AT) and a review of the literature.

After 5 months of dupilumab for AD, a 28-year-old woman with severe AD since infancy and AT presented with micaceous, erythematous scalp and body plaques.

Patient had no psoriasis personal or family history. History included 4 years of AA with recent progression to AT without significant response to scalp corticosteroid topicals or injections. AD was recalcitrant to topical steroids and UV-light-therapy. Patient had no asthma or other atopic disease history. She was started on dupilumab (600 mg initial dose followed by 300 mg every 14 days) and had nearly complete AD and AT resolution after 2 weeks and 2–3 months, respectively.

On exam, erythematous, scaly plaques were present on the abdomen and proximal extremities and covered the scalp but spared two areas of persistent alopecia. Her exam was clinically consistent with psoriasis without active AD. The patient declined scalp biopsy.

The patient discontinued dupilumab and started clobetasol for psoriasis, which completely resolved within 6 weeks. After another 4 months, patient reported no psoriasis or worsening hair loss. Her AD was mild and managed topically. Patient provided informed consent for this publication.

Acute AD is primarily Th2-driven before partially shifting to T-helper-1 (Th1)-mediated in chronic AD.¹³ Psoriasis is a Th1 and T-helper-17 (Th17) cellular-mediated disease.¹ The two diseases, therefore, are considered opposing ends of the Th1 and Th2 spectrum.¹⁴

While IL-4 directly mediates AD Th2 pathways, it restricts Th1 pathways in psoriasis. Specifically, IL-4 inhibits epidermal IL-1b and IL-6 secretion from psoriatic plaques and negatively regulates Th1 and Th17 cells.¹³ By blocking IL-4, dupilumab may shift the AD Th2 pathway toward Th1-mediated psoriasis. Tracey et al.³ originally reported dupilumab-induced psoriasis and postulated this theory. Seventeen additional cases including ours have since been reported (Table 1).^{1, 3-13} Only 11.1% of cases had confirmed personal or family history of psoriasis.

TABLE 1. Cases of dupilumab-induced psoriasis in the literature including the currently presented case (#18)

Case	Age	Gender	Atopic dermatitis (AD)/alopecia areata (AA) history	Age AD/AA onset	Personal or family history of psoriasis	AD dupilumab response	Time of psoriasis onset following dupilumab start	Psoria involve
1	50s	F	AD versus un-diagnosed psoriasis	Adult	Unknown	Progression of potentially un-diagnosed psoriasis	~60 days	Erythr
2	55	M	AD	Infant	None	Significant improvement	60 days	Trunk
3	40	F	AD	2 years old	None	Complete remission	98 days	Trunk, extrem
								Guttate psorias trunk a extrem

Case	Age	Gender	Atopic	Age	Personal or	AD	Time of	Psoria

Of the 18 total reported cases, dupilumab was prescribed for AD in most (94.7%). Of those reporting dupilumab response (12 of 18), 100% had improved AD.

In the remaining non-AD case, dupilumab was used off-label for recalcitrant AT.¹¹ Both AA hair loss and regrowth have been reported on dupilumab, with regrowth primarily in females with severe AD since childhood and other atopic comorbidities, potentially due to stronger Th2 skewing.¹⁵ Our case fits this pattern with alopecia persisting only in areas without psoriasis. We hypothesize that IL-4 blockade was unsuccessful in these areas. Dupilumab was unable to block the Th2-skewed AA or to further shift the local immunologic milieu toward Th1-mediated psoriasis.

Most patients (83%) with reported dupilumab-induced psoriasis had AD since childhood. As chronic AD has a partial Th1 shift, dupilumab may more easily shift the paradigm toward Th1-mediated psoriasis in these patients.^{7, 8} In the single case of dupilumab for adult-onset AD, psoriasis may have been misdiagnosed prior to dupilumab start, which would explain the patient's erythrodermic psoriatic response.³

Dupilumab was discontinued due to psoriasis in 44.4% of reported cases. Most psoriasis cases (94.7%) were well managed topically including those with continued standard-dosing dupilumab.

Dermatologists should be aware of psoriasis as a potential dupilumab adverse event, the risks of dupilumab in patients with psoriasis history, and the ability to topically treat psoriasis while continuing dupilumab.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

Kelly E. Flanagan: Conceptualization (supporting), methodology (equal), formal analysis (lead), writing – original (lead), writing – review and editing (equal). **Isabel M. Pupo Wiss:** Formal analysis (supporting), writing – original (supporting), writing review and editing (equal). **James T. Pathoulas:** Methodology (equal), formal analysis (supporting), writing – original (supporting), writing review and editing (equal). **Chloe J. Walker:** Formal analysis (supporting), writing review and editing (supporting). **Maryanne M. Senna:** Conceptualization (lead), resources (lead), supervision (lead), writing – review and editing (equal).

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DATA AVAILABILITY STATEMENT

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

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