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An Unusual Case of Psoriasiform Dermatitis Treated With Dual Biologic Therapy and Literature Review

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ABSTRACT

Previously believed to be of distinct immunopathogenesis, atopic dermatitis (AD) and psoriasis (PsO) spectrum may permit immunologic shifts to favor the opposing inflammatory states following biologic treatment. Cases of AD that developed following PsO biologics are increasingly reported in the literature. While biologic monotherapy is becoming more widely available, dual biologic therapies have been understudied. Here, we present a patient with biopsy-confirmed PsO who developed psoriasiform spongiotic dermatitis following anti-IL-17A initiation who responded to combination anti-IL-23 and anti-IL-4Ra therapy and discussed the PsO-AD immunologic spectrum and use of dual biologic therapies.

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INTRODUCTION

Psoriasis (PsO) and atopic dermatitis (AD) immunopathogeneses are canonically driven by 2 distinct T-cell-mediated responses. In psoriasis, Th1/Th17 pathways mediated by interleukin (IL)-23/IL-17 cell-mediated signaling, while AD is marked by Th2/Th22 predominant humoral inflammatory response, eliciting IL-4, IL-13, and IL-22.[1] However, certain AD subtypes (eg, intrinsic, pediatric, Asian adults) demonstrate evidence of Th2, Th17, and Th22 co-expression, resulting in a more psoriasiform phenotype.[2] Several cases have also been reported of exacerbations following "unilateral" suppression of this spectrum, with cases of paradoxical reactions in which patients with psoriasis develop eczematous eruptions after initiating anti-IL-17/IL-23 biologics and psoriasiform dermatitis after AD biologics.[3-5] These examples support the idea of a potential PsO-AD spectrum.

Here, we present a case of psoriasiform spongiotic dermatitis in a patient with a long-standing history of biopsy-proven psoriasis and resolution with dual biologic therapy (DBT). We also examine the PsO-AD immunopathogenic spectrum and relevant implications for long-term care, sequelae, and adverse events (AEs).

CASE REPORT

An 80-year-old black veteran with a 20-year history of severe psoriasis (body surface area [BSA] range: 10-75%+), who had failed multiple treatments (Table 1), now on secukinumab, with previously treated hepatitis B (HBV) and C (HCV) and gout was urgently referred to our clinic. He presented with 4 days of severely pruritic papulosquamous plaques on the upper back and bilateral lower extremities, with some showing significant impetiginization (Figure 1). For the past year (since 4/2021), he had been on secukinumab 300 mg every 3 weeks (increased from every 4 weeks due to frequent "exacerbations"), but reported missing his last dose. The patient also self-discontinued concurrent topical therapies (eg, clobetasol and calcipotriene) 2 days prior to presentation. A review of systems was otherwise negative, and he denied any new medications or recent illnesses.

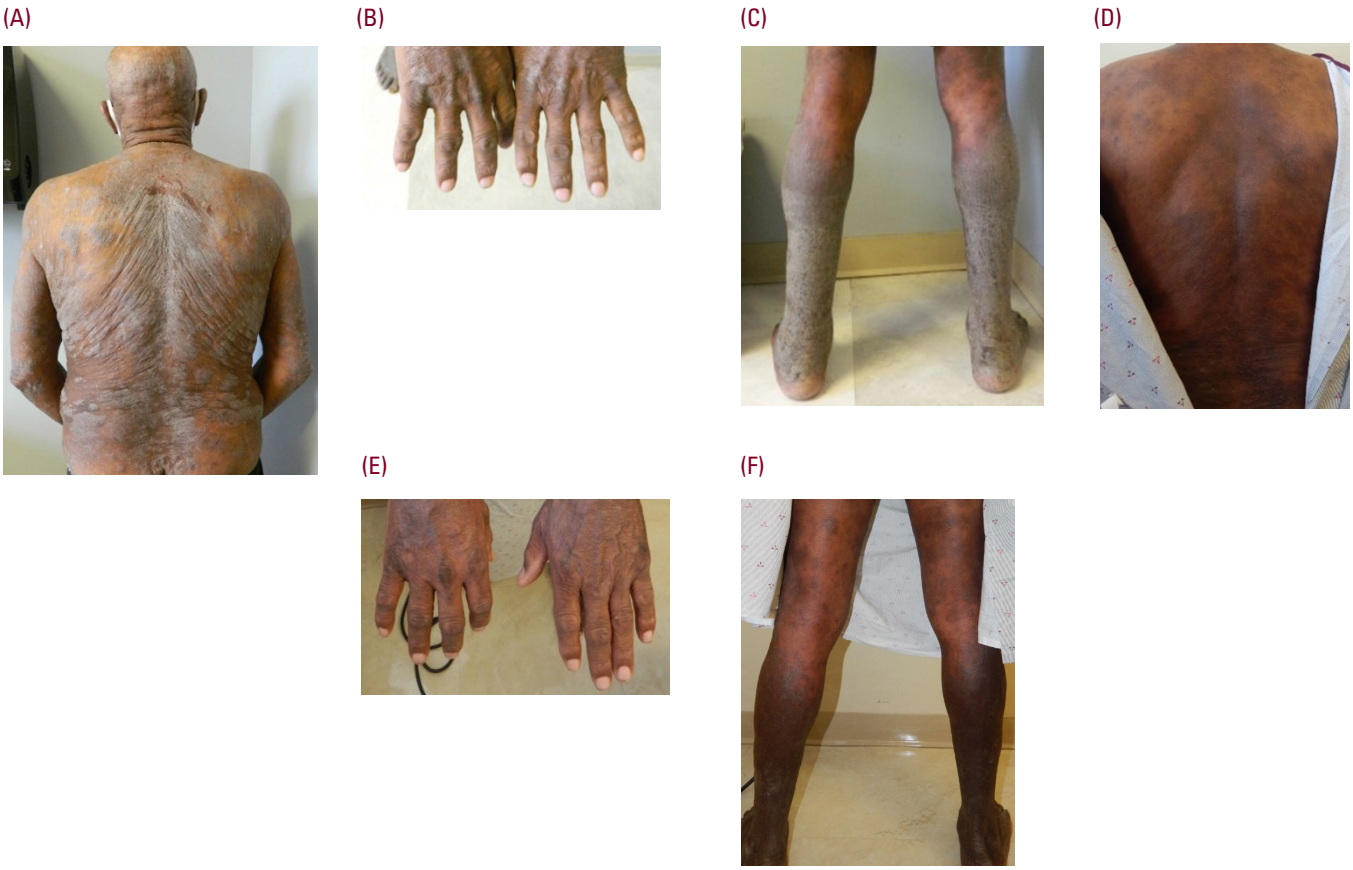
On physical examination, there were diffuse eczematous plaques with thick scale and excoriations on the trunk, buttocks, and bilateral arms/legs (~80% BSA; Figures 1A-C). Labs were notable for peripheral eosinophilia in June 2021 (relative: 20.9%, absolute: 0.98x10^3 cells/μL [normal: 0-0.45x10^3 cells/μL]) and October 2022 (relative: 18.7%, absolute: 0.71x10^3 cells/μL). Biopsies from March/October 2005 and January 2011 (prior biologics) demonstrated classical psoriasis (Figures 2A-C);

TABLE 1.

Psoriasis Treatment Timeline	
Treatment	**Year**
Topical Corticosteroids: Hydrocortisone Triamcinolone Clobetasol Fluocinonide	2005-present
Other Topicals: Calcipitriol Coal Tar Shampoo	2005-present
Narrowband UVB	2007-2008 2010-2018
Acitretin (10-25 mg daily depending on lab values)	2011-2012, stopped due to leukopenia 2012 - restarted due to flare but stopped because of leukopenia and hyperlipidemia 2013-7/2016
Etanercept	7/2016-9/2018, improvement to <5% BSA but then worsening BSA when switched to maintenance dose
Adalimumab	9/2018-8/2020, restarted 11/2020 but stopped due to recurrent flares up to 30-75% BSA with pruritus
Secukinumab	4/2021- improvement to <5% but flares starting around 2/2022 reaching 60-70% BSA in 8/2022 with significant pruritus

BSA, body surface area.

FIGURE 1. Psoriasiform dermatitis before/after treatment. (A-C) October 2022 initial presentation showing widespread erythematous, eczematous, scaly plaques (BSA~80%). (D-F) February 2023 after 4 months of dupilimab and 2 months of guselkumab with post-inflammatory hyperpigmented patches and a few scattered erythematous scaly plaques (BSA<10%).



however, a biopsy of the right posterior auricular scalp in November 2016 (on etanercept) and posterior neck on August 2021 (on secukinumab) showed psoriasiform dermatitis with eosinophils (Figures 3A-D).

Given the severity of the patient's presentation with near-erythroderma, lodging accommodations were made for admission to the Veterans Affairs "hoptel" to start daily triamcinolone applications and restart narrowband-UVB (given prior improvement phototherapy) while awaiting new biologic therapy initiation. He was started on dupilumab 300 mg subcutaneously every 2 weeks following an initial 600 mg loading dose and guselkumab 100 mg subcutaneously every 8 weeks after the weeks 0 and 4 loading doses. On 4-month and 1-year follow-up, there was significant improvement (Figures 1D-F) in pruritus, plaque severity, and scaling (BSA<10%).

DISCUSSION

Although taught as distinct immunological entities, clinical ambiguity and response to DBT challenge the mantra that PsO/AD are mutually exclusive. Studies/reports suggest that biologic suppression of one immunopathologic pathway may instigate a potential Th1/Th2 shift to the opposite extreme of a "PsO-AD" spectrum.[5] Several cases of eczematous eruptions following PsO biologics have been reported, most commonly with secukinumab, as seen with our patient described herein.[5]

Reports suggest that biologics targeting IL-17A(eg, secukinumab) allow other IL-17 cytokines(eg, IL-17C and IL-17E) found in both PsO and Th2-mediated AD inflammation to persist, promoting this paradoxical eczematous eruption.[6] Anti-TNF-α inhibitors for psoriasis (ie, adalimumab/etanercept) have also been reported to induce eczematous reactions.[7] Interestingly, while our patient was found to have eosinophils on biopsy (Figures 3A-D) while on etanercept, he only later developed an exuberant eczematous eruption and peripheral eosinophilia several months following secukinumab initiation.

Conversely, the development of psoriasis/psoriasiform dermatitis following dupilumab-mediated IL-4/IL-13 inhibition may allow Th1 inflammatory profile to predominate over a suppressed Th2 pathway.[8] The clinical course of many of these cases reported by Turowski et al[8] resulted in discontinuation of the biologic and initiation of topical steroids or a systemic agent like methotrexate (avoided in our patient, given history of HBV and HCV) or apremilast.

Only a few cases have described the use of DBT for concomitant PsO and AD (Table 2). Similar to other reports, our patient has had a continued sustained response (~1 year) on DBT (dupilumab and guselkumab) without AEs.[4,9] Other approaches to patients with mixed PsO-AD presentation may include the use of Janus kinase (JAK) inhibitors, such as the selective JAK1 inhibitor

FIGURE 2. Biopsy of psoriasis taken from the abdomen in 2005. Demonstrating parakeratosis with neutrophils, diminished granular layer, acanthosis, and thinning of the suprapapillary plates (H&E) (A) 100x (B) 400x (C) Parakeratosis with neutrophils (H&E, 400x).

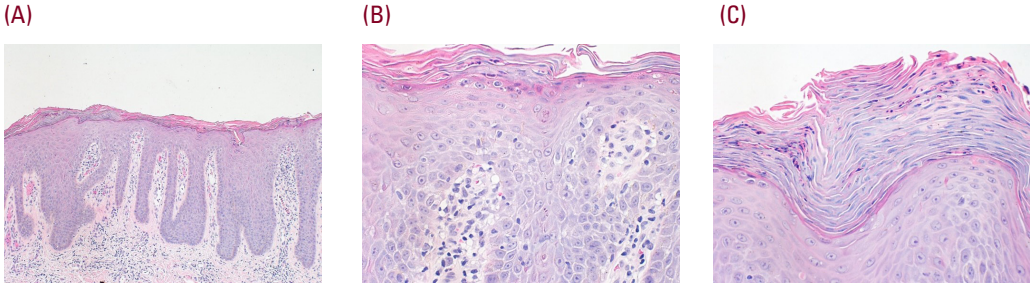
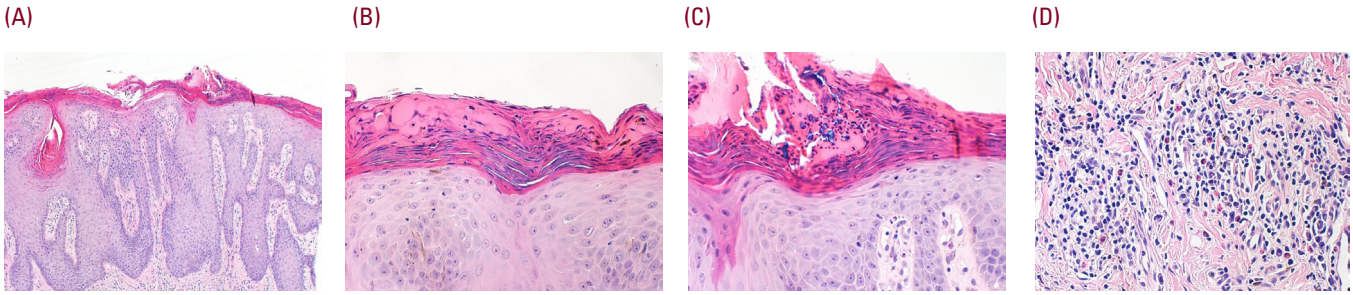


FIGURE 3. Biopsy of chronic psoriasiform spongiotic dermatitis with impetiginization and eosinophils taken from the right posterior auricular in 2016. (A) irregular psoriasiform hyperplasia (H&E, 100x) (B) serum in the corneal layer (H&E, 400x) (C) clusters of bacterial cocci (H&E, 400x), and (D) numerous eosinophils in the dermis (H&E, 400x).



upadacitinib.[10] These cases achieved complete clearance or improvement in subjective symptoms by week 16 without AEs and follow up at week 32.[10] An important consideration with the use of JAK inhibitors is its box warning for increased risk of venous thromboembolisms (VTE) and major adverse cardiovascular events (MACE). Although caution should be applied to those at risk (eg, prior/current tobacco use, increased cardiovascular risk factors, hormonal contraceptives, or a history of malignancies), long-term safety studies demonstrate these selective inhibitors to be generally well-tolerated and safe.[11]

As a final aside, while not yet routinely performed, personalized medicine has demonstrated the ability to identify patients' key driving biomarkers in inflammatory processes and may have promise in targeting efficient and effective treatment options.[12,13] Several initiatives in recent years have also focused on using gene-expression profile testing to pinpoint pathways upregulated in psoriasis and AD disease processes.[14] These diagnostic advancements may help to provide targeted treatment for patients.

TABLE 2.

Cases of Dual Biologic or Janus Kinase Inhibitor Therapy for Concomitant Psoriasis and Atopic Dermatitis

	Case	Treatment and Dose	Length of Treatment	Average Follow-up Duration	Time to Clearance or Significant Improvement	Adverse Event(s)
Dual Biologic Therapies	80-year-old male with 20-year history of PsO	Guselkumab 100 mg every 8 weeks (after loading dose on weeks 0 and 4)	9 months	5.5 months	9 months	None
	62-year-old female with 12-year history of PsO[14]	Guselkumab±	12 months	Unknown	12 months	None
	42-year-old male with 10-year history of PsO[5]	Tildrakizumab± Dupilumab 300 mg every 2 weeks (after 600 mg loading dose)	7 months	4.6 months	12 months	None
	24-year-old female with Crohn's disease[5]	Ustekinumab 45 mg every 12 weeks Dupilumab 300 mg every 2 weeks (after 600 mg loading dose)	12 months	4.6 months	12 months	None
	54-year-old female with 5-year history of PsO and PsA[5]	Ixekizumab± Dupilumab 300 mg every 2 weeks (after 600 mg loading dose)	9 months	4.6 months	2 months	None
	Unknown[3]	Guselkumab± Dupilumab±	12 months	Unknown	12 months	Unknown
JAK Inhibitor	12-year-old male with psoriasis since early childhood9[9]	Upadacitinib 15 mg once per day	1 year	12 months	4 months	None
	39-year-old male with psoriasis since childhood[9]	Upadacitinib 15 mg once per day	36 weeks	36 weeks	4 months	None
	50-year-old female[9]	Upadacitinib 15 mg once per day	32 weeks	32 weeks	4 weeks*	None
	42-year-old female with palmoplantar PsO since 2015[9]	Upadacitinib 30 mg once per day	60 weeks	60 weeks	4 months	None

*Subjective improvement in symptoms. ±Specific dosage not reported
Mg, milligrams; PsA, psoriatic arthritis; PsO, psoriasis.

CONCLUSION

This case report adds to the growing literature on the efficacy and safety of DBT for patients with PsO-AD overlap dermatitis who have failed standard-step therapy. Given our current understanding of the "PsO-AD" spectrum, there should be increased suspicion for immunological shifts when patients on biologics present with morphologically disparate exacerbations compared with the original presentation. Future studies should elucidate this PsO-AD immunopathogenic axis, the safety/efficacy of various DBT systemic agents, and personalized medicine for long-term management of these chronic cutaneous dermatoses.

DISCLOSURES

The authors have no conflicts of interest to declare.

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