Case Presentation

New-onset erythrodermic psoriasis associated with antiepileptic drug use

James Feng, BS[1], Payal Shah, MD, MS[2a], Jeffrey Cloutier, MD, PhD[3], Jonathan S. Glass, MD[4], Dorothea T. Barton, MD[4]

[1] Geisel School of Medicine at Dartmouth, Hanover, NH, USA, [2] Department of Dermatology, University of Alabama at Birmingham Heersink School of Medicine, Birmingham, AL, USA, [3] Department of Pathology and Laboratory Medicine, Dartmouth Health, Lebanon, NH, USA, [4] Department of Dermatology, Dartmouth Health, Lebanon, NH, USA

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Abstract

Erythrodermic psoriasis is a severe and potentially life-threatening dermatologic condition that can be triggered or unmasked by medications. We report the case of a 73-year-old patient who developed new-onset erythrodermic psoriasis following treatment with antiepileptic drugs (AEDs), specifically carbamazepine and levetiracetam, for autoimmune encephalitis. Despite discontinuation of the AEDs, the patient's condition persisted and improved only after systemic biological therapy. Clinical presentation, together with histopathologic findings, supported a diagnosis of new-onset psoriasis rather than a drug-induced psoriasiform reaction. This case highlights the importance of recognizing AEDs as potential triggers for psoriasis in patients with no prior history. Given the severe complications associated with erythroderma, timely diagnosis and initiation of appropriate therapy are essential. Clinicians should maintain a high index of suspicion for medication-induced dermatologic conditions in patients presenting with new or progressive skin eruptions.

Introduction

Antiepileptic drugs (AEDs) are a diverse class of medications frequently associated with cutaneous reactions. These reactions commonly include generalized morbilliform rashes and urticaria, but more severe and potentially life-threatening eruptions can also occur. We report the case of a patient who developed new-onset erythrodermic psoriasis unmasked by commonly used AEDs, specifically carbamazepine and levetiracetam.

Case Synopsis

A 73-year-old patient with recently diagnosed LGI1 antibody–positive autoimmune encephalitis complicated by seizures was treated with a prednisone taper, 2 courses of intravenous immunoglobulin, and maintenance therapy with levetiracetam and carbamazepine. The patient subsequently presented to dermatology with a 3-month history of progressively worsening red, itchy, and burning skin lesions, now involving the entire body. There was no personal or family history of psoriasis or atopic dermatitis.

The primary care provider initially suspected a drug reaction to carbamazepine, which was discontinued first. Persistence of the eruption led to discontinuation of levetiracetam 3 weeks later. A prednisone taper starting at 60 mg initially improved the dermatitis, but symptoms reflared when the dose was reduced to 20 mg.

Physical examination revealed erythroderma with notable bilateral ectropion and confluent scaling with hemorrhagic crust on the lower extremities (**Figure 1**). The palms were hyperlinear, and there were no nail changes. The patient was afebrile and otherwise systemically well. Laboratory workup was notable for markedly elevated C-reactive protein (71 mg/L) and absolute eosinophilia (0.8 × 10^9/L). Complete blood count, metabolic panel, liver function tests, ANA, and dsDNA autoantibody testing were unremarkable. The differential diagnosis included drug reaction, psoriasis, atopic dermatitis, cutaneous T-cell lymphoma, and contact dermatitis.

A 4 mm punch biopsy of the right anterior thigh demonstrated psoriasiform hyperplasia, broad parakeratosis, mild spongiosis, markedly diminished granular layer, dilated capillaries in the superficial papillary dermis, and a mild perivascular lymphocytic infiltrate with occasional eosinophils (**Figure 2**). These findings narrowed the differential to new-onset psoriasis versus a drug-induced psoriasiform reaction.

a Corresponding Author: Payal Shah, MD, MS, Department of Dermatology, University of Alabama at Birmingham Heersink School of Medicine, 500 22nd Street South, Birmingham, AL 35233, Tel: 205-996-7546, Email: pcshah@uabmc.edu

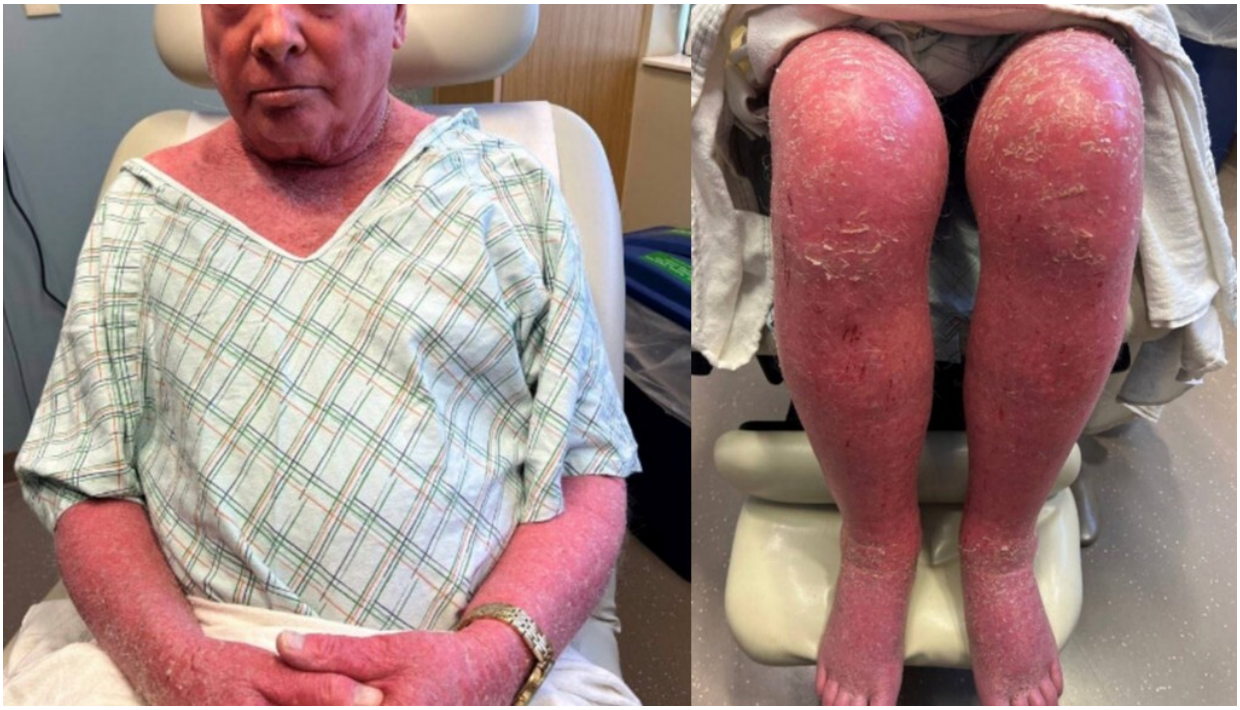

Figure 1. *Patient presenting with erythroderma, bilateral ectropion, confluent scaling, and hemorrhagic crust on the lower extremities.*

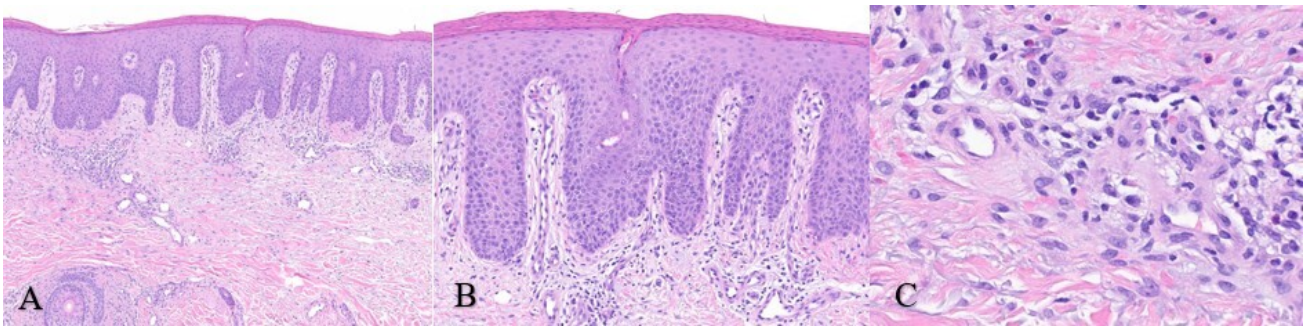

Figure 2. *Punch biopsy of the left anterior thigh showing psoriasiform hyperplasia, broad parakeratosis, diminished granular layer, dilated capillaries in the papillary dermis, and a perivascular lymphocytic infiltrate with occasional eosinophils, consistent with a psoriasiform eruption. Hematoxylin-eosin stain, original magnification:* ***(A)*** *×3.5,* ***(B)*** *×14, and* ***(C)*** *×40.*

The patient was initially treated with a prednisone taper and a 3-month course of cyclosporine (3.5 mg/kg) in combination with topical steroids and topical tacrolimus, resulting in mild improvement but persistent extensive dermatitis on the trunk and extremities. A joint decision was made to initiate systemic biologic therapy for psoriasis. The patient was started on subcutaneous risankizumab, resulting in rapid improvement within 1 month and near-complete resolution after 3 months, with no recurrence of skin lesions or seizures.

Given the persistence of symptoms after discontinuing AEDs and the rapid response to biologic therapy, the patient was ultimately diagnosed with true new-onset erythrodermic psoriasis unmasked by levetiracetam or carbamazepine, rather than a drug-induced psoriasiform reaction.

Case Discussion

AEDs are associated with a range of cutaneous reactions, occurring in approximately 2% to 8% of patients receiving these medications.[1] Most reactions are mild and transient, resolving within a few days after discontinuation. Common presentations include generalized morbilliform rashes and urticaria. However, rare and more severe reactions have been reported, including dermatomyositis, Stevens-Johnson syndrome–toxic epidermal necrolysis, drug reaction with eosinophilia and systemic symptoms, and anticonvulsant hypersensitivity syndrome.[1]

We report a patient who developed new-onset erythrodermic psoriasis following treatment with carbamazepine and levetiracetam for seizure management. The eruption persisted despite discontinuation of AEDs

and improved only after initiation of systemic biologic therapy. Although limited psoriasiform drug reactions have been described with individual use of carbamazepine, valproic acid, and levetiracetam,[2,3] de novo induction of erythrodermic psoriasis by AEDs is rare.

Erythroderma is a severe exfoliative dermatitis characterized by diffuse erythema, typically involving more than 90% of the total body surface area, often accompanied by scaling. Systemic complications include profound fluid and electrolyte disturbances, thermal dysregulation, high-output cardiac failure, acute respiratory distress syndrome, and increased susceptibility to infection and sepsis. Reported mortality ranges from 4% to 64% and can reach approximately 30% in patients with erythroderma secondary to psoriasis.[4,5] Prompt recognition and management are therefore essential.

Distinguishing erythroderma owing to psoriasis from spongiotic (eczematous) dermatitis, cutaneous T-cell lymphoma (CTCL), or drug-induced hypersensitivity reactions can be challenging owing to overlapping clinical and histopathologic features. In the present case, biopsy demonstrated regular psoriasiform epidermal hyperplasia, broad confluent parakeratosis, marked hypogranulosis, and dilated capillaries within the superficial papillary dermis, supporting a diagnosis of psoriasis. Although Munro microabscesses and spongiform pustules of Kogoj were absent, these are not required for diagnosis. Mild focal spongiosis was observed but interpreted as incidental, as it can occur in psoriasis and was insufficient to support a primary spongiotic dermatitis. CTCL was considered unlikely given the absence of epidermotropism, Pautrier microabscesses, or cytologic atypia. Rare dermal eosinophils raised the possibility of a psoriasiform drug hypersensitivity reaction; however, the clinical course and persistence of disease after drug withdrawal ultimately favored a diagnosis of new-onset psoriasis.

Classically, medications associated with drug-induced psoriasis include beta-blockers, lithium, antimalarial drugs such as chloroquine, interferons, and nonsteroidal anti-inflammatory drugs.[6] More recently, monoclonal antibodies and small-molecule therapies used in psoriasis treatment, including TNF-α inhibitors and anti–IL-13, IL-17, and IL-23 agents, have been implicated in "paradoxical psoriasis," affecting 2% to 5% of patients on these therapies.[7-9] Novel immune checkpoint inhibitors, including CTLA-4 and PD-1/PD-L1 inhibitors, have also been associated with drug-induced psoriasis.[10]

The mechanisms underlying drug-induced psoriasis remain incompletely understood, presenting ongoing challenges in management. Psoriasiform eruptions following recent initiation or dose adjustment of medications may result from: (1) exacerbation of preexisting psoriasis, (2) de novo induction of psoriasis, or (3) direct drug-induced immune dysregulation leading to a psoriasiform cutaneous reaction. In many cases, discontinuation of the offending medication leads to remission; however, persistent disease may require long-term systemic immunosuppressive or biologic therapy, as observed in the present case. Although our patient was ultimately diagnosed with new-onset psoriasis, this presentation may exist on a spectrum with previously reported psoriasiform drug eruptions associated with AED use. Persistent disease following drug withdrawal and rapid response to biologic therapy supported a diagnosis of true psoriasis, though early differentiation from drug-induced eruptions can be challenging owing to overlapping clinical and histologic features.

Conclusion

This case underscores the importance of recognizing that AEDs can trigger severe and persistent cutaneous reactions, particularly in patients with autoimmune predisposition. Clinicians should consider the possibility of new-onset psoriasis following initiation of AEDs such as carbamazepine and levetiracetam, as progression to erythroderma can occur. Early identification and appropriate management are essential to prevent complications and optimize patient outcomes.

Potential conflicts of interest

The authors declare no conflicts of interest.

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