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BRIEF ARTICLE

Successful Treatment of Terbinafine-Induced Subacute Cutaneous Lupus Erythematosus With Upadacitinib

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ABSTRACT

Drug-induced subacute cutaneous lupus erythematosus (DI-SCLE) is now the most common form of drug-induced lupus erythematosus (DI-LE). Terbinafine is a well-established DI-SCLE trigger and most cases resolve with traditional therapies, such as topical and oral steroids. However, some cases may be more persistent and refractory to these treatments, or patients may be unable to tolerate systemic steroid use due to underlying conditions. These challenges highlight the limitations of conventional therapies, underscoring the need for alternative treatments in difficult to treat or protracted cases. This report presents a case of terbinafine-induced SCLE that was unresponsive to systemic steroids and quickly resolved with a short course of upadacitinib, an oral selective JAK-1 inhibitor.

INTRODUCTION

Drug-induced lupus erythematosus (DI-LE) is characterized when a patient develops clinical and immunopathological symptoms similar to idiopathic lupus following drug exposure, with symptoms resolving after discontinuation of the medication, and reappearing upon reexposure to the drug.[3]/15/2025 4:11:00 PM DI-LE can mimic any different form of lupus and more than 80 different chemical compounds have been linked to DI-LE with hydrochlorothiazide, procainamide, isoniazid, and minocycline being the most frequently implicated drugs.[1,2] DI-LE is one of many idiopathic immune-mediated conditions that can be drug induced, including conditions like psoriasis, pemphigus, and lichen.[3]

Drug-induced subacute cutaneous lupus erythematosus (DI-SCLE) was once considered rare, and it is now recognized as the most common form of DI-LE.[4,5] DI-SCLE is a non-scarring, non-atrophic, photosensitive cutaneous disorder that typically presents as either papulosquamous or annular erythematous scaly lesions on the trunk and upper and lower extremities.[6] DI-SCLE is most commonly associated with the administration of hydrochlorothiazide, an increasing number of cases have reported development of this entity 4-8 weeks after initiating terbinafine therapy for cutaneous mycoses. Most cases resolve quickly after drug discontinuation with oral or topical steroids, but some cases are more treatment resistant or seen in patients who cannot tolerate systemic steroids. We present a case of terbinafine-induced SCLE which was unresponsive to systemic steroids that quickly resolved with a short course of upadacitinib, an oral selective JAK-1 inhibitor.

CASE REPORT

A 77-year-old woman with a history of hypertension and hyperlipidemia presented to our clinic for evaluation of onychomycosis. She was prescribed a 12 week course of oral terbinafine for treatment. Six weeks after treatment initiation, she presented to clinic with new onset annular, erythematous, pruritic scaly rash on the bilateral upper and lower extremities (**Figure 1**). Lab testing showed a positive ANA (1:320 titer) with a nuclear and homogenous pattern and a positive anti-histone antibody (1.3). Other lab workup was unremarkable, and the patient denied any fevers, malaise, joint pain, mouth ulcers, or other systemic symptoms. Punch biopsy was performed which showed interface dermatitis and degeneration of the basal layer. The histology was consistent with SCLE and lab workup and lack of systemic symptoms confirmed the diagnosis of terbinafine-induced SCLE.

The patient was treated with an intramuscular cortisone injection without improvement followed by high dose oral prednisone (1mg/kg daily) with worsening of pruritus and rash over a 2 week span despite drug discontinuation. The patient was started on upadacitinib 15 mg daily. She had reduction of pruritus within the first day and had complete resolution of rash and pruritus within 2 weeks. After 2 weeks, treatment was discontinued and her rash and pruritus had completely resolved with some residual post-inflammatory pigmentary change (**Figure 2**). Clinical follow up at one month post treatment showed complete resolution of rash and pigmentary change with no recurrence and serum ANA and anti-histone antibodies were both negative.

DISCUSSION

Drug-induced SCLE by terbinafine has become a well recognized entity that is often characterized by a more prolonged disease course compared to other DI-SCLE offenders.[7,8] Some cases resolve by identification and withdrawal of the triggering agent but many cases require further treatment.[9] Traditional therapies include topical and/or systemic steroids or antimalarials, with immunosuppressive agents usually being reserved for more resistant cases of DI-LE.[1,10] Despite rapid resolution of some cases, there are still many cases that are refractory to traditional therapies or in patients who are unable to tolerate systemic steroids due to other underlying conditions. There is an unmet need for alternative therapies that can provide rapid resolution of difficult to treat cases of DI-SCLE beyond the traditional steroid-sparing agents.

In this case, the patient had inadequate response with worsening of DI-SCLE despite drug discontinuation and systemic steroids, prompting the consideration of upadacitinib as an alternative treatment option. Upadacitinib is a novel oral, selective, and JAK-1 inhibitor that has demonstrated a considerable favorable benefit-risk profile with anti-inflammatory benefits across a plethora of dermatologic, rheumatologic, and gastrointestinal diseases.[11] The JAK-STAT signaling pathway is known to play a significant role in pathogenesis of cutaneous lupus erythematosus (CLE) and is being studied extensively in patients with lupus prompting our use of this therapy.[12]

CONCLUSION

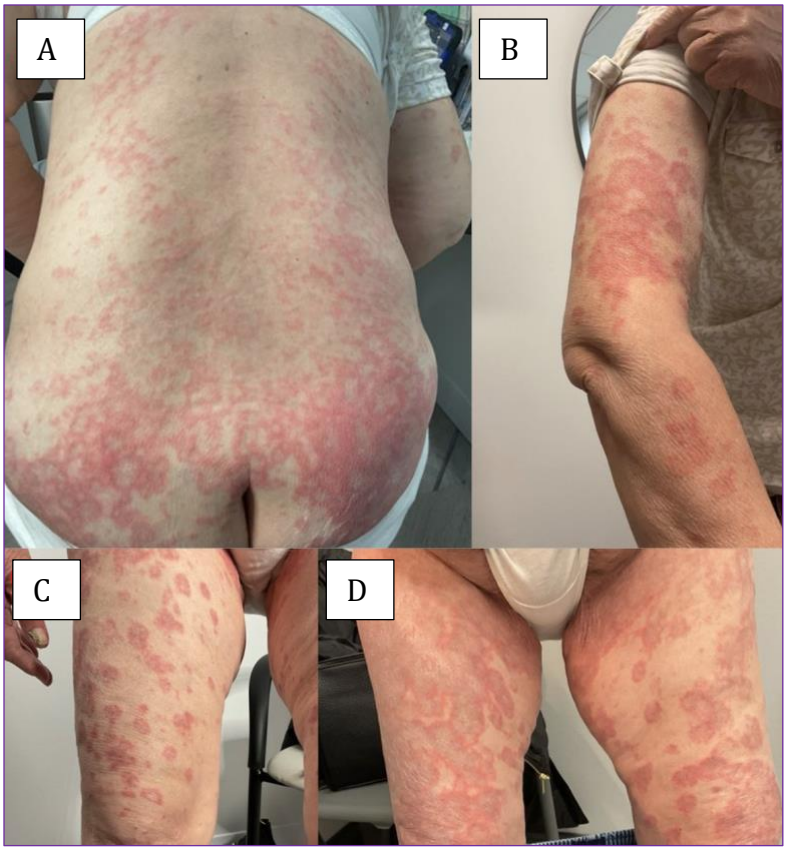

Figure 1. New onset annular, erythematous, pruritic scaly rash on the back (A), bilateral upper (B) and lower extremities (C,D).

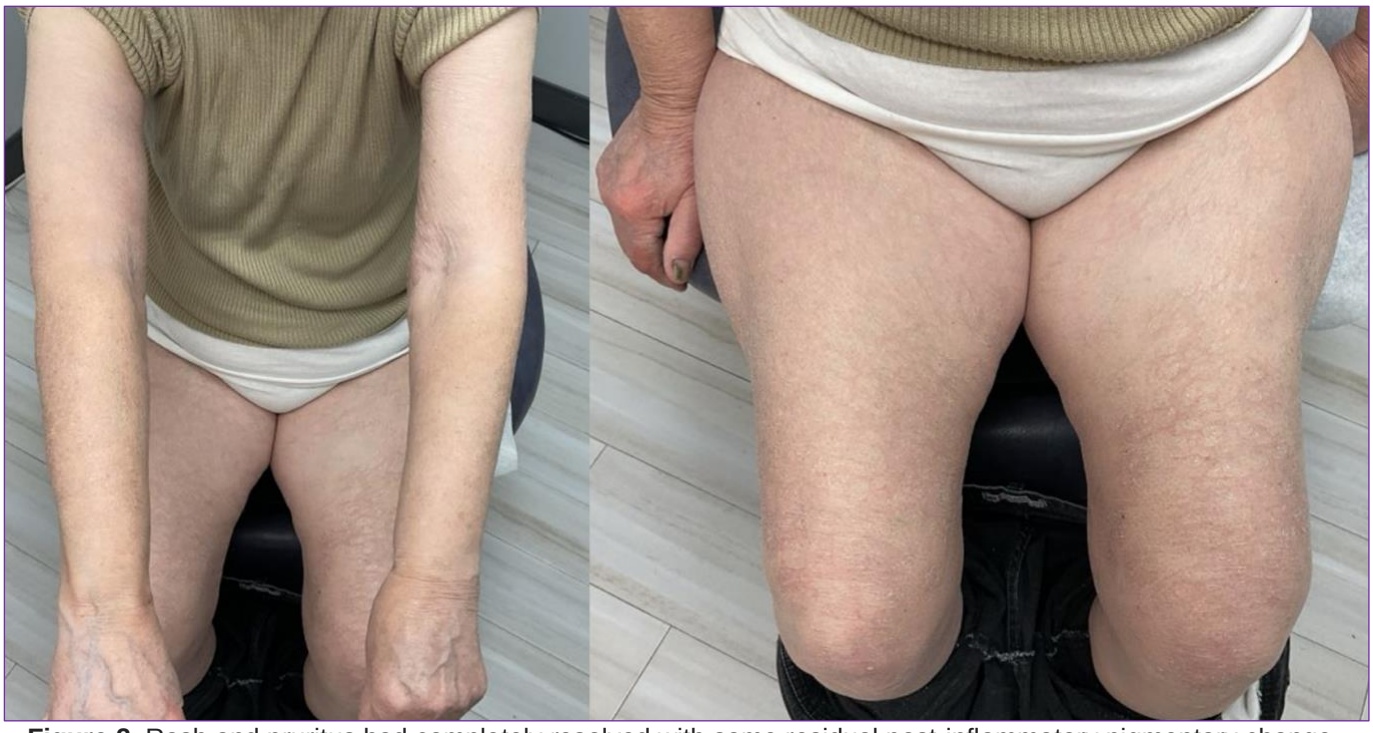

Figure 2. Rash and pruritus had completely resolved with some residual post-inflammatory pigmentary change.

Our patient had a rapid and complete resolution of DI-SCLE after short duration therapy with upadacitinib after failing systemic steroids. Given the success of this drug for similar inflammatory conditions, we suggest that it would be reasonable to consider upadacitinib as an alternative therapy for DI-SCLE, particularly refractory cases. While steroids and antimalarials will remain cornerstones of treatment of DI-SCLE, it is reasonable to consider upadacitinib as a therapy for patients who do not respond to steroids or have other contraindications which limit their ability to use traditional therapies. Although further studies are needed to fully assess the long-term efficacy of upadacitinib in DI-SCLE, this case adds to the growing body of evidence supporting its use in inflammatory and autoimmune dermatologic disorders.

Conflict of Interest Disclosures: None

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