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Cutaneous complications of cyclosporine: a case of drug-induced folliculitis in atopic dermatitis

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Abstract

Cyclosporine-induced folliculitis remains underreported and underrecognized in clinical practice, posing diagnostic challenges for dermatologists and clinicians. In this case report, we present a compelling case of cyclosporine-induced folliculitis.

A 40-year-old patient with severe atopic dermatitis was treated with topical steroids, calcineurin inhibitor, and phototherapy for 6 months. After starting cyclosporine 150 mg, acne-like eruptions appeared, and a 4 mm skin biopsy revealed folliculitis and perifolliculitis.

Cyclosporine-induced folliculitis is a rare adverse effect of cyclosporine therapy, requiring prompt management through dose adjustment, discontinuation, and adjunctive therapies.

Introduction

Cyclosporine, a potent immunosuppressive agent, is widely used to manage various autoimmune and inflammatory conditions, including organ transplantation and autoimmune dermatoses.

While its efficacy in controlling immune-mediated diseases is well-established, cyclosporine therapy is associated with a spectrum of adverse effects, ranging from nephrotoxicity to cutaneous manifestations.[1] One dermatological complication is cyclosporine-induced folliculitis, a relatively uncommon adverse reaction characterized by developing inflammatory papules and pustules involving hair follicles.[2] Despite its recognition as a potential side effect of cyclosporine therapy, cyclosporine-induced folliculitis remains underreported and underrecognized in clinical practice, posing diagnostic challenges for dermatologists and clinicians. This case report presents a compelling case of cyclosporine-induced folliculitis, detailing its clinical presentation, histopathological findings, and management approach. By elucidating the nuances of this adverse drug reaction, we aim to enhance awareness and facilitate timely recognition and management of cyclosporine-induced folliculitis among healthcare professionals.

Case Report

A 40-year-old, medically healthy male presented in our dermatology clinic for severe atopic dermatitis. The patient has been treated with moderate- to high-potency topical steroids, a topical calcineurin inhibitor, and phototherapy for 6 months with minimal response. Consequently, a regimen of cyclosporine at 150 mg twice daily was initiated. Two months later, the patient began to have acne-like eruptions all over the face. Those lesions started suddenly, and they were not itchy or painful. Skin examination showed multiple varying-sized, well-defined, erythematous, infiltrated papules over the

face (Figure 1). A 4 mm skin biopsy was taken from a lesion over the forehead, which showed folliculitis and perifolliculitis with mixed dermal infiltrates (Figure 2). The diagnosis of folliculitis was made based on clinical and histological findings. The patient was advised to discontinue cyclosporine and was prescribed a course of clindamycin 150 mg twice daily for two months. During the follow-up visit, the patient showed significant improvement with few papular eruptions compared to baseline (Figure 3).

Discussion

Cyclosporine is a potent immunosuppressant drug with known anti-inflammatory characteristics. Its various clinical applications include use in diverse immuno-dermatological diseases, such as atopic dermatitis, psoriasis, and urticaria.[3]

Cyclosporine was the first immunosuppressive agent found to act selectively on T-cells. Studies have demonstrated that cyclosporine decreases the quantities of helper/inducer T-cells and the number of activated cells that express Interleukin-2 (IL-2), IL-4, and IL-5. Additionally, it hinders the proliferation and specialization of B-lymphocytes and the operational functions of mononuclear phagocytes, Langerhans cells, and eosinophils. Moreover, a recent immunohistochemistry investigation has demonstrated that cyclosporine may have a therapeutic impact by influencing the cutaneous nerve system through changes in innervation and the expression of neuropeptides in the affected skin of atopic dermatitis. Furthermore, cyclosporine prevents the activation of mast cells (MC) *via* modifying the spatial arrangement between MCs and the nerves in the affected skin, indicating a novel element of cyclosporine's activities in treating disease activity.[4]

Cyclosporine has been approved for the treatment of atopic dermatitis in Europe and has been used off-label for the management of various inflammatory skin conditions. Atopic dermatitis is a chronic inflammatory skin condition requiring an interdisciplinary approach encompassing appropriate skin hydration, pharmacological therapy, and identifying and eliminating triggers.

Traditional treatments don't work for some patients, requiring anti-inflammatory and immunosuppressive medications like cyclosporine, as demonstrated in the case we're presenting.[4]

Although infrequent, cyclosporine is associated with undesirable effects that range in severity, including nephrotoxicity, induction and/or deterioration of systemic hypertension, hyperlipidemia, and CNS toxicity.[5] A previous study[6] found that the skin is the primary site of cyclosporine accumulation and the majority of cyclosporine's cutaneous side effects involve the pilosebaceous unit: hypertrichosis, sebaceous hyperplasia, acne, folliculitis, epidermal cysts, and pilar keratosis.[7] Another

study, which treated 67 out of 100 kidney transplant recipients with cyclosporine and methylprednisolone, revealed that 28% of them had cutaneous manifestations related to their previous uremic state. Most of the lesions concerned the pilosebaceous unit: hypertrichosis (60%), epidermal cysts (28%), pilar keratosis (21%), acne (15%), folliculitis (12%), and sebaceous hyperplasia (10%). In our case, the patient only exhibited folliculitis.

In the reported literature, all patients who experienced cutaneous side effects of cyclosporine were immunocompromised; our patient was medically free.

El-Shahawy *et al.* reported a case of a renal transplant patient who developed severe acne with progression to nodulocystic acne (acne conglobata), which only showed resolution 12 months after discontinuing cyclosporine.[5] In our case, we discontinued the use of cyclosporine. We started the patient on a 150-mg dosage of clindamycin for a month, along with topical clindamycin, which resulted in significant improvement with fewer papular eruptions compared to his baseline.

Conclusions

In conclusion, cyclosporine-induced folliculitis is a rare but recognizable adverse effect of cyclosporine therapy. Clinicians should be aware of this potential complication and manage it promptly through dose adjustment, discontinuation, and adjunctive therapies. Continued documentation and reporting of such cases will enhance understanding and guide best practices in managing cyclosporine-associated dermatological side effects.

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Figure 1. Pre-clindamycin treatment.

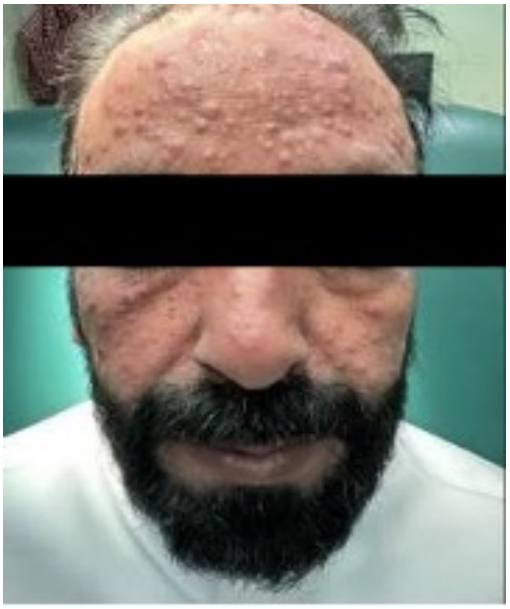

Figure 2. Microscopic section showing marked mixed inflammatory cells, most of which are neutrophils, infiltrating the hair follicle and perifollicular dermis. (H&E x 4) and (H&E x 10).

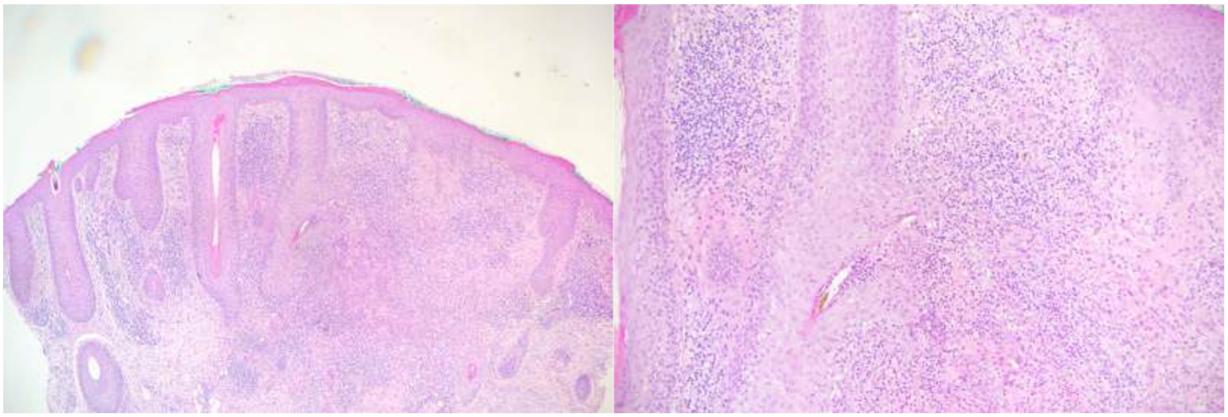

Figure 3. Post-clindamycin treatment.