

Tinea Incognito Mimicking Pustular Psoriasis in a Patient With Psoriasis and Cushing Syndrome

Gerhard Eichhoff, MD, PhD

PRACTICE POINTS

- Tinea incognito and its altered clinical presentation can provide clinical challenges and often is diagnosed with delay.
- Immunosuppression, such as iatrogenic Cushing syndrome, is a risk factor for tinea incognito.
- Pustular tinea incognito is a differential diagnosis of pustular psoriasis that can mimic tumor necrosis factor inhibitor treatment failure in patients with psoriasis.

To the Editor:

The term *tinea incognito* was introduced by Ive and Marks¹ in 1968 and refers to unusual clinical presentations of tinea due to the application of topical corticosteroids. Tinea incognito, which does not feature the classical clinical characteristics of tinea corporis such as well-defined, erythematous, scaly patches and elevated borders, is regularly misdiagnosed as inflammatory dermatosis.² Immunosuppression caused by topical and/or systemic steroids predisposes patients to the development of tinea.³ Herein, a case of widespread pustular tinea incognito mimicking pustular psoriasis along with failure of tumor necrosis factor (TNF) inhibitor treatment is reported in a patient with chronic plaque psoriasis and steroid-induced Cushing syndrome.

A 46-year-old man with a 25-year history of psoriasis was referred to the dermatologic outpatient clinic with a severe flare-up of chronic plaque psoriasis. Prior treatments included methotrexate and acitretin without response. Narrowband UVB treatment was discontinued

due to claustrophobia. Topical treatment with calcipotriol 0.005%–betamethasone dipropionate 0.05% gel was reported to be ineffective. The patient was administered prednisone over several months in a primary care setting at a dosage of 35 mg daily when he presented to the dermatology clinic. Physical examination revealed widespread chronic plaque psoriasis of the trunk and extremities, and a psoriasis area and severity index score of 15 was calculated. The patient had onychodystrophy with subungual hyperkeratosis of all toenails. Signs of prednisone-induced Cushing syndrome, including central obesity, lipodystrophy, and red striae, were noted.

Treatment was started by dermatology with the TNF inhibitor adalimumab at an initial dose of 80 mg, followed by subsequent 40-mg doses every other week; prednisone was tapered off. Topical treatment with a 4-week course of clobetasol propionate cream 0.05% daily for psoriatic lesions was initiated.

Six weeks after the initial consultation, the patient presented to the hospital's emergency department with worsening symptoms of itchy, burning, and painful skin after good initial improvement. The patient's skin started to burn upon application of clobetasol and the rash worsened. The patient did not use emollients. At that point, the patient was on a daily dose of 15 mg of prednisone. On dermatologic review, multiple partially annular lesions with subtle scaling and multiple pustules on the arms and legs as well as the buttocks and groin were noticed. These lesions were confined to sites of prior psoriasis as marked by postinflammatory hyperpigmentation (Figure 1). Widespread tinea was assumed, and treatment with fluconazole 50 mg daily was administered for 4 weeks. Direct examination of skin scrapings from the patient's thigh showed hyphae, and fungal culture was positive for

From the Dermatology Service Capital and Coast District Health Board, Wellington Regional Hospital, New Zealand.

The author reports no conflict of interest.

Correspondence: Gerhard Eichhoff, MD, PhD (gerhard.eichhoff@ccdhb.org.nz).

doi:10.12788/cutis.0239

Trichophyton rubrum. Scrapings from the patient's hallux nail remained inconclusive due to bacterial overgrowth. At 4-week follow-up, the patient's skin had cleared entirely and showed only postinflammatory changes (Figure 2). Healthy proximal nail growth was observed. Fluconazole was continued at a once-weekly dose of 150 mg together with adalimumab at a dose of 40 mg every 2 weeks and a prednisone tapering schedule.

This case describes pustular tinea incognito in a patient with chronic plaque psoriasis. As the name indicates, tinea incognito can mimic other skin conditions and classically is linked to topical application of corticosteroids.¹ Tinea incognito can be a diagnostic challenge. Kim et al⁴ reported a diagnostic delay of 15 months and the frequent requirement for the involvement of a second physician or dermatologist. Treatment with topical or systemic corticosteroids is a risk factor for dermatophyte infections because of their immunosuppressive action.^{3,5} Although recommended by current guidelines, a large number of psoriatic patients are treated with systemic

steroids, predominantly prescribed in primary care, that can lead to iatrogenic Cushing syndrome, as demonstrated in this patient.⁶

In addition to systemic and topical steroids, the reported patient was started on the TNF inhibitor adalimumab prior to the onset of the tinea. Cases of patients on TNF inhibitors with widespread tinea are scarce. Bardazzi et al⁷ reported 2 cases of widespread nonpustular tinea in patients with psoriasis on TNF inhibitor treatment without further immunomodulating treatment. They hypothesized that TNF- α could be an important cytokine in the defense against dermatophytes.⁷

Whether psoriasis itself is a risk factor for tinea is still under debate, but tinea pedum and onychomycosis seem to have higher prevalence among psoriatic patients.^{8,9} As in this patient, bacterial overgrowth of hyperkeratotic nail samples can confound the culture's clinical significance, thereby hindering the diagnosis of onychomycosis in patients with psoriasis.¹⁰ Alteras et al⁸ hypothesized that autoinoculation from preexisting

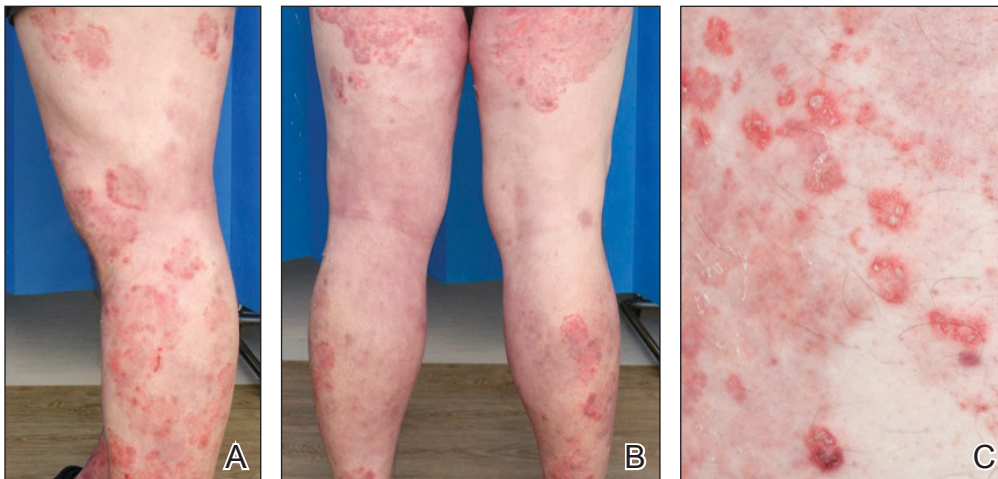


FIGURE 1. A and B, Widespread red annular plaques on the legs and subtle scaling within hyperpigmentation caused by prior psoriatic plaques. C, Close-up revealed multiple pustules.

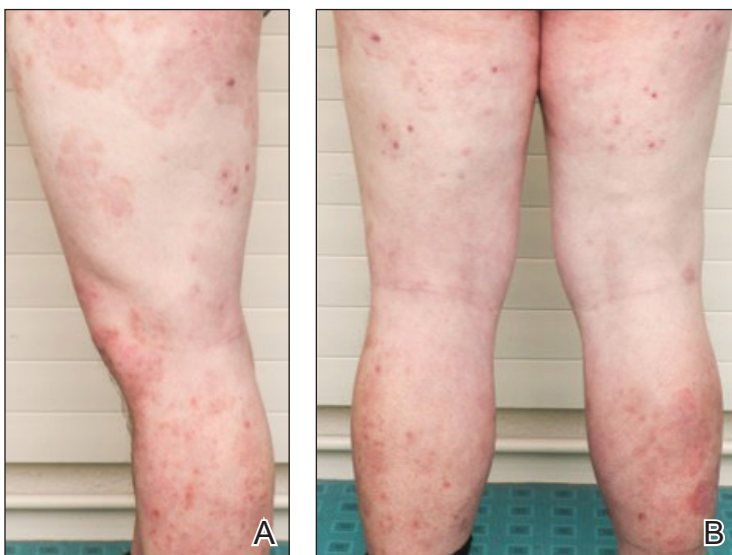


FIGURE 2. A and B, Skin lesions cleared 4 weeks after treatment with fluconazole showing postinflammatory hyperpigmentation only.

onychomycosis or tinea pedum was the underlying mechanism of tinea incognito.

This patient's hyperkeratotic nails showed healthy regrowth after initiation of both fluconazole and adalimumab, though it remained unclear whether preexisting onychomycosis was a possible source of tinea incognito. The finding that the patient's tinea was almost exclusively limited to the sites of prior psoriatic lesions argues for autoinoculation and spreading accelerated by application of topical steroids triggered by the immunosuppressive effects of both topical and systemic steroids. The TNF inhibitor treatment may have helped to unmask the dermatophyte infection rather than contributing to it, as it cleared the psoriatic plaques.

Apart from psoriasis, tinea incognito most commonly is mistaken for other inflammatory conditions such as eczema, folliculitis, rosacea, granuloma annulare, and discoid lupus erythematosus.² Inflammatory tinea can present with pustules due to the increased occurrence of neutrophil invasion.¹¹

This patient's symptoms worsened 4 weeks after the initiation of TNF inhibitor treatment, which suggested treatment failure. However, clearance of the preexisting psoriatic lesions with remnant hyperpigmentation only argued for good response to TNF inhibitor treatment. The main differential diagnosis of this case of tinea incognito was generalized pustular psoriasis. The patient also was being treated with systemic and topical steroids, both known for their potential to trigger pustular psoriasis.^{12,13} Furthermore, TNF inhibitors have been described as a trigger for predominantly palmoplantar pustulosis but also are additionally associated with generalized pustular psoriasis.¹⁴

This case aims to raise awareness that tinea incognito can imitate both pustular psoriasis and TNF inhibitor treatment failure. Furthermore, the presented findings highlight risks associated with the treatment of psoriasis with systemic steroids. Pustular tinea incognito should be considered in the differential diagnosis of pustular psoriasis, especially in the setting of immunosuppression.

After initial improvement, worsening of symptoms such as itching and burning as well as extension of the lesions upon application of topical steroids are regularly described in tinea incognito and can be present in addition to the more typical annular presentation of lesions as a clue to the diagnosis.

REFERENCES

- Ive FA, Marks R. Tinea incognito. *Br Med J*. 1968;3:149-152.
- Arenas R, Moreno-Coutiño G, Vera L, et al. Tinea incognito. *Clin Dermatol*. 2010;28:137-139.
- Rouzaud C, Chosidow O, Brocard A, et al. Severe dermatophytosis in solid organ transplant recipients: a French retrospective series and literature review [published online January 25, 2018]. *Transpl Infect Dis*. doi:10.1111/tid.12799
- Kim WJ, Kim TW, Mun JH, et al. Tinea incognito in Korea and its risk factors: nine-year multicenter survey. *J Korean Med Sci*. 2013;28:145-151.
- Ohta Y, Saitoh N, Tanuma H, et al. Local cytokine expression in steroid-modified tinea faciei. *J Dermatol*. 1998;25:362-366.
- Augustin M, Schäfer I, Reich K, et al. Systemic treatment with corticosteroids in psoriasis—health care provision far beyond the S3-guidelines. *J Dtsch Dermatol Ges*. 2011;9:833-838.
- Bardazzi F, Balestri R, Rech G, et al. Dermatophytosis during anti-TNF- α monoclonal antibody therapy. *Mycoses*. 2011;54:E619-E620.
- Alteras I, Ingberg A, Segal R, et al. The incidence of skin manifestations by dermatophytes in patients with psoriasis. *Mycopathologia*. 1986;95:37-39.
- Leibovici V, Ramot Y, Siam R, et al. Prevalence of tinea pedis in psoriasis, compared to atopic dermatitis and normal controls—a prospective study. *Mycoses*. 2014;57:754-758.
- Tsentemidou A, Vyzantiadis TA, Kyriakou A, et al. Prevalence of onychomycosis amongst patients with nail psoriasis who are not receiving immunosuppressive agents: results of a pilot study. *Mycoses*. 2017;60:830-835.
- Hirschmann JV, Raugi GJ. Pustular tinea pedis. *J Am Acad Dermatol*. 2000;42:132-133.
- Brenner M, Molin S, Ruebsam K, et al. Generalized pustular psoriasis induced by systemic glucocorticosteroids: four cases and recommendations for treatment. *Br J Dermatol*. 2009;161:964-966.
- Boxley JD, Dawber RP, Summerly R. Generalized pustular psoriasis on withdrawal of clobetasol propionate ointment. *Br Med J*. 1975;2:255-256.
- Kucharekova M, Winnepenninckx V, Frank J, et al. Generalized pustulosis induced by adalimumab in a patient with rheumatoid arthritis—a therapeutic challenge. *Int J Dermatol*. 2008;47:25-28.