

When Psoriasis Is Not Psoriasis: Tinea Incognito Induced by Long-Term Steroid Abuse

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Abstract

Background: Tinea incognito is a dermatophyte infection with atypical clinical features caused by inappropriate use of topical or systemic corticosteroids. It often mimics other dermatoses, particularly psoriasis, leading to delayed diagnosis and inappropriate management. Misuse of steroids may mask inflammation while allowing fungal proliferation and dissemination. **Case Presentation:** A 60-year-old male farmer presented with a two-week history of pruritic, thick, scaly erythematous plaques involving the chest, abdomen, back, and upper arms, accompanied by fever, myalgia, and malaise. Initially, the patient noticed mildly pruritic erythematous patches one month prior and self-treated with topical dexamethasone, resulting in temporary relief but progressive worsening. He also reported long-term oral dexamethasone use (0.5 mg daily for 10 years). Lesions evolved into widespread plaques with thick scales and pustules, clinically resembling pustular psoriasis. KOH examination of skin scrapings revealed long septate hyphae, confirming dermatophytosis, while Gram staining was negative for bacteria. Laboratory findings were unremarkable. The diagnosis of extensive tinea incognito with concurrent viral infection was established. The patient was treated with oral itraconazole (200 mg/day) and topical ketoconazole for four weeks, showing significant clinical improvement by the third week. **Conclusion:** This case highlights the importance of considering tinea incognito in steroid-modified dermatoses mimicking psoriasis. Early mycological examination is essential to avoid misdiagnosis and inappropriate steroid use. Rational antifungal therapy leads to favorable outcomes.

Keywords: *Tinea Incognito, Corticosteroid Abuse, Psoriasis Mimicry, Dermatophytosis.*



A. INTRODUCTION

Inappropriate topical or systemic corticosteroid use can lead to tinea incognito, a dermatophyte infection with unusual clinical symptoms. Corticosteroids' immunosuppressive effects reduce inflammation and change the traditional dermatophytosis morphology, which commonly results in incorrect diagnosis and postponed therapy (Arora & Gupta, 2025; Nenoff, 2021). Steroid-modified fungal infections are becoming more common because to the growing abuse of corticosteroids, especially in environments where they are easily accessible over-the-counter. This presents a serious clinical problem globally (Kokandi, 2024; Kruithoff et al., 2023; Zacharopoulou et al., 2024).

Clinically, tinea incognito can resemble psoriasis that is characterized by well-defined erythematous plaques with silvery scales (Ghaderi et al., 2023; Zacharopoulou et al., 2024). Steroid-modified dermatophytosis may present with psoriasiform features like thick scaling and pustular lesions, which frequently results in misdiagnosis and inappropriate corticosteroid escalation. Increased morbidity,

broader fungal dispersion, and disease progression could all be consequences of this diagnostic overlap (Gallegos Espadas et al., 2024).

Prolonged corticosteroid exposure, especially systemic dosing, is linked to more widespread and resistant dermatophyte infections (Arora & Gupta, 2025). In order to prevent misdiagnosis and improper treatment of atypical or treatment-resistant dermatoses, early mycological evaluation, including potassium hydroxide (KOH) examination, is crucial (Kokandi, 2024; Zacharopoulou et al., 2024).

This case report highlights the significance of precise diagnosis and logical treatment approaches by describing a case of tinea incognito brought on by chronic corticosteroid misuse that clinically resembled psoriasis.

B. CASE PRESENTATION

A 60-year-old man presented with complaints of thick, rough, scaly erythematous patches associated with itching on the chest, abdomen, back, and upper arms for the past two weeks. These symptoms were accompanied by fever for three days, myalgia, and general malaise. Approximately one month earlier, he had initially noticed the appearance of mildly pruritic, faint erythematous patches on the chest, abdomen, back, and arms. The patient had been routinely applying desoxymethasone 0,25% cream twice daily, which he obtained over the counter from drug store. Although the itching improved with its use, the patches became increasingly erythematous. About two weeks later, the lesions worsened, with more pronounced redness and thickening of scales. One week prior to hospital admission, he began experiencing body aches, and the thick scaly patches expanded over the chest, abdomen, back, and upper arms. Two days before admission, pustular lesions developed over some of the patches on the back, accompanied by the appearance of new erythematous patches on both arms and legs.

The patient is a farmer who has been living alone on his plantation for approximately 30 years. He has a history of taking dexamethasone 0.5 mg tablet once daily for the past 10 years, which he uses on his own initiative as an energy booster. He reports experiencing body aches and muscle pain when he does not take the medication. The patient has no history of diabetes or other chronic illnesses.

On physical examination, the patient had a body weight of 56 kg and a height of 162 cm, with a body mass index (BMI) of 21.3 kg/m², consistent with a normal weight status. The patient's general condition was good and he was fully conscious. Vital signs revealed a blood pressure of 100/70 mmHg, a pulse rate of 100 bpm, a temperature of 39°C, and a respiratory rate of 22 breaths per minute.

Dermatological examination of the chest, abdomen, back, and upper arms revealed erythematous macules, erythematous plaques, and erythematous plaques covered with thick, coarse white scales, with some lesions showing overlying pustules. Some lesions have polycyclic edges and are sharply demarcated to poorly demarcated. Dermatological examination of both lower extremities demonstrated erythematous macules, erythematous plaque accompanied by xerosis cutis. The clinical features resemble as inflammatory erythematous-squamous disease. The

working diagnosis in this case is psoriasis pustulosis and the differential diagnosis is dermatomycosis with secondary infection.

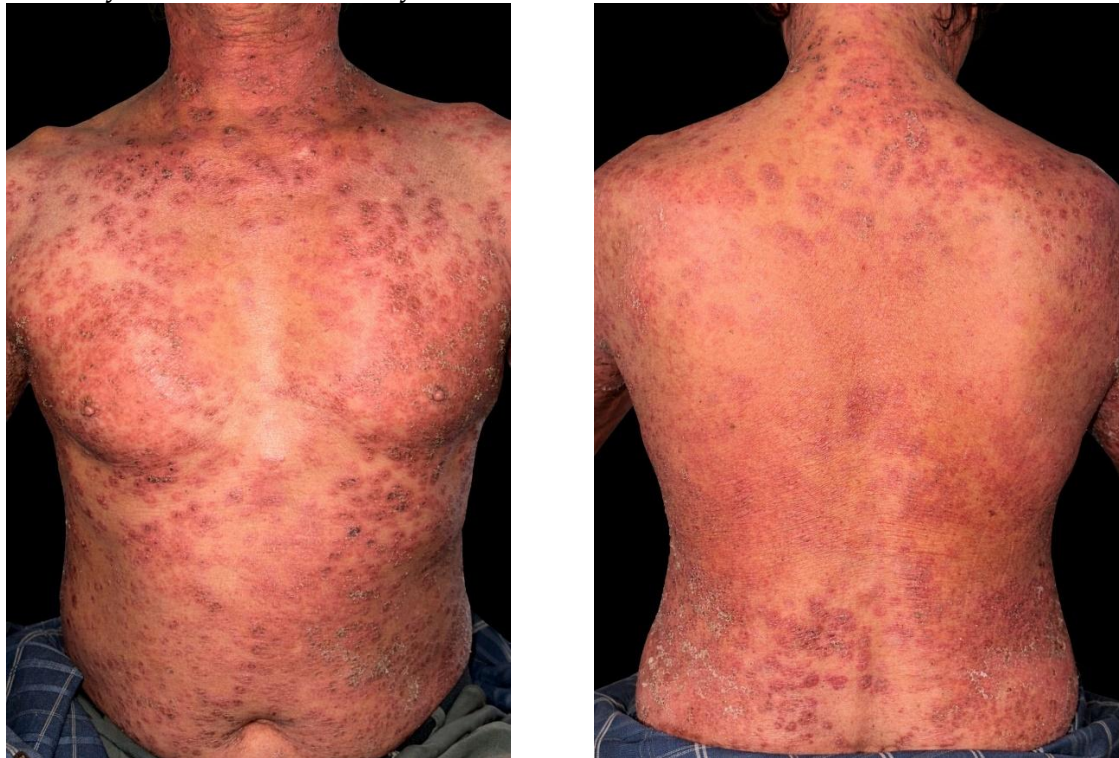


Figure 1. Dermatological examination on the neck, chest, stomach, back, there are erythematous macules, erythematous plaques, rough and layered scales.



Figure 2. Dermatological examination on the left upper arm revealed rough scaly, erythematous plaque with a distinct border and polycyclic edges is visible. Pustules are visible on top (black arrow)

Skin scrapings with KOH 10% microscopic examination from the edges of the reddish plaques lesions revealed long, septate hyphae. Gram staining of the pustules revealed multiple polymorphonuclear cells without any gram-positive or gram-

negative bacteria. We suspected for viral fever, liver, kidney dysfunction and glucose metabolism disorders due to long-term steroid use. However, the results of routine blood tests, liver function, renal function and blood sugar were within normal limits.



Figure 3. Microscopic examination of KOH shows long, septate hyphal structures (black arrow)

The diagnosis was established as widespread tinea incognito with suspected fever due to viral infection. The patient was admitted to the internal medicine department for 3 days for observation and treatment of fever and myalgia. The patient was given supportive fluid therapy, antipyretics, and vitamins for 3 days. For antifungal treatment, we administered oral itraconazole 200 mg/day for 1 month, and ketoconazole 2% cream applied twice daily for 1 month. Observations after 3 weeks of therapy revealed that the patient did not complain of itching. Dermatological examination did not reveal any reddish spots on the neck, chest, stomach, back or arms.



Figure 4. After 3 weeks of treatment, clinical improvement was evident. There were no thick, scaly red patches on the neck, chest, abdomen, back, and arms.

C. RESULTS AND DISCUSSION

Tinea incognito remains a diagnostic challenge in dermatology, primarily because its clinical presentation can be significantly altered following inappropriate corticosteroid use. With the increasingly widespread and often unsupervised use of steroids, this condition—originally described as a dermatophyte infection with atypical features—has become more clinically relevant. In the present case, prolonged administration of both topical and systemic dexamethasone appeared to suppress and modify the host's inflammatory response, resulting in skin manifestations that closely mimicked pustular psoriasis.

Corticosteroids are known to suppress cell-mediated immunity, a key defense mechanism in controlling dermatophyte infections. This immunosuppressive effect not only facilitates uncontrolled fungal proliferation but also masks the classic clinical features, such as well-demarcated annular borders and central clearing. Instead, lesions often appear more diffuse, less erythematous, and may present with pustules or prominent scaling, as observed in this patient. Recent studies have highlighted that steroid-modified dermatophytosis frequently exhibits a polymorphic presentation, which can easily lead to misdiagnosis as conditions such as seborrheic dermatitis, psoriasis, or eczema (Dhaher, 2020; Nenoff, 2021).

The patient's clinical presentation was further aggravated by prolonged use of systemic corticosteroids. Chronic exposure to these agents has been associated with disturbances in the skin microbiome, impairment of epidermal barrier integrity, and an increased susceptibility to infections, including more severe forms of

dermatophytosis. Moreover, the widespread misuse of fixed-dose combination creams and over-the-counter topical steroids remains a major contributing factor to the rising incidence of tinea incognito in Southeast Asia (Chopra et al., 2023). This pattern reflects an important public health concern, as patients often experience temporary symptomatic relief, which in turn reinforces continued and inappropriate use.

In this instance, there is a notable clinical similarity to pustular psoriasis. Both conditions may present with erythematous plaques and pustules; however, their underlying pathophysiology differs significantly. Psoriasis is an immune-mediated inflammatory disease whereas dermatophytosis is an infectious disorder caused by keratinophilic fungi (Nenoff, 2021). Self-treating may lead to inappropriate escalation of immunosuppressive therapy, potentially worsening fungal infection and promoting widespread dissemination (Chopra et al., 2023). Therefore, rapid and simple diagnostic methods such as potassium hydroxide (KOH) examination remain essential. In this case, identification of septate hyphae was crucial in establishing the correct diagnosis and preventing further inappropriate steroid use.

This patient may have had a concomitant viral infection, which could have further altered immune responses, given the coexistence of systemic symptoms including fever and malaise. Even though dermatophytosis usually only affects keratinized tissues, systemic immunosuppression and widespread infection can cause unusual presentations and systemic symptoms. According to recent research, examination for underlying immunosuppression, including iatrogenic causes, should be prompted by severe or widespread dermatophytosis (Arora & Gupta, 2025).

Management of tinea incognito requires prompt discontinuation of corticosteroids alongside initiation of appropriate antifungal therapy. Oral itraconazole remains a preferred systemic agent due to its efficacy against dermatophytes and its favorable pharmacokinetic properties in keratinized tissues. In more complex cases, combining systemic treatment with topical antifungals can further enhance therapeutic outcomes. In this patient, marked clinical improvement within three weeks supports existing evidence that early and adequate antifungal therapy is associated with a better prognosis (Arora & Gupta, 2025; Sooriya et al., 2021).

This case underscores several important considerations. First, dermatophytosis should be included in the differential diagnosis of atypical or steroid-resistant psoriasiform eruptions. Second, early mycological evaluation is essential to ensure accurate diagnosis and appropriate management. Finally, stricter regulation and improved education regarding corticosteroid use are urgently needed to prevent similar cases. Increasing awareness among both patients and healthcare providers is crucial to reducing the burden of steroid-modified fungal infections.

D. CONCLUSION

Patients presenting with atypical, steroid-modified dermatoses that mimic psoriasis should be carefully assessed for the possibility of tinea incognito,

particularly in those with a history of prolonged corticosteroid use. The misuse of steroids can mask the classic features of dermatophytosis, delay diagnosis, and contribute to disease progression. Early mycological examination is therefore essential to establish an accurate diagnosis, while prompt discontinuation of corticosteroids combined with appropriate antifungal therapy is key to achieving favorable clinical outcomes. Increasing awareness and promoting rational use of corticosteroids are crucial steps in preventing similar cases.

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