

What's Your Diagnosis?®

Sharpen Your Physical Diagnostic Skills

Boy With Pruritic Rash on Both Cheeks

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HISTORY

A 9-year-old boy presented with a pruritic, erythematous rash on his cheeks. He had been treated for 1 month with 0.1% betamethasone valerate cream. There was no associated fever or joint symptoms. His past health was unremarkable. All other family members were healthy and with no history of skin problems.

PHYSICAL EXAMINATION

Erythematous and slightly scaly patches on both cheeks.
Remaining examination findings unremarkable.

WHAT'S YOUR DIAGNOSIS?

(Answer on page 22.)

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ANSWER: Tinea incognita



Tinea incognita refers to a dermatophytosis that has lost its typical morphological features because of the use of corticosteroids or calcineurin inhibitors.^{1,2} The term “tinea incognita” was coined in 1968 by Ive and Marks who described 14 cases of atypical dermatophytoses secondary to the use of corticosteroids.³

PATHOGENESIS AND EPIDEMIOLOGY

Normally, the dermatophytes that cause tinea faciei—*Trichophyton rubrum*, *Trichophyton mentagrophytes*, *Trichophyton tonsurans*, *Microsporum canis*,^{4,5} and rarely *Microsporum gypseum*⁶—can be eliminated from the stratum corneum by a cell-mediated immune response.⁷ It is believed that tinea incognita is caused by a corticosteroid/calcineurin inhibitor-modified response of the host to a fungal infection rather than a pharmacologic effect on the fungus.⁶

Corticosteroids mediate their anti-inflammatory effects through binding to a cytoplasmic glucocorticoid receptor in the target cells and forming complexes that enter the nucleus of the cell.^{8,9} Once inside the nucleus, the corticosteroid-receptor complex interacts with glucocorticoid-response elements and alters transcription of various proinflammatory genes, with resultant suppression of inflammatory cell lines and cytokines.¹⁰

Calcineurin inhibitors work by binding to a cytoplasmic immunophilin.⁹ The complex inhibits the activity of calcineurin to dephosphorylate the nuclear factor of activated T-cell, a transcription factor required to activate IL-2 gene transcription. Inhibition of IL-2 production blocks the activation of T helper cells and T regulatory cells, and the activation of natural killer cells and monocytes.¹¹ The immune responses that stimulate inflammation are therefore down-regulated.

Corticosteroids/calcineurin inhibitors suppress the immune response mounted by the host and allow the fungus to proliferate easily.¹² As a result, the inflammation and pruritus is inhibited, erythema and scaling are decreased, and the tinea

may take on a bizarre appearance.^{2,12} Potent fluorinated corticosteroids seem most likely to produce tinea incognita.^{12,13} This is especially so when they are applied under occlusive dressings.⁴

Reports of tinea incognita are often sporadic. We list here the findings of a few large retrospective studies. In a study of 200 cases of tinea incognita (98 male and 102 female patients, with a mean age of 42 years) in Italy by Romano and colleagues,¹⁴ *T rubrum* and *T mentagrophytes* were most commonly isolated. In a study of 6325 subjects with suspected dermatophytoses in Iran by Ansar and colleagues,¹⁵ 56 patients (29 male and 27 female patients, with a mean age of 32.6 years) had tinea incognita. The most common type of infection was tinea corporis (32.1%) followed by tinea faciei (26.8%). *T mentagrophytes* and *T rubrum* were isolated in 28.6% and 12.5% of cases, respectively. In another study involving children in Spain by del Boz and colleagues,¹ 54 cases of tinea incognita were identified. Of the 28 male and 26 female patients, 9 were between 1 and 3 years of age, 20 between 4 and 10 years, and 25 between 10 and 14 years. *T mentagrophytes* and *T rubrum* were isolated in 44.4% and 13% of cases, respectively. The most usual clinical forms were tinea corporis (46.3%) and tinea faciei (38.9%).

CLINICAL MANIFESTATIONS

The clinical manifestations of tinea incognita are highly variable. The rash can be rosacea-like, eczema-like, or discoid lupus erythematosus-like, especially on the face; and impetigo-like or eczema-like on the trunk and limbs.¹⁴ The lesion can sometimes be pruritic and may be accompanied by a burning sensation.¹⁶

DIAGNOSIS

Tinea incognita should be suspected in a patient with any erythematous, scaly patch or plaque that fails to respond to treatment with corticosteroids or calcineurin inhibitors; this is especially the case when the rash is unilateral. The diagnosis can be confirmed by finding the fungal mycelium in the stratum corneum using a potassium hydroxide preparation of scrapings from the lesion.

Fungal culture from skin scrapings can also establish the diagnosis. Scrapings from scales at the edge of the lesion produce the best results. A skin biopsy at the edge of the lesion can also clarify the diagnosis, although this is seldom performed in children. Recently, it has been shown that *in vivo* reflectance confocal microscopy provides high-resolution and real-time imaging for the diagnosis of tinea incognita.¹⁷

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes atopic dermatitis, seborrheic dermatitis, cutaneous candidiasis, contact dermatitis, acne vulgaris, nummular eczema, granuloma annulare, discoid lupus erythematosus, lupus pernio, psoriasis, neurodermatitis, lichen-

oid dermatitis, impetigo, erythema annulare centrifugum, and polymorphous light eruption.^{2,12,14}

MANAGEMENT

Topical antifungal agents, such as miconazole, ketoconazole, econazole, naftifine, clotrimazole, ciclopirox olamine, and terbinafine, are the treatment of choice for tinea incognita.¹⁸ Adding 1% hydrocortisone powder to an antifungal cream may be warranted if the rash is very symptomatic. Oral antifungal agents, such as itraconazole, fluconazole, and terbinafine, can be considered for extensive lesions or lesions that are resistant to topical antifungal treatment.¹⁹ ■

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Evaluation of Facial Lesions

Tinea faciei is one of the most commonly misdiagnosed dermatophytoses.⁷ It is characterized by an erythematous, often circular, scaly patch or plaque with a well-defined border on the face (Figures 1 and 2).⁵ As the lesion spreads peripherally, the center often clears and produces the classic annular lesion that is responsible for the designation of ringworm. However, the clinical presentation can be atyp-



Figure 1 – The asymptomatic plaques on the left cheek of a 12-year-old girl did not respond to a cream that her physician had prescribed when the rash began.

(Photo courtesy of Kirk Barber, MD.)



Figure 2 – This itchy, erythematous scaly plaque on the left eyelid of a 10-year-old girl was treated with triamcinolone acetonide cream for 2 weeks, as per her pediatrician's instructions. The rash, shown here 4 weeks after it first appeared, had persisted and worsened.

(Photo courtesy of Smitha Kuppalli, MD and Barbara B. Wilson, MD.)

ical, as in the case described. Tinea faciei is often unilateral, in contrast to our patient's bilateral lesions. It occurs in children often as a result of contact with domestic pets, which our patient denied.

To prevent misdiagnosis of tinea faciei and ensure appropriate treatment, consider a mycological examination in all patients with a discrete centrifugal scaly lesion or atypical, erythematous, scaly plaque on the face.²