

# A man with a widespread pruritic skin eruption

ALEXANDRA SAVAGE BA, MD

REBECCA BRONWYN SAUNDERSON BMedSci(Hons), MB BS(Hons), MPhil(Cantab), FACD

Test your diagnostic skills in our regular dermatology quiz. What is this itchy eruption, which has not responded to topical corticosteroid treatment?

## Case presentation

A 37-year-old man presents with a widespread, intensely pruritic eruption (Figures 1a to c). He reports a three-year history of recurrent eruptions, which began as a small patch on the legs that progressively extended. He has been applying calcipotriol 0.005% with betamethasone dipropionate 0.05% ointment daily, along with emollients.

The patient has a history of asthma. His medications include a regular fluticasone propionate and salmeterol combination inhaler, and he uses a salbutamol inhaler and loratadine as needed. He has no reported allergies or hay fever. There is a family history of eczema but no other dermatological conditions.

On examination, an erythematous eruption is observed that involves the patient's face, trunk and both upper and lower extremities. The eruption is characterised by excoriation, impetiginised crusting, ichthyotic scaling and sharply marginated plaques with a leading edge of scale. The distal skin beyond these lesions appears spared. No nail or mucosal involvement is noted.

## Differential diagnoses

Conditions to consider among the differential diagnoses include the following.

### Plaque psoriasis

Psoriasis is a chronic immune-mediated inflammatory condition that affects 2 to 3% of the population, most commonly people under the age of 50 years.<sup>1</sup> It has a strong genetic predisposition and may be triggered or exacerbated by infections, trauma, stress or medications (e.g. lithium, beta blockers).<sup>1,2</sup> The classic



**Figures 1a to c.** The case patient at presentation. The eruption is characterised by excoriation, areas of impetiginised crusting, ichthyotic scaling and sharply marginated plaques with a leading edge of scale. Images published with patient consent.

form presents as well-demarcated, erythematous plaques covered by silvery-white scale that typically occur on extensor surfaces (elbows, knees), scalp and lower back. Nail involvement (pitting, onycholysis or subungual hyperkeratosis) is common and can serve as a diagnostic clue.<sup>1,3</sup> Although lesions are often symmetrical in distribution, there are differences in morphology and distribution among various subtypes (e.g. guttate, flexural,

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Dr Savage is a Dermatology Research Fellow at Drummoynne Dermatology, Sydney; and Honorary Medical Officer at Prince of Wales Hospital, Sydney. Dr Saunderson is a Visiting Medical Officer at Prince of Wales Hospital, Sydney; and Senior Lecturer in Dermatology, The Northern Clinical School, The University of Sydney, Sydney, NSW.

pustular).<sup>1</sup> Psoriasis tends to respond to appropriate therapy with potent topical corticosteroids.<sup>4</sup>

For the case patient, the erythematous and scaly appearance of the lesions meant psoriasis was an important consideration. However, the progression of the rash despite corticosteroid application and its distribution (including sparing of extensor surfaces) made this a less likely diagnosis.

### Infected dermatitis

Chronic dermatitis, such as atopic eczema or contact dermatitis, is common and secondary bacterial infection is a frequent complication.<sup>5,6</sup> 'Infected dermatitis' refers to an eczematous dermatitis complicated by secondary infection, most often by *Staphylococcus aureus* or *Streptococcus pyogenes* (impetiginised eczema).<sup>5-7</sup> Bacteria colonise the broken skin and can trigger immune reactions that exacerbate inflammation.<sup>8</sup>

Clinically, infected dermatitis typically presents as an acutely worsening rash characterised by increased erythema, warmth and pain, with pustules or oozing honey-coloured crusts over eczematous patches.<sup>8,9</sup> In severe cases, patients may experience systemic symptoms such as fever or malaise.<sup>9</sup> It is signs of infection (purulent crusting and tenderness) that make this clinical picture distinct from psoriasis or simple dermatitis.

For the case patient, infected eczematous dermatitis was an important diagnosis to consider because the eruption had features of chronic dermatitis (pruritus, excoriations, erythema and scale) and had responded poorly to topical corticosteroid treatment. However, there were few classic signs of impetiginisation (serous exudate, swelling, honey-yellow crust), and the sharp margins and extensive nature of the eruption also made this diagnosis less likely.

### Dermatitis herpetiformis

Dermatitis herpetiformis is a chronic immunobullous dermatosis associated with gluten-sensitive enteropathy. It is a rare autoimmune condition in which IgA antibodies directed against epidermal

transglutaminase deposit in the skin.<sup>10</sup> Patients classically present with groups of very itchy vesicles and excoriated papules on extensor surfaces, most commonly the elbows, knees and buttocks.<sup>10</sup> The rash is usually symmetrically distributed and causes intense pruritus, with patients often showing crusted erosions. Dermatitis herpetiformis tends to affect young and middle-aged adults and is more common in Caucasian men. Patients may have accompanying signs of coeliac disease, such as gastrointestinal symptoms or nutritional deficiencies, although many do not.<sup>10,11</sup>

The diagnosis of dermatitis herpetiformis is made by skin biopsy with direct immunofluorescence testing that shows granular IgA deposits in dermal papillae. In addition, many patients will have positive serology for coeliac disease (e.g. anti-transglutaminase, anti-gliadin antibodies, or both).<sup>11</sup>

The case patient's rash demonstrated plaques of varying sizes with a truncal distribution, rather than clusters of small vesicles symmetrically distributed on extensor surfaces, which made dermatitis herpetiformis much less likely. In addition, he did not have any history to suggest gluten sensitivity or coeliac disease.

### Scabies

Human scabies, an infestation by *Sarcoptes scabiei var. hominis*, affects over 200 million people worldwide at any one time.<sup>12</sup> Patients commonly present with an intensely pruritic, papular eruption, often with excoriations. The lesions are typically small erythematous papules with burrows, invisible to the naked eye, distributed in characteristic areas: interdigital web spaces, wrists, axillae, waist, buttocks and genital regions.<sup>13</sup> Infestations that involve the face and scalp are seen in infants or elderly patients but are rare in immunocompetent adults. Outbreaks usually occur in enclosed communities, such as schools or housing units.<sup>14</sup> Multiple family members or contacts may have similar itching.

The diagnosis of scabies is based on clinical assessment and laboratory tests. The visual identification of mites, eggs or burrows in skin scrapings or by dermoscopy is confirmatory.<sup>14</sup>

For the case patient, scabies was an unlikely explanation. The characteristic areas were largely spared, with the eruption involving his face and persisting as a chronic, recurring condition over the past three years. The edge was sharply demarcated, which is less usual in scabies. No mites, eggs or burrows were observed. In addition, his close contacts did not have similar symptoms.

### Tinea incognito

This is the correct diagnosis. Tinea incognito is a dermatophyte infection where morphology has been altered by the use of topical or systemic corticosteroids, calcineurin inhibitors or other immunosuppressants.<sup>15,16</sup> It is usually caused by *Trichophyton rubrum*, *Trichophyton mentagrophytes* or *Microsporum canis* and less commonly by *Epidermophyton floccosum* or *Trichophyton tonsurans*. When these dermatophytes invade keratinised tissues (stratum corneum, hair shafts), they elicit an inflammatory host response that produces the characteristic ring-shaped, scaly, raised border of tinea lesions.<sup>17</sup> However, immunosuppression dampens the inflammation, allowing unrestricted fungal growth, leading to loss of the classic annular appearance. The lesions evolve into ill-defined erythematous plaques with minimal scale, serpiginous borders, follicular pustules or even psoriasiform morphology.<sup>16-18</sup> The result is an infection that can spread beyond its initial borders and penetrate deeper into hair follicles. When use of the corticosteroid or other immunosuppressant is stopped, the rash rebounds with worsening erythema and extension. This cycle can continue for months, leading to an 'incognito' infection that mimics other skin disorders.

The lifetime risk of acquiring tinea corporis is between 10 and 20%, but the true prevalence of tinea incognito is unknown

because lesions are frequently mislabelled as eczema or psoriasis.<sup>18-20</sup> It affects people of all ages but is most common in children and young adults.

Predisposing factors for dermatophytosis increase the risk of tinea incognito. Immunocompromised patients are more susceptible to both primary fungal infections and atypical presentations under corticosteroid therapy.<sup>21</sup> Chronic dermatophyte infections (e.g. untreated tinea pedis or onychomycosis) can act as reservoirs; corticosteroid application to these sites may trigger tinea incognito. Other risk factors include excessive sweating, occlusive clothing and close contact with infected people or animals.<sup>22</sup> It is more prevalent in warmer regions with high humidity.<sup>19</sup>

### Diagnosis

Tinea incognito is a great imitator. The borders of the lesions are smudged, scaling is minimal, colour may be less erythematous or hypopigmented and pustules can develop at the periphery.<sup>19</sup> Lesions often spread more extensively than in typical tinea corporis and pruritus is variable – often suppressed during corticosteroid use but flaring on withdrawal.<sup>19,21</sup> Clues include satellite lesions, ‘ring-within-a-ring’ patterns, concurrent tinea pedis or onychomycosis, as well as signs of prior corticosteroid use such as atrophy, striae or telangiectasia in sites atypical for eczema.<sup>17,20</sup> In practice, it is important to be alert to recurrent or chronic rashes treated repeatedly with corticosteroids (especially in atypical locations for eczema) and patient self-medication with corticosteroid creams. Other sites (feet, nails, scalp, groin) should be examined for fungal reservoirs, and epidemiological clues such as exposure to infected pets or household contacts should be considered.<sup>20</sup> Recognising these factors can prevent misdiagnosis and inappropriate corticosteroid use.

Although microscopy and culture of skin scrapings, subungual debris, nail clippings or plucked hair was previously the quickest test to confirm the diagnosis,

polymerase chain reaction (PCR) tests are surpassing traditional diagnostic methods.<sup>23,24</sup> Dermoscopy may reveal peripheral ‘ruff’ or ‘moth-eaten’ scale, comma or corkscrew hairs. Skin biopsy with periodic acid-Schiff staining is reserved for culture-negative cases with high suspicion.<sup>20</sup> An empirical antifungal trial can be diagnostic when testing is delayed, but objective confirmation is preferred to guide treatment and prevent mismanagement.<sup>17</sup>

### Management

Management of tinea incognito encompasses two main principles: discontinuing misuse of topical corticosteroids or immunosuppressants and eliminating the fungus.

#### Discontinuing corticosteroids or immunosuppressants

Use of the offending corticosteroid or calcineurin inhibitor should be ceased immediately. Patients should be advised not to apply any corticosteroid creams to the area and made aware that lesions might become temporarily more inflamed or pruritic (a rebound effect as the immune response recovers) after stopping a potent corticosteroid. To manage this, adjunctive measures such as cool compresses can be used. Oral antihistamines can provide relief for severe itch without compromising infection clearance.

#### Eliminating the fungus

The choice between topical and oral antifungal treatment for tinea depends on the site, severity and response to any previous therapy. Topical agents are suitable for early, localised infections affecting the body, face, groin or web spaces between the fingers or toes.<sup>22</sup> All sites of infection should be addressed to prevent recurrence.

#### Topical antifungal therapies

The topical antifungal agents that are used to treat tinea incognito are terbinafine 1% cream and gel, and imidazoles (clotrimazole 1%, miconazole 2%, econazole 1%, ketoconazole 2%, bifonazole 1% creams).

Terbinafine has fungicidal activity and can often clear dermatophyte infections faster: a 2014 Cochrane review of treatments for tinea corporis and tinea cruris found that both imidazoles and allylamines are effective, with terbinafine achieving slightly higher cure rates and leading to faster symptom resolution in some cases.<sup>25</sup> Terbinafine is the preferred topical agent for most superficial fungal infections because of its shorter treatment duration, once-daily application and potential for faster cure as advantages, which can improve patient adherence.<sup>22</sup> Terbinafine cream is applied once daily for at least two to four weeks, whereas azoles are usually applied twice daily for four weeks or more.<sup>23</sup>

Regardless of the topical agent chosen, patients should be advised to apply the cream to the entire lesion and 1 to 2 cm beyond its margin and to continue for at least two weeks beyond visible resolution to ensure full eradication. Longer treatment duration is often needed for patients with tinea incognito. They should also refrain from using corticosteroid–antifungal combination products.<sup>23</sup>

#### Oral antifungal therapies

The Australian *Therapeutic Guidelines* recommend oral therapy for any tinea that has been modified by topical corticosteroid use, as well as for infection that is extensive, persistent despite treatment, recurring quickly or affecting difficult-to-treat areas such as the scalp, palms or soles.<sup>22</sup>

Terbinafine is the first-line oral therapy and is associated with a lower risk of drug interactions compared to azoles (fluconazole and itraconazole) as it causes less inhibition of cytochrome P450 enzymes.<sup>22,23</sup> The recurrence rates are similar: 33.3% for terbinafine and 37% for azoles at 10 to 13 months follow up.<sup>22</sup> The duration of therapy depends on site – for a typical *T. rubrum* infection on glabrous skin, a two-week course of terbinafine 250 mg daily is often sufficient (shorter duration than for tinea of nails or hair). Duration for tinea incognito is two to four weeks, with clinical review as a longer treatment

**TABLE. SYSTEMIC ANTIFUNGAL THERAPIES FOR TINEA<sup>22,23,25</sup>**

Drug	For skin tinea	For scalp tinea	For nail tinea
Terbinafine	<b>Dosage</b> Adult: 250 mg, once daily Child <20 kg: 62.5 mg, once daily Child 20 to 40 kg: 125 mg, once daily	<b>Dosage</b> Adult: 250 mg, once daily Child <20 kg: 62.5 mg, once daily Child 20 to 40 kg: 125 mg, once daily	<b>Dosage</b> Adult: 250 mg, once daily Child <20 kg: 62.5 mg, once daily Child 20 to 40 kg: 125 mg, once daily
	<b>Duration</b> 2 weeks	<b>Duration</b> 4 weeks	<b>Duration</b> Until clinical clearance
Fluconazole	<b>Dosage</b> Adult: 150 mg, once weekly Child: specialist advice	<b>Dosage</b> Adult: 6 mg/kg (max 400 mg), once daily Child: 6 mg/kg, once daily, or 8 mg/kg, once weekly	<b>Dosage</b> Adult: 150 to 300 mg, once weekly Child: specialist advice
	<b>Duration</b> 6 weeks	<b>Duration</b> 3 to 6 weeks (daily dosing) or 8 to 12 weeks (weekly dosing)	<b>Duration</b> Until clinical clearance
Itraconazole* (conventional capsules)	<b>Dosage</b> Adult: 100 mg, once daily Child ≥20 kg: 100 mg, once daily	<b>Dosage</b> Adult: 200 mg, once daily Child: 3 to 5 mg/kg (max 200 mg), daily	<b>Dosage</b> Adult: 200 mg, twice daily for one week per month (pulse) Child: specialist advice
	<b>Duration</b> 2 weeks for tinea corporis or tinea cruris, 4 weeks for tinea pedis	<b>Duration</b> 4 to 6 weeks	<b>Duration</b> Until clinical clearance
Itraconazole* (Lozanoc)	<b>Dosage</b> Adult: 50 mg, once daily Child: specialist advice	<b>Dosage</b> Adult: 100 mg, daily Child: 3 to 5 mg/kg (max 100 mg), daily	<b>Dosage</b> Adult: 100 mg, twice daily for one week per month (pulse) Child: specialist advice
	<b>Duration</b> 2 weeks for tinea corporis or tinea cruris, 4 weeks for tinea pedis	<b>Duration</b> 4 to 6 weeks	<b>Duration</b> Until clinical clearance
Griseofulvin†	<b>Dosage</b> Adult: 500 mg, once daily Child >1 month: 10 mg/kg (up to 500 mg), once daily	<b>Dosage</b> Adult: 20 mg/kg (max 500 mg), once daily Child: specialist advice	<b>Dosage</b> Adult: 500 to 1000 mg, once daily Child: specialist advice
	<b>Duration</b> 8 to 12 weeks	<b>Duration</b> 6 to 8 weeks when <i>Microsporum</i> species identified	<b>Duration</b> Until clinical clearance

\* Lozanoc has improved bioavailability and is not interchangeable with conventional capsules. The same brand of itraconazole should be used throughout course.

† Less preferred due to slower clearance and resistance patterns.

course may be required. Use of systemic antifungal therapies is described in the Table.<sup>22,23,25</sup>

Monitoring requirements differ for oral antifungal therapies. For terbinafine, routine blood monitoring is not required in healthy patients receiving short courses (six weeks or less). Baseline liver function testing and follow-up monitoring should be arranged if treatment is prolonged (over six weeks), if the patient has pre-existing

liver disease or risk factors, or if clinical symptoms suggest hepatotoxicity. Itraconazole therapy requires baseline and regular liver function monitoring.<sup>22,26</sup> Fluconazole requires baseline liver function testing and a full blood count, but no repeat testing is needed for once-weekly regimens.<sup>22,25</sup> Terbinafine should be used cautiously in patients with psoriasis or lupus, as it may exacerbate these conditions.<sup>22,23,27</sup> Fluconazole is contraindicated

in severe liver disease and may prolong the QT interval, whereas itraconazole should be avoided in heart failure and may be poorly absorbed when coadministered with proton pump inhibitors.<sup>22</sup>

#### Other measures

All patients should be educated about general skincare measures to help prevent reinfection, with the affected area kept clean and dry. Emollients can help restore

barrier function. Loose, breathable clothing (e.g. cotton fabrics) should be encouraged and occlusive attire or footwear that promotes sweating avoided.

If secondary bacterial infection is present then a topical or systemic antibiotic might be needed, but this is case-dependent. Generally, once antifungal therapy is commenced and the corticosteroid is stopped, the skin will become temporarily more inflamed and then progressively improve as the fungus clears. Follow up should be arranged in two to four weeks to ensure the rash is improving, with reduced scaling and erythema and regression of the edges of the lesion.

### Outcome

For the case patient, further investigations were undertaken given the extensive distribution of the eruption and poor response to topical corticosteroid therapy. Fungal scrapings and a 4 mm punch biopsy were performed at the active margin of a lesion on the lower back. The diagnosis of tinea incognito was confirmed by culture of *Trichophyton rubrum* from the scraping. Histopathology demonstrated intracorneal neutrophilic aggregates, a solitary intracorneal fungal hyphal element, mild acanthosis, spongiosis, and superficial perivascular infiltrates composed of eosinophils and neutrophils. These features were consistent with a psoriasiform tissue reaction pattern (resembling psoriasis), but importantly fungal hyphae were identified within the stratum corneum. A superficial swab taken from a wound on the leg with crusting for bacterial microscopy and culture, grew heavy *Staphylococcus aureus* and *Streptococcus dysgalactiae*.

The patient commenced treatment with oral terbinafine 250 mg daily as well as a course of cephalexin (500 mg three times daily for 14 days) for the secondary bacterial infection. He was also advised to use emollients. Hepatic function testing was scheduled for eight weeks.

At review three months later, substantial clinical improvement was observed and the patient reported no adverse effects

from terbinafine. Examination revealed marked resolution of the eruption, although residual erythematous patches with lichenification and excoriation persisted, particularly in the popliteal fossae and on the trunk. Onychomycosis of the toenails was noted and xerosis remained pronounced, so he was advised to continue oral terbinafine and the need to apply emollients was frequently emphasised. Repeat blood tests were requested and a further review was scheduled in three months for ongoing monitoring.

### Key point

The pathogenesis of tinea incognito involves an iatrogenic factor (corticosteroid or immunomodulator treatment) superimposed on a common fungal infection, creating an altered host-pathogen interaction. This mechanism underscores why proper diagnosis of the initial eruption is crucial, as treating an undiagnosed fungal infection with corticosteroids ‘feeds’ the fungus by disarming the skin’s defences. **MT**

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