A Challenging Case of Recalcitrant Facial and Disseminated Rash in a Patient with Autoimmune Comorbidities

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What's Your Diagnosis?

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Introduction. A 35-year-old woman with systemic lupus erythematosus (SLE), antiphospholipid syndrome (APS), chronic urticaria, asthma, Hashimoto thyroiditis, and recurring seizures presented to dermatology with a 3-month rash.

History. The scaly, erythematous rash started on her abdomen and spread to her shins, groin, wrists, arms, and face. Previous treatments with clobetasol, hydrocortisone, 0.025% triamcinolone, and clotrimazole reduced itching but failed to clear the rash.

She was recently hospitalized for a seizure and discharged on lamotrigine extended release 200 mg two times a day (BID) and levetiracetam 1000 mg BID for maintenance therapy. Recent laboratory examinations show normal C3, C4, and anti-dsDNA, with positive anticardiolipin antibody, beta-2 glycoprotein, and lupus anticoagulant. The patient has a history of pregnancy loss and pulmonary embolism and is on warfarin. She lives with a cat that has been ill recently from a skin infection. Her medical history includes hydroxychloroquine treatment for SLE and APS. The patient was a fall risk because of her recent seizures.

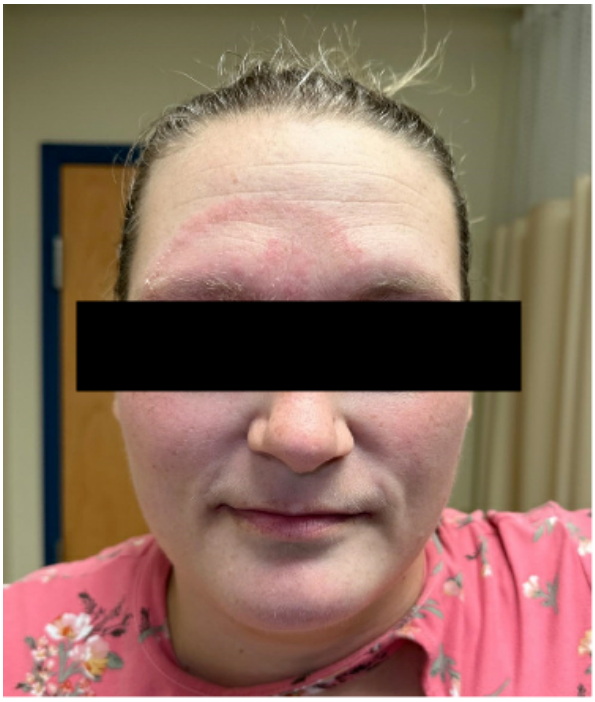

Figure 1. *The patient presented with a rash that started on her abdomen, but spread to her face as well as her shins, groin, wrists, and arms.*

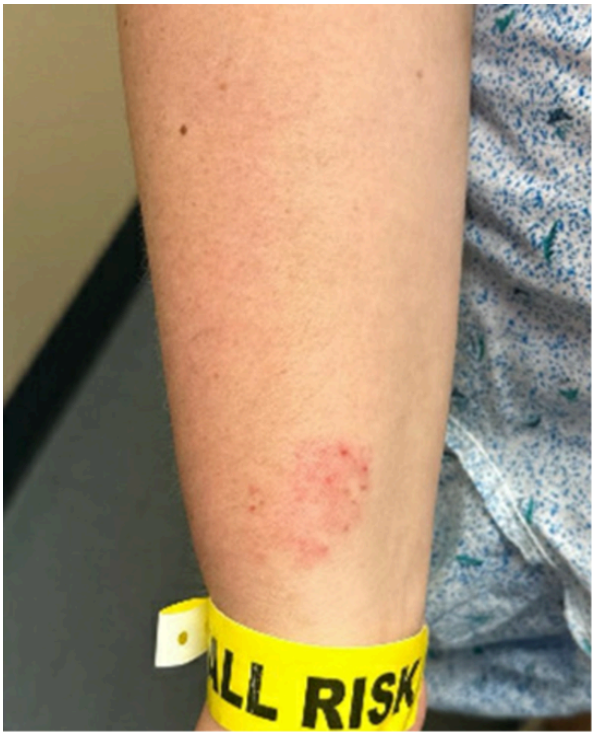

***Figure 2**. The rash was present on both wrists.*

The patient's physical examination reveals erythematous, scaly, well-demarcated plaques involving the forehead, bilateral eyebrows (hair follicles), nasal bridge, and periorbital regions (**Figure 1**). The distribution appears to spare the nasolabial folds. There is mild desquamation noted within the affected areas, and no signs of ulceration, crusting, or pustules are evident. The rash is symmetrically distributed. The rash is also present on her groin, legs, wrist (**Figure 2**), and abdomen (**Figure 3**).

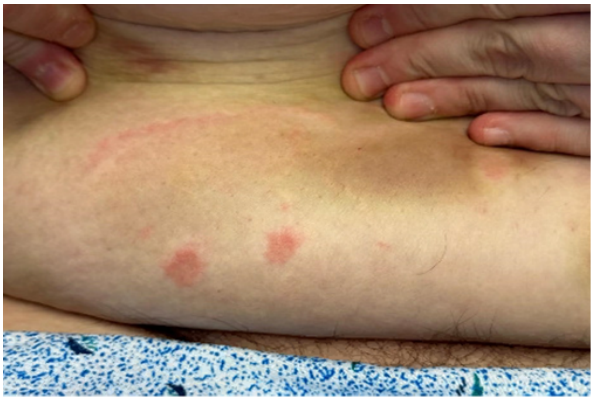

***Figure 3**. A rash can be seen on lhe patient's lower abdominal fold.*

Diagnostic Testing. Skin biopsy was performed on patient's abdomen and wrist. Further diagnostic testing included a potassium hydroxide (KOH) stain **(Figure 4).**

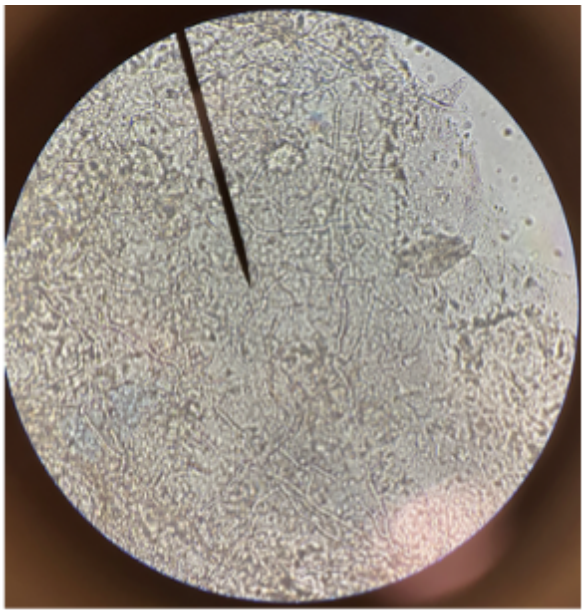

Figure 4. *Diagnostic testing included a potassium hydroxide (KOH) stain*

Correct Answer: B. Tinea faciei

Tinea faciei is the most likely diagnosis, given the positive KOH findings of septate hyphae. The patient most likely was exposed to tinea faciei from her cat, who she reported had an unknown skin infection. Tinea faciei can be spread by direct contact with an infected person or animal[1].

Lupus erythematous was originally considered, given the past medical history of flares. However, the current treatment regimen failed to alleviate her symptoms. Demodex folliculitis and seborrheic dermatitis involving other body parts outside of the head are both rare and less likely.

Treatment and management. Given the widespread infection, the patient was prescribed oral terbinafine 250 mg taken daily for 2 weeks, and her topical steroid was discontinued. Prior treatment of clotrimazole (dosage and duration was unknown) did not eliminate the dermatophyte.

Due to the possibility of lupus erythematous, skin biopsy was performed on the abdomen and wrist. Histopathology revealed no evidence of lupus erythematous; spongiosis of epidermis and parakeratosis was seen and the biopsy was positive for fungal elements after staining with Periodic acid–Schiff (PAS).

Outcome and follow-up. The patient adhered to her treatment regimen when assessed in a follow-up visit 6 weeks later and did not experience adverse effects or unanticipated events.

Discussion. Tinea faciei is a dermatophyte infection of the face that typically presents as flat, scaly macules or patches with raised borders and an annular configuration. Diagnosing tinea faciei can be difficult because up to 70% of lesions are misdiagnosed as lupus erythematosus, seborrheic dermatitis, rosacea, or granuloma annulare.[1,2] The clinical presentation can become further complicated when treated with corticosteroids, as seen in our patient. Topical steroid use alters the characteristic clinical appearance of the lesion, making it resemble other skin disorders—a phenomenon known as tinea incognito.[3] Additionally, topical steroids can spread the infection to hair follicles, including the eyebrows, as was observed in our patient (**Figure 1**).

A retrospective cohort analysis of 200 cases of tinea incognito in Italy found that the disease frequently mirrored lupus, eczema, or rosacea on the face, as well as eczema-like lesions on the trunk and limbs.[4] This overlap in clinical appearance may obfuscate the diagnosis, prolonging favorable clinical outcomes. In our patient, past clotrimazole treatment may have removed the dermatophyte from the stratum corneum but left fungal spores in the hair follicles potentially extending the rash symptoms.[5] Although tinea faciei is typically easy to treat, its ability to mimic other skin disorders can result in delayed diagnosis and prolonged disease. Oral terbinafine is the first-line treatment for extensive tinea infections due to its tolerability, high cure rate, and low cost.[6]

The histopathologic features of lupus erythematosus and tinea infections differ significantly, providing diagnostic clarity when necessary. On histology, cutaneous lupus can show liquefactive vacuolar degeneration of basal cells, thickened basement membranes, dermal mucin deposition, perivascular infiltrates of lymphocytes, histiocytes, and plasma cells, follicular plugging, and telangiectasia.[7] In contrast, dermatophyte infections may exhibit compact orthokeratosis, epidermal spongiosis with mononuclear cell and neutrophil infiltration, and fungal hyphae in the cornified layers.[8] While a biopsy is valuable for diagnosing cutaneous lupus erythematosus when suspicion is high, its sensitivity may be limited in detecting hyphae, making KOH preparation, as shown in Figure 4 with branching, septate hyphae in clusters and chains, a preferable initial test in suspected fungal infections due to its cost-effectiveness, non-invasiveness, and rapid results[6].

Hematoxylin and eosin staining may lack the sensitivity needed to detect fungal hyphae, potentially delaying diagnosis. Therefore, PAS staining is recommended to confirm the presence of hyphae.[8] While demodex folliculitis may clinically resemble tinea faciei, the application of a KOH stain would detect demodex mites. Furthermore, demodex folliculitis seldom causes symptoms outside of the face.

This case highlights the diagnostic challenge of tinea faciei. Differential diagnoses may include cutaneous lupus erythematous, demodex folliculitis, and seborrheic dermatitis. Early recognition is crucial to prevent mismanagement, such as the continued use of a topical steroid for tinea. Physicians should consider fungal infection in atypical facial lesions unresponsive to therapies as part of their differential.

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