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Crusted scabies in an immunocompromised host presenting as erythrodermic psoriasis: a diagnostic pitfall

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Dear Editor,

Crusted scabies (CS) is a severe and highly contagious variant of scabies caused by *Sarcoptes scabiei* var. *hominis*, characterized by the presence of millions of mites, unlike classic scabies, where infestations are typically limited to 10-15 mites.[1] It may occur in immunocompromised or debilitated individuals[2] and can present with thick hyperkeratotic plaques that, in certain cases, evolve into erythroderma, often mimicking chronic dermatoses such as psoriasis. The absence of pruritus, frequently observed in CS,[3] may further contribute to diagnostic delay. We describe a case of CS initially suspected to be erythrodermic psoriasis, highlighting a distinctive dermoscopic pattern.

A 63-year-old Caucasian male presented with generalized erythroderma. Physical examination revealed widespread, thick, friable yellow-brown scaly crusts with a "dirty", sand-like appearance. Lesions involved the proximal limbs, back, peri-areolar region, abdomen, pubis, glans penis, face, scalp, and the dorsal areas of the hands and feet, with sparing of skin folds (Figure 1). On the dorsal hands, the crusts appeared particularly thick and brownish, causing mechanical entrapment of the fingers and imparting an "armadillo-like" appearance. Fissures were present over extensor surfaces, while palms and nails remained unaffected.

The patient reported that the condition had developed approximately 2 months earlier, coinciding with an unintentional weight loss of about 15 kg. He denied systemic or cutaneous symptoms, including pruritus. His past medical history included chronic plaque psoriasis treated with methotrexate 15 mg/week and folic acid for approximately 11 months. The brownish hue of the crusts, the peri-areolar involvement, and the sparing of palms and nails raised clinical suspicion for CS.

Dermoscopy confirmed the diagnosis by revealing numerous mites. Multiple intersecting burrows containing adult mites and eggs produced a striking dermoscopic pattern, which we termed the "railway network" sign (Figure 2). This feature proved particularly helpful in differentiating CS from erythrodermic psoriasis, a frequent diagnostic pitfall suggested by the patient's history.

CS, formerly known as Norwegian scabies, is caused by a massive infestation of *S. scabiei* var. *hominis*. It represents a severe and highly contagious form of scabies, characterized by millions of mites, in contrast to classic scabies, where infestations are typically limited to 10-15 mites.[1] CS is more commonly observed in immunocompromised individuals (both congenital immunodeficiency disorders and AIDS) or debilitated patients affected by malnutrition, mental retardation, physical decline, and poor hygiene.[2] Organ transplant recipients or patients with graft-*versus*-host disease, malignancies, or systemic diseases such as diabetes mellitus or systemic lupus erythematosus are also more susceptible.

Long-term immunosuppressive treatment with corticosteroids or methotrexate represents an additional risk factor. Several studies have identified a dysregulated Th1/Th2 profile in CS, characterized predominantly by an impaired Th2-mediated response. Whereas in classical scabies parasite proliferation is controlled by both cellular and humoral immunity, in CS parasite proliferation remains uncontrolled despite markedly elevated IgE levels and peripheral blood hypereosinophilia. Histopathological examination consistently demonstrates an absence of B lymphocytes and parasite-specific antibodies, accompanied by a dense T-cell infiltrate with a markedly increased $CD8^+/CD4^+$ ratio. The precise contribution of cytotoxic T cells remains unclear; however, their potential cytopathic activity on keratinocytes may partially account for the intense inflammatory response observed in this condition.[4]

Clinically, CS presents with widespread thick hyperkeratotic plaques that may mimic chronic dermatoses such as psoriasis or eczema. In certain cases, the infestation may evolve into erythroderma. Pruritus may be mild or absent due to impaired cellular immunity.[3] Secondary bacterial infections and loco-regional lymphadenopathy are common, and immunocompromised individuals are at heightened risk for systemic complications.

A comprehensive differential diagnosis is essential, as erythrodermic CS may resemble erythrodermic psoriasis or pityriasis rubra pilaris, both of which share overlapping features.[5] Erythrodermic psoriasis typically presents with diffuse erythema, fine whitish scaling, and nail involvement, whereas pityriasis rubra pilaris shows orange-red plaques, follicular prominence, and wax-like palmar keratoderma. Other differential diagnoses include severe atopic dermatitis, drug-induced erythroderma, cutaneous T-cell lymphomas, and pemphigus foliaceus.

We initiated treatment with oral ivermectin (3 tablets of 3 mg as a single dose, 200 μg/kg/day) combined with topical permethrin 5% applied for two consecutive nights, repeating the protocol weekly. After two cycles, the patient showed marked improvement, with significant reduction of erythroderma and complete resolution of crusted lesions.

Delayed diagnosis may lead to serious complications, including impetiginization, cellulitis, and sepsis. Aggressive combination therapy is therefore essential. The German S1 guidelines recommend oral ivermectin together with topical scabicides.[6] European guidelines indicate daily application of permethrin 5% or benzyl benzoate 25% for the first 7 days, followed by twice-weekly applications until resolution, combined with ivermectin on days 1, 2, and 8; additional doses may be required in refractory cases.[7] Given the high transmissibility of CS, patient isolation is strongly recommended. Secondary bacterial infections, particularly those caused by *Streptococcus pyogenes* or *Staphylococcus aureus*, require prompt antibiotic therapy.[8]

In conclusion, this case highlights the diagnostic challenge posed by this atypical form of scabies, which may present with erythroderma, leading to a broad differential diagnosis. Early diagnosis is crucial, given the highly contagious nature of the disease and the compromised general condition of affected patients. Prompt treatment is essential to prevent potentially fatal complications and should rely on a combined systemic and topical approach.

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Figure 1. Erythroderma with diffuse yellow-brown scaly crusts, sparing the skin folds (groin, navel). A similar presentation is also observed on the back, with areas of erythrodermic skin mixed with scaly crusts. Characteristic involvement of the peri-areolar region. “Armadillo-like” encasement of the dorsum of the hand with sparing of the palm and nails.



Figure 2. Dermoscopic details with a “railway track” appearance showing the intersection of multiple burrows. Note the presence of at least 5-10 mites (in black) per burrow.

