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Beyond Psoriasis: Recognizing Crusted Scabies in Patients with Communication Barriers

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Introduction
Crusted (Norwegian) scabies is a rare, highly contagious form of scabies caused by infestation with the ectoparasite Sarcoptes scabiei var. hominis. In patients with Down syndrome, immunological aberrations - specifically T-cell abnormalities - and altered pruritic perception facilitate massive parasitic proliferation [2,3]. Clinically, crusted scabies may overlap with or mimic other dermatologic disorders, including psoriasis, eczema, seborrheic dermatitis, lichen planus, pityriasis rubra pilaris, and cutaneous lymphoma. In high-risk patients, pre-existing dermatoses often obscure the clinical picture, making differentiation between a psoriasis flare and secondary infestation particularly challenging [1]. Our case illustrates how the inability to report pruritus due to severe intellectual disability, combined with pre-existing psoriasis, resulted in a significant diagnostic delay and subsequent clinical deterioration.

Materials and Methods
A case report.

Results
A 35-year-old female with Down syndrome and severe intellectual disability was referred for evaluation of progressive skin lesions. Due to her cognitive impairment, the patient was unable to communicate or characterize subjective complaints; however, skin lesions had been observed by caregivers since 2010, when the patient was diagnosed with psoriasis. Physical examination revealed widespread erythematous plaques on the scalp, trunk, and extensor surfaces of the limbs (BSA >20%), accompanied by prominent palmoplantar keratoderma and dystrophic, hyperkeratotic nails. In the absence of a reported pruritus, the presentation was initially interpreted as an exacerbation of psoriasis vulgaris. Treatment with calcipotriol/betamethasone foam and systemic acitretin 20 mg/day was initiated. Although a transient clinical response was observed, the patient's condition subsequently regressed, prompting an increase in acitretin to 30 mg/day. Despite dose escalation, skin lesions progressed to erythroderma with extensive exfoliation. Biological therapy with adalimumab was then initiated; however, following the first administration, the patient developed a febrile fever and was diagnosed with pneumonia, necessitating the immediate cessation of the TNF-alpha inhibitor. Due to treatment resistance and worsening of a clinical skin condition, a 5 mm punch biopsy was performed. Histopathological analysis of the skin biopsy revealed massive *Sarcoptes scabiei* infestation within the stratum corneum, accompanied by serum crusts (plasma inclusions) and neutrophilic infiltrates. The epidermis exhibited significant acanthosis and spongiosis. In the superficial dermis, a prominent perivascular inflammatory infiltrate consisting of lymphocytes, plasma cells, and eosinophils was observed. These findings are pathognomonic and confirm the definitive diagnosis of crusted (Norwegian) scabies. Antiscabietic therapy was initiated with topical permethrin 5% for seven consecutive days, combined with oral ivermectin 200 micrograms/kg on days 1, 2, and 8. Following successful parasitic eradication, guselkumab was introduced to manage the underlying psoriasis.

Conclusions

Crusted scabies frequently masquerades as refractory psoriasis in patients with Trisomy 21, driven by underlying immunological and sensory aberrations that facilitate hyperinfestation. Treatment-resistant dermatosis in high-risk patients necessitates urgent diagnostic re-evaluation. Microscopic or histopathological screening is mandatory to optimize therapeutic outcomes, ensure the appropriate use of systemic agents, and maintain epidemiological safety within healthcare environments.