Eleven Years of Itching: A Case Report of Crusted Scabies

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Practice Points

- Crusted scabies can mimic a variety of conditions such as psoriasis, eczema, seborrheic dermatitis, and contact dermatitis. Therefore, suspicion is the prerequisite for disease control.
- Scabies usually is found in individuals with a compromised immune system as well as those with decreased sensory functions. Thus patients should be investigated for an underlying immunodeficiency.
- Treatment can be challenging, and effective management of the condition requires a keratolytic agent in conjunction with a scabicidal agent.

Crusted scabies is a rare and highly contagious form of scabies that is characterized by uncontrolled proliferation of mites in the skin, extensive hyperkeratotic scaling, crusted lesions, and variable pruritus. We report the case of a 48-year-old man with an 11-year history of pruritic, hyperkeratotic, psoriasiform plaques and widespread erythematous papules that was diagnosed as crusted scabies.

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Case Report

A 48-year-old man presented to our dermatology clinic with pruritus of 11 years' duration that worsened at night. He had been followed at a different clinic for several years and was unsuccessfully treated with topical permethrin and oral antihistamines on multiple occasions for scabies. He also had been intermittently treated for contact dermatitis with topical and systemic steroids, which also brought no relief. Just prior to his presentation, the patient's wife and 8-year-old son had sought medical attention at our institution for chronic pruritus and elevated IgE levels. They had been unsuccessfully treated with topical permethrin, topical steroids, and oral antihistamines for atopic dermatitis at a different clinic. When they presented to our clinic, they were both diagnosed with and treated for scabies. At this visit the patient revealed similar concerns and subsequently underwent examination.

Physical examination revealed large erythematous, hyperkeratotic, scaly plaques on the gluteal fold, umbilicus, glans penis, scrotum, bilateral elbows, knees, nipples, and ear helices (Figure 1). Numerous small erythematous papules and wavy threadlike gray burrows measuring 1 to 10 mm in diameter were distributed primarily around the wrists, ankles, proximal extremities, abdominal and pubic area (Figure 2), and interdigital spaces. Wide oval patches of nonscarring alopecia developed on the scalp, and atrophic glossitis and angular cheilitis were noted on the oral mucosa, along with a white pseudomembranous exudate on the palatum. The patient's nails also were thickened and discolored (Figure 3). His medical history was remarkable for hypoparathyroidism (42 years), alopecia areata (15 years), oral candidiasis and angular cheilitis (10 years), and primary hypothyroidism (1 year) that was currently being treated with levothyroxine.

Laboratory studies revealed normal hemogram with an eosinophil level of 0% (reference range, 0.9%–6%). Biochemistry and hormone profiles were consistent with hypoparathyroidism, with the following levels: serum calcium, 7.6 mg/dL (reference range, 8.4–10.2 mg/dL); phosphorus, 5.1 mg/dL (reference range, 2.4–4.4 mg/dL); and parathyroid hormone, 5.23 pg/mL (reference range, 15–65 pg/mL). The patient tested negative for human immunodeficiency virus.

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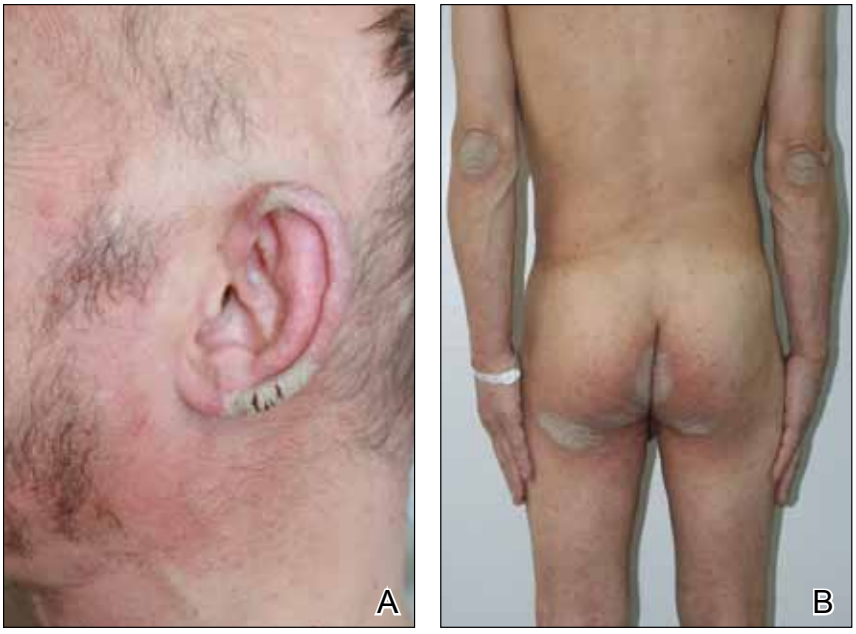

Figure 1. Large erythematous, hyperkeratotic, scaly plaques on the ear helices (A), gluteal fold, and bilateral elbows (B) in a 48-year-old man with crusted scabies.

Dermoscopic examination of the gray threadlike burrows revealed distinctive brown-colored triangular structures that were removed with a fine scalpel. Microscopic examination of the tissue revealed moving mites, eggs, and red-brown scybala.

A biopsy was taken from the thick, scaly, crusted white plaques in the gluteal area. The epidermis showed massive hyperkeratosis and burrows in the subcorneal layer containing a large number of female mites and feces, while the remainder of the epidermis was substantially psoriasiform. A substantial lymphohistiocytic infiltrate with numerous eosinophils was seen diffusely throughout the dermis, though it was most prominent within the upper half (Figure 4). The patient subsequently was diagnosed with crusted scabies.

Crusted scabies typically develops in patients with defective T-cell immunity or a reduced ability to mechanically debride the mites. Because our patient had a history of persistent oral candidiasis, further investigation for immunosuppression was initiated. An immunosuppression panel revealed low IgA levels (46 mg/dL [reference range, 82–453 mg/dL]), low absolute CD8 level (145 cells/μL [reference range, 300–1800 cells/μL), and CD4:CD8 ratio of 4.1. Serum IgG, IgM, IgE, complement levels, and immunoelectrophoresis were within reference range. Abdominal ultrasonography was unremarkable. Taking into account the patient's history of autoimmune hypoparathyroidism, hypothyroidism, oral candidiasis, and alopecia areata, he was diagnosed with autoimmune polyglandular syndrome.

The patient and his family were successfully treated with modified Wilkinson ointment

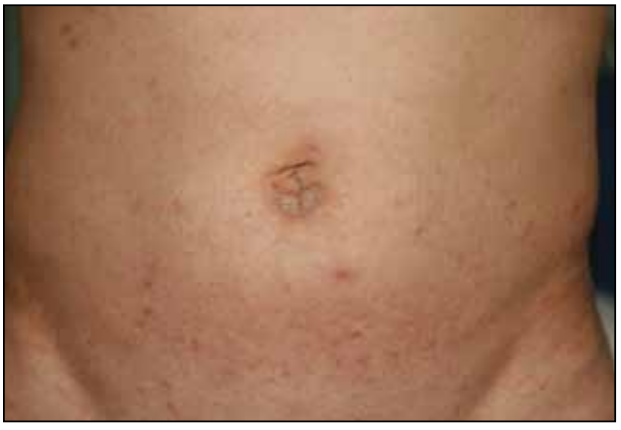

Figure 2. Numerous small erythematous papules and wavy, threadlike, grayish, 1- to 10-mm burrows distributed in the abdominal and pubic area.

Figure 3. Thickened and discolored nails in a 48-year-old man with crusted scabies.

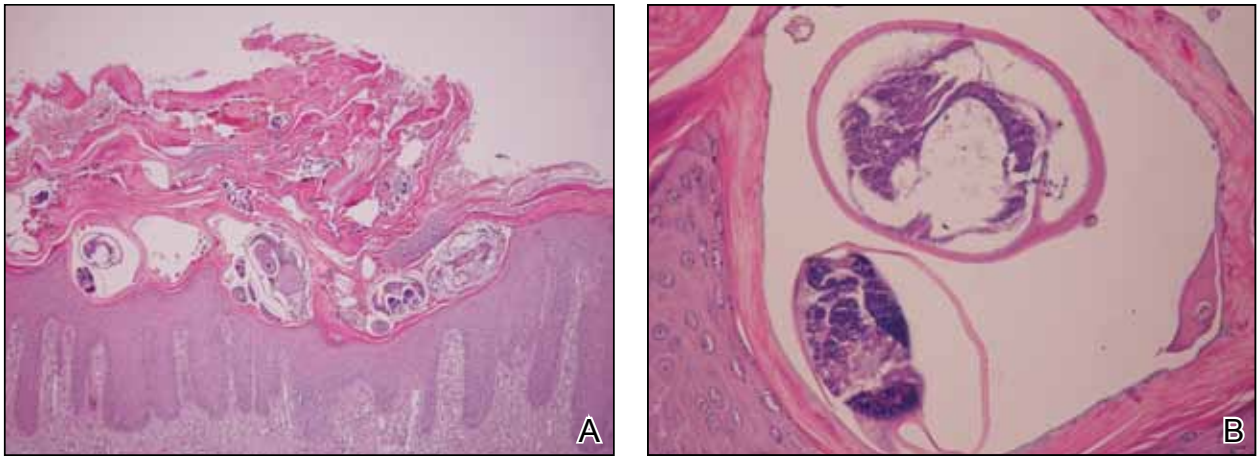

Figure 4. The psoriasiform epidermis showed massive hyperkeratosis and burrows in the subcorneal layer containing a large number of female mites and feces (A)(H&E, original magnification ×40). A substantial lymphohistiocytic infiltrate with numerous eosinophils was seen diffusely throughout the dermis, though most prominently within the upper half. High-power examination demonstrated mites in the burrow (B)(H&E, original magnification ×400).

(goudron végétal 12.5%; sulfur 12.5% in petrolatum) for 3 consecutive days. Within 1 week, the scaly plaques had disappeared and the erythematous papules had faded. The pruritus had resolved and no new papules emerged. Treatment was reapplied once more the following week and complete cure was achieved. We have been following this family for 12 months and no recurrences have occurred.

Comment

Crusted scabies is a rare and highly contagious form of scabies that is characterized by uncontrolled proliferation of mites in the skin, extensive hyperkeratotic scaling, crusted lesions, and variable pruritus.[1] The stratum corneum thickens and forms warty crusts as a reaction to the high mite burden.[2] The uncontrolled proliferation of mites in the skin typically develops in patients with a defective T-cell response or decreased cutaneous sensation and reduced ability to mechanically debride the mites.[3] Patients should be investigated for a predisposition to crusted scabies due to an underlying condition. Crusted scabies also has been shown to develop in Australian natives with normal immunity, though the etiology of the increased susceptibility in this patient population remains unclear. Some studies have shown an association with HLA-A11.[4,5] It also has been hypothesized that these patients may have a specific immunodeficiency predisposing them to hyperinfestation.[6]

Unlike classic scabies, crusted scabies usually does not present acutely, and it usually is insidious at onset. The eruption typically has 2 components: localized horny plaques and a more distinct erythema.[3] Crusted scabies can mimic a variety of conditions such as psoriasis, eczema, seborrheic dermatitis, Darier disease, contact dermatitis, and pityriasis rubra pilaris.[7] When pruritus is resistant to permethrin therapy, as in our patient, crusted scabies often is misdiagnosed as eczema or contact dermatitis. Topical and systemic corticosteroids often are prescribed, causing progression to scabies incognito.

The diagnosis of crusted scabies is confirmed by examination of scrapings and biopsies, as in classic scabies; however, treatment can be challenging due to compromised immunity, a large mite burden, and limited penetration of topical medications into the hyperkeratotic lesions. Thus treatment should include both keratolytic and scabicidal agents to remove the crusts, reduce the mite load, and enhance the scabicidal therapy.[1] Our patient and his affected family members had previously been treated with topical permethrin several times without any benefit. Oral ivermectin has been proven to be effective but is not available in Turkey. Therefore, we treated the patient and his household contacts (other extended family members treated separately) with modified Wilkinson ointment (goudron végétal 12.5%; sulfur 12.5% in petrolatum) for 3 consecutive days, which is known to have both a keratolytic and scabicidal effect.[8-11]

Conclusion

This case highlights the importance of obtaining a complete family history, skin examination, and

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thorough investigation for underlying immunodeficiencies that can lead to a predisposition for crusted scabies. It is important to note that the treatment of crusted scabies can be challenging, and effective management of the condition requires a keratolytic agent in conjunction with a scabicidal agent.

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