Misdiagnosis of Crusted Scabies: Skin Excoriations Resembling Brown Sugar Are Characteristic

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PRACTICE **POINTS**

- Crusted scabies often is misdiagnosed because it mimics common dermatologic conditions, such as atopic dermatitis, psoriasis, drug eruption, and seborrhea. A unique feature of crusted scabies is fine or coarse scaling that resembles brown sugar.
- Immunosuppressants, such as topical corticosteroids, worsen the skin's immune response to classic scabies infestations, which leads to parasitic overgrowth and the development of crusted scabies.
- Treatment of crusted scabies requires topical and oral scabicide; in addition, all clothing, bedding, and towels should be machine-washed and dried with hot water and hot dryer cycles to prevent environmental transmission and reinfection.

To the Editor:

Crusted scabies (formerly known as Norwegian scabies) is a rare and highly contagious variant of scabies, in which the skin is infested with thousands to millions of *Sarcoptes scabiei* var *hominis* mites. We present a case of skin changes that were misdiagnosed as atopic dermatitis, seborrhea, xerosis, and drug eruption on initial presentation, which prompted treatment with a corticosteroid that inadvertently caused progression to crusted scabies.

A 79-year-old woman who uses a wheelchair presented to the clinic with skin changes that consisted of diffuse, severely pruritic, erythematous plaques on the head, neck, trunk, face, and extremities of 2 years' duration. She had a medical history of hyperlipidemia, hypertension, and hyperglycemia, as well as a stroke that required hospitalization 2 years prior to the onset of the skin changes. She had no history of allergies.

Prior clinical diagnoses by primary care and dermatology included xerosis, atopic dermatitis, seborrhea, and drug eruption. She was treated with a mid-potency topical corticosteroid (triamcinolone acetonide cream 0.1%) twice daily and prednisone 40 mg once daily for 2- to 4-week courses over an 8-month period without reduction in symptoms.

Physical examination at the current presentation revealed golden, crusted, fine, powdery but slightly sticky flakes that spread diffusely across the entire body and came off in crumbles with a simple touch. These widespread crusts were easily visible on clothing. There was underlying diffuse erythema beneath the flaking skin on the trunk and proximal extremities. The scale and shedding skin laid in piles on the patient's lap and resembled brown sugar (Figure 1). The patient also reported decreased hand function and dexterity due to the yellow-brown, thick, crusty plaques that had developed on both the palmar and dorsal sides of the hands (Figure 2). Erythematous plaques on the scalp, forehead, and inner ears resembled seborrhea (Figure 3). Pruritus severity was rated by the patient as 10 of 10, and she scratched her skin the entire time she was in the clinic. The patient was emotional and stated that she had not been able to sleep due to the discomfort. We suspected scabies, and the patient

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The authors report no conflict of interest.

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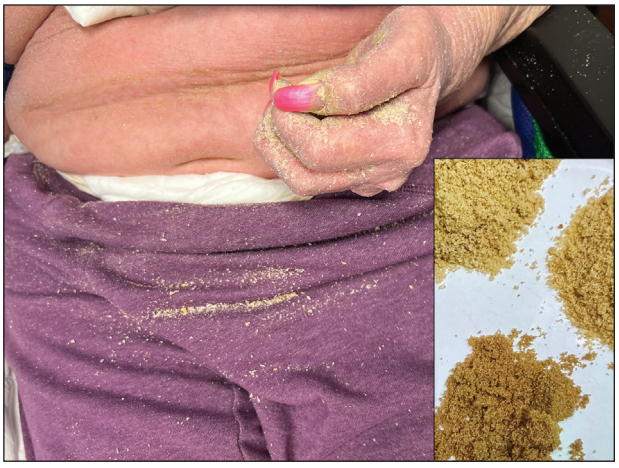

FIGURE 1. Scale resembling brown sugar (insert) piling on the patient's clothing, characteristic of crusted scabies.

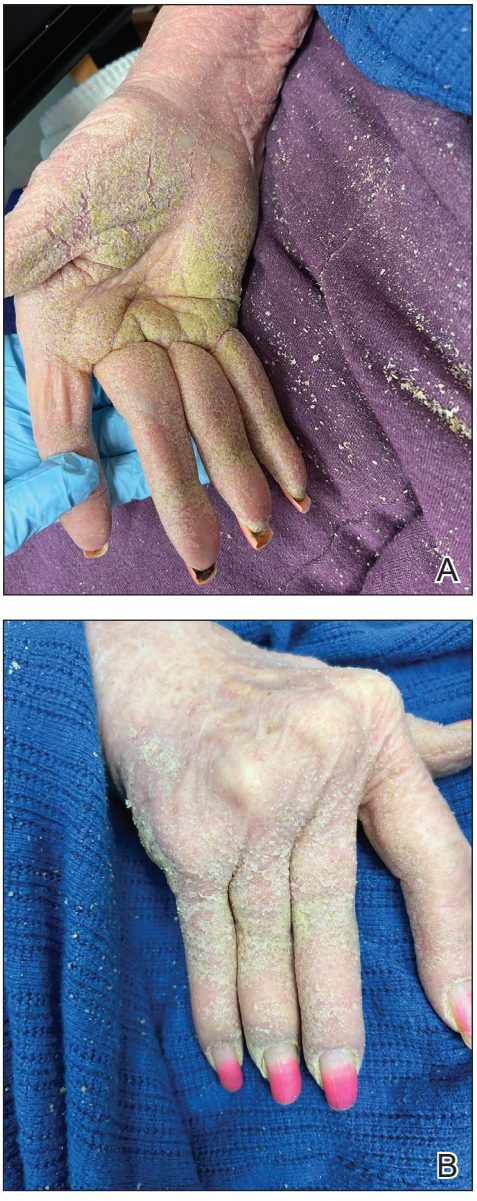

FIGURE 2. A and B, The skin on the patient's hands was hyperkeratotic and caused immobility due to the presence of thousands of scabies mites.

was reassured to learn that it could be confirmed with a simple skin scrape test.

The crusted lesions on the patient's hands were scraped with a #15-blade scalpel, and a routine potassium hydroxide mount was performed. The skin scrapings were placed on a slide with a drop of 10% potassium hydroxide and observed under low-power (×10) and high-power (×40) microscopy, which revealed thousands of mites and eggs (along with previously hatched eggs) (Figure 4) and quickly confirmed a diagnosis of crusted scabies—an extremely contagious form of scabies seen in older patients with compromised immune systems, malnutrition, or disabilities. The patient was prescribed oral ivermectin (3 mg dosed at 200 µg/kg of body weight) and topical permethrin 5%, neither of which she took, as she died of a COVID-19 infection complication 3 days after this diagnostic clinic visit.

Classic and crusted scabies are both caused by infestation of the *Sarcoptes scabiei* var *hominis* mite. Classic scabies is a result of an infestation of a small number of mites (commonly 5–15 mites), while crusted scabies is due to hyperinfestation by as many as millions of mites, the latter often requiring more aggressive treatment. The mites are first transmitted to humans by either skin-to-skin contact or fomites on bedding and clothing. The scabies mite undergoes 4 life cycle stages: egg, larvae, nymph, and adult. Once female mites are transmitted, they burrow under the skin and lay 2 to 3 eggs per day. The eggs hatch within 3 to 4 days, after which the larvae migrate to the skin surface. The larval stage lasts for 3 to 4 days, during which the larvae burrow into the stratum corneum to create molting pouches, until they molt into slightly larger nymphs. Nymphs can be found in hair follicles or molting pouches until they further molt within 3 to 4 days into adults, which are round, saclike mites. The adult male and female mites then mate, leaving the female fertile for the rest of her 1- to 2-month lifespan. Impregnated female mites traverse the skin surface in search of a burrow site, using the pulvilli on the anterior aspect of 2 legs to hold onto the skin. Once burrowed, the female mite continues to lay eggs for the rest of her life, with approximately 10% of her eggs resulting in adult mites. Male mites feed in shallow pits of the skin until they find a female burrow site for mating.[1] This continuous life cycle of the scabies mite gives rise to highly transmissible, pruritic skin excoriations, as demonstrated in our patient.

The skin has a relatively late inflammatory and adaptive immune response to scabies, typically occurring 4 to 6 weeks after the initial infestation.[2] This delayed inflammatory response and onset of

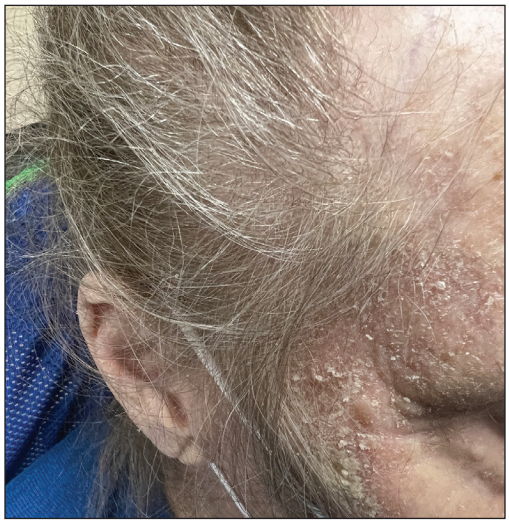

FIGURE 3. Hyperkeratotic scaly plaques on the patient's scalp, forehead, and inner ears that mimicked seborrheic dermatitis but were symptomatic of crusted scabies.

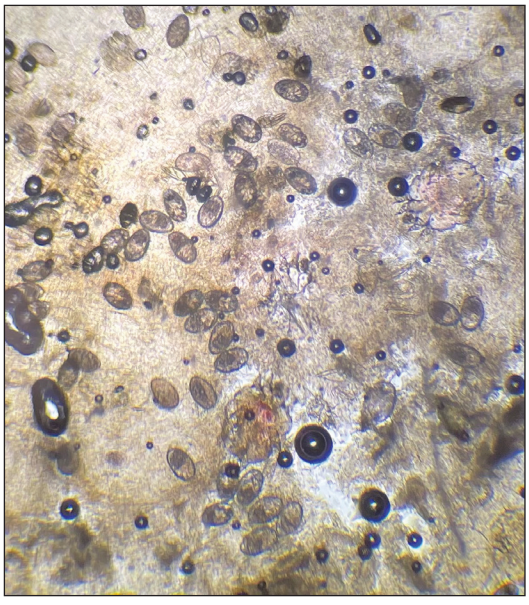

FIGURE 4. Microscopic findings from a skin scraping revealed scabies mites and eggs (original magnification ×10).

symptoms may be due to the scabies mite's ability to alter aspects of the host's immune response, which differs in classic vs crusted scabies. In classic scabies, there is a predominance of $CD4^+$ T cells in the dermis and minimal $CD8^+$ T cells. The opposite is true in crusted scabies—there is an overwhelming infiltration of $CD8^+$ T cells and minimal $CD4^+$ T cells.[3] The $CD8^+$ T-cell predominance in crusted scabies is hypothesized to be the cause of keratinocyte apoptosis, resulting in epidermal hyperproliferation. Keratinocyte apoptosis also secretes cytokines, which may lead to the immunologic targeting of healthy skin cells. The damage of healthy dermal cells contributes to the inability of the skin's immune system to mount an effective response, allowing the parasite to grow uncontrollably in patients with crusted scabies.[4]

This ineffective immune response is further exacerbated by corticosteroids, which are commonly prescribed for pruritus experienced by patients with scabies infestations. The mechanism of action of corticosteroids is the production of anti-inflammatory, antimitotic, and immunosuppressive effects.[5] Because the integumentary immune system is imbalanced during crusted scabies infestation, the immunosuppressive mechanism of oral and topical corticosteroids further reduces the cellular immune response to scabies. The flourishing of the scabies mites along with keratinocyte apoptosis[4] results in the development of hyperkeratotic skin crusting, most frequently on the palms, soles, arms, and legs. Risk factors for crusted scabies include immunosuppression, hospitalization, crowded living conditions, and poor hygiene, though no known risk factors were documented in up to 42% (33/78) of patients with crusted scabies in one study.[6]

Patients with crusted scabies typically present with generalized, poorly defined, erythematous, fissured plaques covered by scaling and crusts. Plaques on bony prominences such as finger articulations and elbows may have a thick verrucous aspect.[1] Skin flaking that resembles brown sugar—a mixture of white sugar and molasses—is a clue to the diagnosis of crusted scabies. Brown sugar has a slightly sandy and sticky texture that ranges in color from very light brown to very dark brown. When present, flakes always appears slightly lighter than the patient's skin tone. Although skin burrows are pathognomonic and clinically recognizable features of scabies, these burrows can be disguised by lesions, such as the hyperkeratotic plaques seen in our patient. The lesions may or may not be associated with pruritus, which may occur only at night, and bacterial superinfection has been reported in severe cases of crusted scabies,[7] as scratching can cause sores, which may lead to infection. In severe cases, the constant scratching could lead to sepsis if the infection enters the bloodstream.[8] Another symptom of scabies is a rash that causes small bumps that tend to form in a line, resembling small bites, hives, or pimples, and scaly plaques can lead to misdiagnosis as atopic dermatitis.

Treatment often is delayed due to misdiagnosis, as seen in our patient. Common misdiagnoses include atopic dermatitis, pityriasis rosea, systemic lupus erythematosus, bullous pemphigoid, lichen planus, pediculosis corporis, seborrheic scalp dermatitis, and adverse drug reactions.[9] Patients with extensive infestations of crusted scabies should be treated with a 4-week course of permethrin cream 5% daily for 1 week, then twice per week until resolved, and oral ivermectin 200 µg/kg dosed 1 week apart for up to 4 weeks, if needed.[1] Topical permethrin works by producing a selective neurotoxic effect on invertebrates such as scabies

mites, which disrupts the function of voltage-gated sodium channels, thereby paralyzing the adult mites to halt the spread of infestation. However, treatment with topical medications can be difficult due to the thick crusts that have formed, which make it more challenging for the skin to properly absorb the treatment. Additionally, surgical debridement as an adjunct procedure has been done to improve the effectiveness of topical medications by removing all the mites in skin.[10] On the other hand, the mechanism in which ivermectin treats scabies infestations is poorly understood. Current research suggests that ivermectin works by causing persistent opening of pH-gated chloride channels in scabies mites.[11] There is emerging concern for drug resistance to these scabicides,[12] revealing a need for further research of treatment options.

Patients with crusted scabies can have an extremely large number of mites (up to 2 million), making them more infectious than patients with classic scabies.[13] As a result, it is imperative to reduce environmental transmission and risk for reinfection with mites during treatment. Because crusted scabies is transmitted by prolonged skin-to-skin contact or by contact with personal items of an infected person (eg, bedding, clothing), treatment guidelines require all clothing, bedding, and towels of a patient with scabies to be machine-washed and dried with hot water and hot dryer cycles. If an item cannot be washed, it should be stored in a sealed plastic bag for 1 week, as scabies mites cannot survive more than 2 to 3 days away from their host of human skin.[13] Treatment of close contacts of patients with scabies is recommended, as well as for those in endemic areas or closed communities, such as nursing homes or jails.

REFERENCES

1. Salavastru CM, Chosidow O, Boffa MJ, et al. European guideline for the management of scabies. *J Eur Acad Dermatol Venereol*. 2017; 31:1248-1253. doi:10.1111/jdv.14351
2. Morgan MS, Arlian LG, Markey MP. *Sarcoptes scabiei* mites modulate gene expression in human skin equivalents. *PLoS One*. 2013;8:e71143. doi:10.1371/journal.pone.0071143
3. Walton SF, Beroukas D, Roberts-Thomson P, et al. New insights into disease pathogenesis in crusted (Norwegian) scabies: the skin immune response in crusted scabies. *Br J Dermatol*. 2008;158:1247-1255. doi:10.1111/j.1365-2133.2008.08541.x
4. Bhat SA, Mounsey KE, Liu X, et al. Host immune responses to the itch mite, *Sarcoptes scabiei*, in humans. *Parasit Vectors*. 2017;10:385. doi:10.1186/s13071-017-2320-4
5. Binić I, Janković A, Jovanović D, et al. Crusted (Norwegian) scabies following systemic and topical corticosteroid therapy. *J Korean Med Sci*. 2009;25:188-191. doi:10.3346/jkms.2010.25.1.188
6. Roberts LJ, Huffam SE, Walton SF, et al. Crusted scabies: clinical and immunological findings in seventy-eight patients and a review of the literature. *J Infect*. 2005;50:375-381. doi:10.1016/j.jinf.2004.08.033
7. Yari N, Malone CH, Rivas A. Misdiagnosed crusted scabies in an AIDS patient leads to hyperinfestation. *Cutis*. 2017;99:202-204.
8. American Academy of Dermatology Association. Scabies: signs and symptoms. Accessed July 12, 2024. https://www.aad.org/public/diseases/a-z/scabies-symptoms
9. Siegfried EC, Hebert AA. Diagnosis of atopic dermatitis: mimics, overlaps, and complications. *J Clin Med*. 2015;4:884-917. doi:10.3390/jcm4050884
10. Maghrabi MM, Lum S, Joba AT, et al. Norwegian crusted scabies: an unusual case presentation. *J Foot Ankle Surg*. 2014;53:62-66. doi:10.1053/j.jfas.2013.09.002
11. Currie BJ, McCarthy JS. Permethrin and ivermectin for scabies. *N Engl J Med*. 2010;362:717-725. doi:10.1056/NEJMct0910329
12. Andriantsoanirina V, Izri A, Botterel F, et al. Molecular survey of knock-down resistance to pyrethroids in human scabies mites. *Clin Microbiol Infect*. 2014;20:O139-O141. doi:10.1111/1469-0691.12334
13. Centers for Disease Control and Prevention. Preventing scabies. Published December 18, 2023. Accessed August 9, 2024. https://www.cdc.gov/scabies/prevention/index.html