THE CASE

An 85-year-old woman sought care at our outpatient clinic for a 9-month history of severe pruritus and crusted lesions on her face, extremities, and trunk. She had been diagnosed with hepatitis C virus (HCV) infection one year ago and was not taking any medication. The patient, who had been living with her family, had visited various clinics for her complaints and was diagnosed as having contact dermatitis and senile pruritus. She was prescribed topical mometasone furoate and moisturizers.

After 6 months of using this therapy, widespread grey-white plaques and minimal excoriation appeared on her face, scalp, and trunk. This was diagnosed as psoriasis, and the patient was prescribed topical corticosteroids, which she used for 9 months until she came to our clinic. She said the lesions regressed minimally with the topical corticosteroids, but did not fully clear.

Dermatologic examination revealed widespread erythema and grey-white, cohesive, thick, pruritic plaques on her scalp, face, trunk, and bilateral extremities (FIGURE 1). A punch biopsy specimen was taken from the border of a plaque on her trunk.

THE DIAGNOSIS

A complete blood cell count and wide biochemistry panel, including tumor markers and viral serology for human immunodeficiency virus (HIV), were normal. The patient had lymphadenopathy in her posterior cervical, bilateral preauricular, and bilateral inguinal regions.

Histopathologic examination revealed hyperkeratosis, acanthosis, and spongiotic edema in the epidermis, and vesiculation and mites in the stratum corneum. The dermal changes consisted of perivascular and diffuse cell infiltrates that were mainly mononuclear cells and eosinophilic granulocytes.

Based on the dermatologic examination and the histopathologic findings, we diagnosed the patient with crusted (Norwegian) scabies.

DISCUSSION

Crusted (Norwegian) scabies is a rare, highly contagious form of scabies that is characterized by the presence of millions of *Sarcoptes scabiei* var *hominis* mites in the epidermis.¹ This variant of scabies can affect individuals of any age, gender, or race.² It was first described by Boeck and Danielssen in 1848 in Norway and was named Norwegian scabies by von Hebra in 1862.³ In 2010, more than 200 cases of crusted scabies were reported in the literature.⁴

Crusted scabies is usually seen in immunocompromised patients, such as the elderly, those who’ve had solid organ transplantation, and those with HIV, malignancy, or malnutrition. Crusted scabies may also occur in patients with decreased sensory function (such as those with leprosy) or decreased ability to scratch, intellectual disabilities, and in those who use biologic agents or systemic/topical corticosteroids.⁴ ⁶ ⁸

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Crusted scabies is characterized by hyperkeratosis and wart-like crusts that are due to extreme proliferation of mites in the stratum corneum of the epidermis. Lesions are usually localized on acral sites (especially the hands), although the entire body, including the face and the scalp, can be involved. Psoriasiform or bullous pemphigoid-like eruptions have also been reported in the literature. Our patient presented with widespread erythema and psoriasiform grey-white crusts on her scalp, face, chest, periareolar region, and extremities. In addition, she did not have an immunosuppressant disease or medication history.

However, the fact that our patient was using topical corticosteroids for so long explained the extent of her condition. Topical corticosteroids have been linked to scabies incognito. Topical or systemic corticosteroid use for long periods of time may alter the skin immune system by suppressing cellular immunity, thereby reducing the inflammatory response. This may lead to progression of the regular variant of scabies to crusted scabies, as our patient had.

Topical treatments, oral ivermectin proven to be effective
Topical keratolytics, permethrin 5%, lindane 1%, crotamiton 10%, sulfur ointment (5%-10%), malathion 0.5%, benzyl benzoate (10%-25%), oral ivermectin (2 doses of 200 mcg/kg/dose), and systemic antihistamines are appropriate therapies. While oral ivermectin is effective, it is not available in Turkey.

Because of our patient’s hepatic disorder, we opted for a topical, rather than a systemic, treatment and recommended repeated applications of topical permethrin. Repeated treatment with topical permethrin is often sufficient in patients who are unable to take systemic therapy. In fact, Binic et al reported a case in which an elderly patient with crusted scabies (who had previously been treated with systemic and topical corticosteroids) responded well to repeated topical treatment with lindane 1%, 25% benzyl benzoate, and 10% precipitated sulfur.

Our patient. We prescribed topical 5%
permethrin lotion for our patient to apply to her entire body 4 times a week and advised her to wash her clothing and bed linens at 140° F. She was scheduled for biweekly check-ups. We also advised the patient’s family to use the same topical therapy 2 times per week because crusted scabies is highly contagious. One month later, our patient’s lesions had resolved (FIGURE 2).

THE TAKEAWAY
Early diagnosis and treatment of crusted scabies is important, both for the treatment of the patient and to stop the spread of the disease. Although rare, crusted scabies should be included in the differential diagnosis of long-term pruritic papulosquamous diseases, and the possibility of an atypical presentation in all patients should be considered—whether their immunity is compromised or not. Scabies should also be considered in patients with a positive family history of the disease and in those with chronic pruritus that is unresponsive to topical therapies.

References