Ivermectin for Crusted Norwegian Scabies Induced by Use of Topical Steroids

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An 11-year-old girl from a rural area was referred to our institution for a generalized dermatologic condition diagnosed as seborrheic dermatitis or psoriasis. The disease began 3 years earlier as pruritic lesions on her abdomen and buttocks. The patient had received multiple topical treatments for 3 years; the treatments had consisted of mainly fluorinated corticosteroids prescribed by general practitioners and pharmacy attendants. The intense pruritus caused by her lesions was mitigated transiently by the use of corticosteroids, but the lesions progressively worsened to the point that, at consultation, the patient walked with pain and difficulty and was not able to feed, dress, or clean herself. All family members with whom the patient lived had developed pruritic skin lesions in the previous few months.

On examination, the patient was in acceptable general condition but with widespread lesions. There were erythematous and desquamative plaques on her scalp, auricular regions, axillae, and umbilicus; hyperkeratotic and fissured plaques on her palms, soles, elbows, and sacral region; and hyperkeratotic nodules on the dorsa of her hands and feet (Figure 1). Her nails were markedly dystrophic with hyperkeratosis of the hyponychia. There was also a myriad of excoriated papules on her trunk and extremities. The patient experienced severe pain when walking and standing, as well as with flexion and extension of her hands.

A diagnosis of crusted Norwegian scabies (CS) was confirmed by scrapings from several skin sites that yielded a huge number of mites, eggs, and fecal pellets (Figure 2). Human immunodeficiency virus antibody test results were negative, and except for a slight peripheral eosinophilic leukocytosis (8%), routine laboratory values were within normal limits.

Initial treatment consisted of lindane lotion for the patient and her relatives. The symptoms of the family members rapidly resolved, but despite additional therapy with several regimens of topical permethrin and keratolytics, the patient’s lesions failed to achieve any significant improvement.

The patient’s lesions failed to improve with both conventional and aggressive topical scabicidal therapy. She continued to have intense pruritus, painful lesions, and disability. Most probably, marked hyperkeratosis of the patient’s lesions impaired the absorption of the topical agents and precluded an adequate contact with mites. A different therapeutic approach was required.

A single oral dose of ivermectin (Mectizan, MSD Holland, Paris, France), 6 mg, was given to the patient and treatment with topical keratolytics was continued. The pruritus subsided within 4 hours. The lesions started to clear 2 days later. A mild, asymptomatic, nonerythematous, centrofacial edema was noted 2 hours after ingestion of the medicine. The edema persisted for 10 to 12 hours and resolved spontaneously. A second 6-mg dose of ivermectin was given to the patient 3 weeks later when no skin lesions were apparent (Figure 3). No facial edema developed this time. For the past several months, the patient has remained free of symptoms and has resumed her normal life, including returning to school.

Crusted Norwegian scabies is an infrequent, massive form of infestation by Sarcoptes scabiei var hominis due to an inadequate host response to the mites. In common scabies, the number of parasites that infests the epidermis is relatively small. Such restriction is basically attributable to mechanical destruction by scratching, regular cleansing, and cell-mediated immune response. In CS, millions of parasites colonize the epidermis, inducing characteristic hyperplastic changes. It has been described in patients who do not scratch because of an absence of pruritus or immobility, such as occurs in pa-
tients with mental illness, sensory neuropathy, paresis, debilitating illness, or severe arthropathy. Crusted Norwegian scabies has also been described in patients whose immune defenses are impaired either as a result of disease or therapy as seen in patients with the acquired immunodeficiency syndrome or those who have undergone transplantation.1-3

In our case, the presumed immunosuppressive condition was the chronic use of topical fluorinated corticosteroids, mainly clobetasol propionate, with the patient using up to 60 g of cream per week. This rather rare situation was found in only 3 single case reports in the medical literature.4-6

Clinically, CS is characterized by extensive hyperkeratosis and crusting of the skin, especially on acral areas. Erythema and scaling are variable but may evolve into erythroderma. Pruritus is usually absent but can be moderate to severe. Diagnostic confusion of CS with other skin conditions, such as psoriasis, seborrheic dermatitis, contact dermatitis, and keratosis follicularis, is common.1,7,8 When the diagnosis of CS is delayed or no adequate isolation of the patient is provided, the highly contagious condition usually results in outbreaks of typical scabies among patients and health care personnel in hospitals and family members at home.9

Failure of the lesions, such as in our patient, to respond to recommended regimens of repeated applications of scabicidal agents in conjunction with keratolytics has prompted the use of ivermectin for CS.10 Ivermectin, a modified avermectin related to the macrolide antibiotics, has been widely used in veterinary practice against a variety of parasitic diseases caused by nematodes and arthropods. In humans, it is currently the drug of choice for onchocerciasis, but it has also been effective against several other endoparasitic and ectoparasitic infestations. In countries where onchocerciasis and other filarial diseases are endemic, millions of people have received safe and effective chemoprophylaxis and mass treatment with ivermectin.11 The use of ivermectin in human scabies is relatively new.12 It has been shown to be safe and effective in a single oral dose for the treatment of common and crusted scabies.13

The extensive, heavily crusted and hyperkeratotic lesions that produced such severe pain, pruritus, incapacity, and emotional distress in our patient rapidly and dramatically resolved following the initial dose of ivermectin. The fact that the patient’s pruritus disappeared a few hours after the oral treatment suggests that in our case the pruritus was not a hypersensitivity-induced circumstance, but probably a manifestation of a huge parasitic population of living and moving mites.

Adverse reactions with the large-scale use of ivermectin for filarial diseases, although infrequent, have been reported and include exacerbation of pruritus, edema, headache, and rash. Such reactions have been related to the sudden death of microorganisms and/or the release of their products. Therefore, they tend to occur especially in patients who have a high pretreatment load of parasites.14 Interestingly, the only adverse effect noted in our patient was facial edema with the first oral dose. No adverse effects were produced by the second dose that was given when no skin lesions remained.

Ivermectin has been used in doses ranging from 100 to 400 µg/kg. However, the common validated dose is 200 µg/kg.13 Ivermectin was approved by the US Food and Drug Administration on November 22, 1996, for the...
treatment of strongyloidiasis and onchocerciasis. It is marketed under the brand name Stromectol (Merck & Co Inc, Whitehouse Station, NJ). In children younger than 5 years, the use of ivermectin is not recommended. In the elderly, ivermectin has to be used with caution since a probable association between its use and an increased risk of death has been suggested.

Ivermectin should be the drug of choice in forms of scabies that are nonresponsive to conventional topical therapy, such as those seen in patients with the acquired immunodeficiency syndrome, or in large epidemic outbreaks.

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**REFERENCES**