CASE REPORT

Bullous Scabies Responding to Ivermectin Therapy

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Abstract
Bullous scabies is a rare disease that is usually diagnosed in elderly patients. The clinical, histological, and immunological findings are identical to those of bullous pemphigoid. In a review of the literature, we found reports of 24 cases. We present a new case of bullous scabies in a 72-year-old man. The lesions responded to treatment with oral ivermectin.

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Introduction
Scabies is the disease caused by infestation of the skin by the parasite Sarcoptes scabiei. While it can affect individuals of all ages, it is most common in children and in the elderly. The elderly are likely to present extensive and somewhat atypical lesions in the form of wheals, eczema-like plaques, or blisters. We review the literature on bullous scabies and describe the case of an elderly man whose disease responded to treatment with ivermectin.

Case Report
The patient was a 72-year-old man with a history of type 2 diabetes mellitus, hypertension, and chronic renal failure; on treatment with metformin, thiazides, and enalapril. He consulted for pruritic lesions that had appeared 4 months earlier and that did not respond well to therapy with prednisone (5 mg/d) and hydroxyzine.
Physical examination revealed scaly erythematous papules that coalesced to form generalized plaques and that had first appeared on the trunk (Figures 1 and 2). Pruritus was so severe that it interfered with activities of daily living and with sleep. A diagnosis of scabies was established and treatment was started with topical 5% permethrin. The pruritus did not improve, however, and 7 days later, tense blisters filled with clear fluid and measuring 1 to 5 cm began to appear on erythematous skin on the genitals (Figure 3) and in the inguinal folds. Nikolsky sign was negative and the scaly erythematous plaques took on the appearance of urticaria. There was no involvement of the palms, soles, mucosas, or face. A blood test showed normocytic anemia; 14 570 leukocytes/mL (82% polymorphonuclear cells, 11% lymphocytes, and 7% eosinophils); creatinine 2.5 mg/mL; normal protein electrophoresis; and normal antitransglutaminase antibodies. The chest radiograph was also normal. One of the blisters was examined using conventional histology and direct immunofluorescence. A perivascular infiltrate with eosinophils in the papillary
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Can be indistinguishable, especially when skin scraping shows neither the parasite nor mite feces. Bean described the first case in 1974 and 24 cases have been described in the literature since then. It has been suggested that the mechanism of blister formation in this condition might involve the persistence of *S. scabiei* in the skin for an extended period of time. This may trigger a specific immune response that activates T helper 2 cells, thereby elevating levels of interleukin 5 and, consecutively, of eosinophils with the secretion of proteolytic enzymes near the basement membrane, leading finally to the formation of blisters. There has been some debate as to whether these blisters are features of true scabies or rather of bullous pemphigoid triggered by the parasite. In order to distinguish between the 2 entities, Nakamura et al reviewed the cases of bullous scabies reported in the literature and compared them with those of patients who had been diagnosed with both bullous pemphigoid and scabies. They found positive immunofluorescence with granular or linear deposits of IgG or C3 in 73% of cases of bullous scabies. Indirect immunofluorescence, however, was negative in most cases, with only 1 case positive for IgG, albeit with low values. In patients with both bullous pemphigoid and scabies, on the other hand, linear deposits of IgG and C3 were found. Indirect immunofluorescence was also positive, with high IgG values. In conclusion, indirect immunofluorescence...
that is either negative or that shows low values supports a diagnosis of bullous scabies. The Table summarizes the cases of bullous scabies described in the literature.

Good responses (with resolution of the blisters) have been reported to topical therapy with 5% permethrin administered on days 1, 8, and 15, 1% lindane, 1% gamma benzene hexachloride, and 1% malathion. In cases where palmar-plantar hyperkeratosis is present, a keratolytic agent such as urea or salicylic acid should be added so that the active ingredient penetrates the skin. Of the cases reviewed, only 2 were treated with oral ivermectin (12 mg in 2 doses administered 10 days apart), with no improvement in pruritus until a month and a half after therapy. In our patient, there was complete remission of both the pruritus and the blisters 15 days after treatment with ivermectin at a dose of 200 µg/kg in 2 doses (on days 0 and 10); at 6 months’ follow-up, the patient remained asymptomatic and no new blisters had appeared. While the authors of a recent evidence-based study concluded that 5% permethrin is more effective than oral ivermectin for the treatment of scabies, they made no specific reference to bullous scabies. In our case, however, we reached the opposite conclusion, as the patient did respond to oral ivermectin. A possible explanation for this difference might be that, due to his age, our patient was unable to apply the permethrin correctly.

We report a new case of bullous scabies in an elderly patient with positive direct and negative indirect immunofluorescence that showed a good therapeutic response to oral ivermectin.

**Conflict of Interest**

The authors declare no conflicts of interest.

**References**