

etanercept and infliximab than in those taking methotrexate alone.² The incidence of large cell transformation of mycosis fungoides has also been found to be significantly higher (14.3% vs 1.8%) in patients taking methotrexate.³ Cyclosporine has been reported to induce transformation of mycosis fungoides in a patient with a psoriasiform dermatitis and atypical lymphocytic infiltrate, similar to our case.⁴

Alefacept was the first biologic agent approved for use in the treatment of psoriasis. Phase III trials showed little toxicity.⁵ Of possible significance, 2 out of 1357 patients who had been treated with various systemic agents before receiving alefacept developed Hodgkin's lymphoma and a folliculocentric lymphoma during or after alefacept therapy.⁵

It is possible, in this patient with an unclassified atypical infiltrate of the skin, that the alefacept-induced lymphopenia triggered an aggressive transformation in the presence of reduced tumor immunosurveillance. The timing of the alefacept injections suggests a causal role in the transformation to tumor stage mycosis fungoides, but this is open to debate. While the association between biologics and transformation of mycosis fungoides has not been previously reported, until further studies have been done, alefacept should be used with caution in patients with known mycosis fungoides or an unclassified atypical lymphocytic infiltrate of the skin.

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Buckwheat allergy and atopic dermatitis

To the Editor: Buckwheat (*Fagopyrum esculentum*), which is gluten-free and taxonomically unrelated to wheat, is a common alternative offered to patients with celiac disease (gluten sensitive enteropathy). Allergy to buckwheat induces an IgE-mediated immediate-type I hypersensitivity reaction, which has been found after ingestion of buckwheat, occupational exposure to buckwheat, or domestic exposure after sleeping on a pillow stuffed with buckwheat husk.¹ While the more common allergic reactions (anaphylaxis, asthma, allergic rhinitis, and urticaria) to buckwheat have been reported in the literature,²⁻³ the isolated occurrence of atopic dermatitis has not.

A 64-year-old African American male presented to the dermatology clinic with a complaint of a pruritic rash. Past medical history was significant for non-insulin dependent diabetes mellitus, hypertension, and hyperlipidemia. The patient had started eating cream of buckwheat cereal for breakfast daily. After 5 weeks on this regimen, he noticed a rash on his arms, wrists, legs, and back. The patient denied any medication changes. His physical examination revealed discrete erythematous thin scaly plaques on the upper back, chest, and abdomen, along with hyperpigmented thin plaques on the arms and legs bilaterally. Laboratory evaluation revealed negative IgA and IgG antibodies to gliadin. The patient was advised to discontinue eating the buckwheat, and the rash subsequently improved. Evaluation of the patient 3 months later showed that the dermatitis had resolved with evidence of only minimal residual postinflammatory hyperpigmentation.

Immunologic studies on buckwheat have shown the allergens to be thermo-stable, remaining even after cooking. A pepsin-sensitive 24-kDa protein unit has been identified as the most frequently recognized allergenic component in buckwheat, binding to IgE antibodies in the serum of almost all patients with proven buckwheat allergy. A 16-kDa buckwheat protein is also thought to be responsible for the immediate hypersensitivity reaction in patients with buckwheat allergy.⁴ These protein units may be a very useful tool in the diagnosis of buckwheat allergy in the future.

This case emphasizes the fact that buckwheat allergy should be added to the differential diagnosis of puzzling cases of atopic dermatitis. The development of allergy to buckwheat related to cross-sensitization with natural rubber latex has also been reported,⁵ and patients with latex allergy should be aware of the possible associated food hypersensitivity to buckwheat. More recently recognized for its antioxidant benefits and its natural whole grain properties, buckwheat has been cultivated on a larger scale for the growing market in the United States, and we should consider its allergenic properties more carefully in the evaluation of our patients.

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Treatment of steroid-resistant nodular scabies with topical pimecrolimus

To the Editor: Nodular lesions in male genitalia are a well-known clinical presentation of scabies, which are extremely pruritic and may persist for months after the specific treatment.¹

A 40-year-old white male was examined in our outpatient clinic. Five weeks before, he presented with typical lesions of scabies on his trunk and forearms followed by nodular lesions on his penis, scrotum, and thighs (Fig 1, A). Treatment with synthetic pyrethroids cleared the lesions, but the genital nodules persisted with disabling pruritus. Potent topical steroids did not reduce the nodules; neither did a course of systemic prednisone, which induced steroid acne on his back. Topical pimecrolimus was prescribed twice daily and improved his condition within a week (Fig 1, B).

Pimecrolimus and tacrolimus are calcineurin inhibitors and are approved for the treatment of atopic dermatitis, a condition mediated by T-lymphocytes, the target-cell of this new class of drugs.² Recent reports have shown their efficacy on other inflammatory skin diseases that could be treated with topical steroids, such as psoriasis, vitiligo, seborrheic dermatitis, morphea, lichen sclerosus, subacute lupus, circinate erosive balanitis, and lichen striatus. Nodular scabies is an exuberant variant of this infestation, with histologic features of pseudolymphoma, in which T-lymphocytes were shown to be



Fig 1. A, Nodular scabies. B, Marked improvement after topical pimecrolimus therapy.