

Efavirenz is a benzoxazinone and an orally active non-nucleoside reverse transcriptase inhibitor. The combination of efavirenz plus 2 nucleoside reverse transcriptase inhibitors has been added to the list of preferred regimens in the treatment guidelines. It is especially attractive as a way to simplify complex regimens with protease inhibitor. Efavirenz is metabolized by the liver, sharing the same metabolic pathway of all protease inhibitors. However, no clinically significant drug interaction in combination with zidovudine and lamivudine has been evidenced.³ In clinical trials 50% of the patients experienced adverse effects. The incidence of dermatologic side effects reached 15%, mainly mild and transient maculopapular exanthems.

Only 2 reports of photodermatitis induced by EFV have been published until now, one diagnosed on clinical findings and the other by photopatch testing.^{4,5}

Interestingly, cutaneous lesions in all these cases appeared from 4 to 8 weeks after patients had been put on a regimen of lamivudine, zidovudine, and efavirenz, suggesting that this combination could facilitate the induction of the photoreaction. However, the metabolite/pharmacokinetic mechanisms of this reaction in such patients remain unclear.

Evidence for drug-induced photosensitivity is sometimes difficult to establish and tends to emerge only after large-scale use of a drug.

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Atopic dermatitis-like eruption precipitated by infliximab

To the Editor: Infliximab, an anti-tumor necrosis factor- α therapy, is effective against rheumatoid arthritis and psoriasis. The mechanism of action involves alteration of the cytokine pattern with suppression of type 1 T-cell response.¹ Treatment with infliximab has been associated with precipitation of lupus erythematosus, as has been seen with etanercept, another anti-tumor necrosis factor- α agent.² Atopic dermatitis, like systemic lupus erythematosus, shows a pattern of type 2 T-cell pattern. Two cases are reported in which successful treatment with infliximab for rheumatoid arthritis precipitated an atopic dermatitis-like eruption.

In case 1, an 83-year-old man was referred by his rheumatologist because of a skin eruption that developed after his first infliximab infusion. He had a history of psoriasis, although the current eruption seemed different to him. He also gave a history of asthma. Treatment with topical clobetasol ointment 0.1% for involvement on the trunk and 2.5% hydrocortisone for facial involvement was prescribed. He returned after a subsequent infliximab infusion indicating that the eruption improved with the topical treatment but flared after the subsequent infliximab infusion. The eruption involved the face, arms, and trunk with antecubital accentuation. There was moist dermatitis with no scale. A skin biopsy specimen showed a perivascular round cell inflammatory infiltrate with no evidence of psoriasis. Topical treatment was changed to triamcinolone ointment 0.1% with tacrolimus ointment 0.1% on alternate days. He has continued the infliximab infusions with dramatic improvement for his rheumatoid arthritis and his psoriasis. His dermatitis has continued to flare with each infliximab infusion, but responds to topical treatment.

In case 2, a 68-year-old woman was seen on referral from her rheumatologist for a recurrent eruption that developed after infliximab infusions for rheumatoid arthritis. Her rheumatoid arthritis was responding dramatically to the infliximab. She denied any previous skin problems, although she had an uncle with a severe lifelong pruritic skin problem. She presented with dermatitis having a patchy distribution with accentuation in the antecubital fossae. Treatment with 2.5% hydrocortisone had been unhelpful. Fluocinonide ointment 0.05% was prescribed for involvement on the trunk. Treatment with tacrolimus ointment 0.03% was subsequently added. The dermatitis continues to flare after each infliximab infusion, but it does respond to topical therapy. The rheumatoid arthritis is responding well to the infliximab therapy.

Both patients were referred for atopic dermatitis–like eruptions associated with infliximab infusions. In both cases the skin problem was nonallergic in nature and permitted continuing the successful infliximab infusions. Both the referring physicians and the patients were relieved to be able to continue the therapy. This cytokine-altered eruption can be added to the list of nonallergic reactions to medications.

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An article published after submission of this Case Note describes 4 cases that the authors considered to be immunologically mediated (Vergara G, Silvestre JF, Betloch I, Vela P, Albares MP, Pascual JC. Cutaneous drug eruption to infliximab: report of 4 cases with an interface dermatitis pattern. *Arch Dermatol* 2002;138:1258-9).

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NOTES & COMMENTS

Detergent allergies: An urban legend

To the Editor: I read with interest the recent report by Belsito et al,¹ which found that detergents are a rare cause of allergic contact dermatitis. Although this study is useful, the belief it debunks is not likely to wash away anytime soon.

Many patients who consult physicians about dermatitis have already gone through the exercise—often several times—of changing soaps, detergents, fabric softeners, makeup, and shampoos. Only when they find this search futile do they seek medical attention. People learn that detergents aren't the culprit, yet remain convinced they are. After all, everyone knows that soaps and detergents cause rashes.

The reason everyone knows this is that everyone—laypeople and professionals—keeps saying so. Beyond its status as received wisdom, the soap-detergent hypothesis has other attractions. First, it purports to explain the otherwise inscrutable ups and downs of dermatitis. Second, these very fluctuations make the hypothesis plausible: after a change in cleanser, the condition may get better for a while, “proving” that the old detergent was to blame—until the next outbreak, when the old “allergy” goes away, to be replaced by the next usual suspect, and so on.

Belsito et al¹ have devoted admirable energy to refute the belief that detergents cause rashes. Their sponsors, Procter and Gamble, have an interest in learning the value of the detergents they market as

“free and clear” of fragrances and additives, because the commercial viability of these products depends on the belief that rashes are caused by the stuff of which they're free and clear. Like an urban legend, however, the conviction that detergents cause rashes is impervious to empirical refutation. No matter how many New York City sewers are checked and found to be free of alligators, we all know those reptiles are in there somewhere.

Several years ago I called the laundry departments of several teaching hospitals in Boston, Massachusetts, to ask about their procedures when wards send down sheets with the message that a patient is allergic to the detergent in their bedclothes.

“Can I be frank?” asked one laundry director, in a conspiratorial tone. “We wash them again, pack them in a different-colored wrapper, and send them back up.”

“Then what happens?” I asked.

“Nothing,” he said.

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