

## Lymphomatoid papulosis while on efalizumab.

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### Published In/Presented At

Leo AM, Ermolovich T. Lymphomatoid papulosis while on efalizumab. *J Am Acad Dermatol*. 2009 Sep;61(3):540-1. doi: 10.1016/j.jaad.2008.12.010.

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diagnosis, and treatment of tumors in their early stages.

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Funding sources: None.

Conflicts of interest: None declared.

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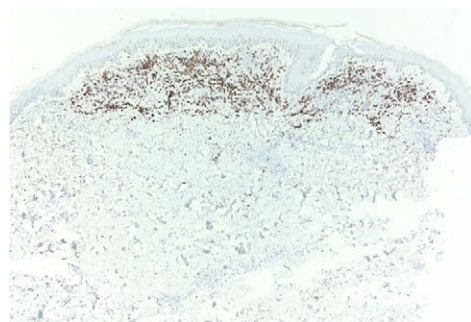
doi:10.1016/j.jaad.2008.12.008

#### Lymphomatoid papulosis while on efalizumab

*To the Editor:* We report a 60-year-old female with a 25-year history of psoriasis who developed lymphomatoid papulosis during treatment with efalizumab. Previous psoriasis treatments included etanercept for 2 years, narrowband ultraviolet B phototherapy for 6 months, and various topical medications. Thirteen years earlier, before treatment with biologic or light therapies, the patient developed small, crusted, red papules on her extremities that lasted 7 to 8 months and resolved without treatment. Clinically, these lesions were consistent with pityriasis lichenoides; a biopsy was suggestive of—but did not definitively confirm—the clinical impression. A CD30 stain was not performed on the biopsy and the block is no longer available for further staining. The patient remained



**Fig 1.** Red, ulcerated plaque of lymphomatoid papulosis in the left axilla.



**Fig 2.** CD30 stain highlights the large, pleiomorphic lymphocytes.

free of any pityriasis lichenoides-like eruptions for more than 10 years, including 2 years while on etanercept. However, after taking efalizumab for 8 months, red, crusted papules and plaques appeared on the patient's forearms and back. Biopsies of the lesions were consistent with CD30<sup>+</sup> lymphomatoid papulosis. Efalizumab was discontinued and computed tomographic scans of the chest, abdomen, and pelvis revealed no lymphadenopathy. The lesions resolved during a 6-week course of narrowband ultraviolet B phototherapy. However, despite the discontinuation of efalizumab, 4 months later, a red, ulcerated plaque formed in her left axilla (Fig 1). The biopsy was again consistent with CD30<sup>+</sup> lymphomatoid papulosis (Fig 2). The lesion resolved over 2 months, during which intralesional triamcinolone injections were administered. The patient has not developed any new lesions for the past 8 months.

Lymphomatoid papulosis has a cyclic and unpredictable course, and often resolves spontaneously. This rare condition falls within the spectrum of lymphoproliferative disorders, and 4% to 5% of patients progress to systemic lymphoma (Hodgkin lymphoma, CD30<sup>+</sup> anaplastic large cell lymphoma,

and mycosis fungoides). Several therapeutic options have been reported, including corticosteroids, methotrexate, bexarotene, ultraviolet radiation, excimer laser, interferon alfa-2a, and imiquimod cream. Some of these—methotrexate and ultraviolet radiation, for example—also serve as therapies for psoriasis.

Immunosuppression is known to potentiate lymphoproliferative disorders, and biologics, including efalizumab, have been associated with lymphoma.<sup>1,2</sup> It appears that lymphomatoid papulosis could develop while on biologic therapy. Flendrie et al<sup>3</sup> reported a case of a lymphomatoid papulosis–like eruption in a patient with rheumatoid arthritis on adalimumab. The patient developed CD30<sup>+</sup> lymphomatoid papulosis that resolved in 6 weeks without discontinuing the medication. Two years later, however, the patient developed large cell anaplastic non-Hodgkin lymphoma.<sup>3</sup> Other immunosuppressant agents implicated in drug-induced lymphomatoid papulosis include cyclosporine<sup>4</sup> and psoralen plus ultraviolet A light phototherapy.<sup>5</sup> These cases developed in patients with underlying immune dysfunction (eg, atopic dermatitis in the former and mycosis fungoides in the latter).

According to Guttman-Yassky et al,<sup>6</sup> efalizumab produces a unique state of T cell hyporesponsiveness.<sup>6</sup> Therefore, efalizumab may have unmasked the patient's underlying susceptibility to a lymphoproliferative disorder. It may have been a factor in the occurrence of lymphomatoid papulosis while she was on efalizumab and not while she was on etanercept. However, the capricious nature of lymphomatoid papulosis combined with the patient's history of psoriasis (an immune disorder) makes it difficult to confirm efalizumab as the sole cause of the patient's lymphomatoid papulosis. We hypothesize that the combination of the patient's underlying proclivity for a lymphoproliferative

disorder combined with the concomitant immunosuppression from efalizumab triggered the lymphomatoid papulosis. Therefore, when treating with efalizumab, clinicians should be aware that lymphomatoid papulosis may be triggered in susceptible individuals by a unique immunosuppressive mechanism that is specific to efalizumab.

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*Funding sources: None.*

*Conflicts of interest: None declared.*

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doi:10.1016/j.jaad.2008.12.010