Innovative therapies and new targets in psoriasis

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DERMATITIS DURING EFALIZUMAB TREATMENT IN A PATIENT WITH PSORIASIS VULGARIS

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Efalizumab is a fully humanized monoclonal antibody against CD11a, the \( \alpha \)-chain of the lymphocyte function associated (LFA)-1 adhesion molecule. By binding to CD11a, LFA-1 is prevented from binding with its ligand, intercellular adhesion molecule (ICAM-1). This inhibits various T-cell processes believed to be important in the pathogenesis of psoriasis, including T-cell activation, T-cell adhesion to endothelial cells and T-cell migration. Clinical trials demonstrate that efalizumab, given subcutaneously once weekly, provides clinical benefit in patients with moderate to severe plaque psoriasis \(^1\text{-}^6\). We report a case in which a patient with psoriasis vulgaris developed dermatitis during efalizumab therapy.

A 48-year-old male, diagnosed with psoriasis in 1991, received weekly subcutaneously injections efalizumab (0.7 mg/kg) for 12 weeks. Apart from budesonide and formoterol inhalation medication, the patient used no other medication.

During week 9 of the treatment the patient developed multiple moderately defined erythemato-squamous papules varying in size from 1 to 2 centimeters on the extremities and trunk, next to the classical psoriasis lesions. The majority of those lesions showed excoriations, as seen in Figure 1.

![Figure 1](image1.png)  
**Figure 1** Multiple moderately defined erythemat-o-squamous papules, covered with crusts next to excoriations, and classical psoriasis lesions on the trunk and extremities

![Figure 2](image2.png)  
**Figure 2** Detail of the right arm with multiple moderately defined erythematosquamous papules, covered with crusts next to excoriations
A skin biopsy taken from a papule of the wrist on week 10 showed reactive epidermal changes which were compatible with the diagnosis lichen simplex chronicus.

One month after the patient had his last injection of efalizumab, a second biopsy was taken from another lesion. This showed a widened acanthotic epidermis with parakeratosis and a chronic inflammation infiltrate in the dermis with local neutrophilic and eosinophilic granulocytes infiltrating the upper epithelium.

Simultaneously with the dermatitis the patient developed unbearable pruritus for which he was admitted to the dermatology ward. After treatment with UVB (311 nm) in combination with topical betamethason twice daily and daily baths with bath oil, the atypical lesions disappeared and the psoriasis improved.

Efalizumab is one of the new biological therapies targeting T-lymphocyte activity for the treatment of chronic plaque psoriasis. Common adverse events include headaches, nonspecific infection, nausea, chills and fever. A variety of unusual forms of psoriasis have been observed in patients receiving efalizumab, such as guttate psoriasis, psoriatic erythroderma and pustular psoriasis. However, all these manifestations occurred after withdrawal of efalizumab and the lesions did not resemble dermatitis. In contrast, our patient developed lesions of dermatitis during treatment with efalizumab.

Although drug eruptions due to efalizumab have not been reported yet, it cannot be ruled out that this was the case in our patient. Our patient developed dermatitis 9 weeks after entering the open label phase and this could not be attributed to change of medication. No rechallenge was performed.

Our patient was known to be atopic. His medical history showed asthma for several years as well as a positive family history for atopic disorders. Laboratory results showed a very high IgE of 18200 U ml⁻¹ (normal range < 100U ml⁻¹), particularly positive for grass, trees and house dust mite, as well as eosinophilia (1569 10E⁶ l⁻¹, normal range 11-330 10E⁶ l⁻¹), confirming atopy. Although the concomitant manifestation of atopic dermatitis and psoriasis is very rare, this could be the case in this patient.

Alternatively, since efalizumab influences the natural course of psoriasis, the lesions could be an atypical presentation of new developing psoriasis lesions during the treatment.
REFERENCE LIST


