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DOI: [10.1159/000117708](https://doi.org/10.1159/000117708)**Multiple Eruptive Dermatofibromas in a Patient Receiving Efalizumab**Jorge Santos-Juanes^a, Pablo Coto-Segura^a, Susana Mallo^a, Cristina Galache^b, Jorge Soto^c^aService of Dermatology II, Hospital Universitario Central de Asturias, Universidad de Oviedo, Oviedo, ^bService of Dermatology, Hospital de Cabueñes, Gijón, and ^cDepartament of Dermatology, Universidad del País Vasco, Bilbao, Spain**Key Words**

Multiple eruptive dermatofibromas • Efalizumab • Psoriasis

Multiple eruptive dermatofibromas (MED) are very rare and usually have been associated with autoimmune diseases, immunosuppressant therapy, hematologic malignancy and other conditions [1]. We present the case of a 44-year-old woman with psoriasis who developed MED while undergoing efalizumab treatment. To our knowledge this association is a novel finding that has not previously been described [2, 3].

Report of Case

A 44-year-old Caucasian woman with recalcitrant psoriasis was treated with efalizumab (1 mg/kg once a week) after several therapeutic failures with other agents. After 8 months she suddenly developed 7 erythematous, brownish papules on both extremities.

Physical examination revealed 7 firm, brownish, dome-shaped nontender papules ranging from 5 to 10 mm in diameter with lateral dimpled signs on the thighs (3 on the right and 4 on the left). Excision biopsy of 1 papule on the right thigh showed a poorly demarcated dermal nodule composed of tightly interdigitating fascicles of spindle-shaped fibrohistiocytic cells. In the peripheral part of the dermal tumor, the dermal collagen was entrapped by the fibrohistiocytic cells. Special stains for infectious agents were negative. The immunohistochemical study revealed that many of the lesion cells were positive for factor XIIIa.

Laboratory findings were either normal or negative for the following: complete blood cell count; metabolic and hepatitis panels; erythrocyte sedimentation rate; anti-HIV antibody, and levels of antinuclear antibody, double-stranded DNA antibody, immunoglobulins and thyroid-stimulating hormone.

Treatment was suspended and at 6 months no new lesions were found.

Comment

Although dermatofibromas (DFs) are very common, their pathogenesis is poorly understood. It has recently been proposed that this should be regarded as an immunoreactive process, mediated by dermal antigen-presenting cells that trigger an abortive immune response to as yet unidentified antigenic stimuli. According to this hypothesis, the development of MED in immunodeficient states could be facilitated by the inhibition of downregulatory T cells; alternatively, multiple DFs could develop as an exaggerated response to a putative pathogen that could not be cleared by the suppressed immune system [4–6].

Efalizumab (anti-CD11a) is a humanized monoclonal antibody, which blocks multiple T-cell-dependent functions involved in the pathogenesis of psoriasis, including T cell activation and migration to the skin provoking a partial immunosuppressed state [7].

We believe that this combination is not just coincidental and that the development of DF can be attributed to the medication. In a review of the literature we found only 1 patient with psoriasis receiving prednisolone and ultraviolet B phototherapy who developed MED, but the authors attributed the eruption of DF to the HIV infection which the patient suffered [8]. Moreover, the development of new DFs stopped after immunosuppressive therapy had been discontinued in our patient.

As dermatologists prescribing new biological drug therapies we should be alert to the appearance of new side effects not found in clinical trials.

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