CORRESPONDENCE

Development of lichen striatus in a psoriasis patient treated with adalimumab

Dear Editor,

A 37-year-old man was diagnosed with psoriasis and psoriatic arthritis in February 2006. Previous treatment with sulfasalazine, leflunomide, nonsteroidal anti-inflammatory drugs, etanercept, and topical steroids showed limited effects. Adalimumab at 40 mg every 2 weeks was given since April 2013. His skin condition and arthritis improved during the treatment, with the Psoriasis Area and Severity Index decreasing from 2.2 to 0.3 and tender joint counts from 5 to 0. However, asymptomatic whitish linear patches and pinpoint lichenoid papules were noted over the right arm (Figure 1A) and shoulder area (Figure 1B) 3 months later. According to the patient, mild erythema preceded the appearance of the eruptions, and he denied viral infection or local trauma history on the right upper extremity in recent 3 months. Laboratory data were all within normal limits and he was otherwise healthy, without atopy history. A skin biopsy showed psoriasiform hyperplasia with basal vacuolization and pigment incontinence. Superficial perivascular and perieccrine lymphocytic infiltration was noted as well (Figure 1A and D). Lichen striatus was diagnosed. Adalimumab was discontinued since December 2013, owing to the improvement of psoriasis and psoriatic arthritis. We followed this patient regularly, and the linear skin lesions improved spontaneously. Figure 1E shows the significant improvement 6 months after the patient stopped taking adalimumab.

Cutaneous eruptions have been increasingly reported after the use of tumor necrosis factor (TNF) blockers, such as acenfine eruption, alopecia areata, urticaria, and palisaded neutrophilic and granulomatous dermatitis.1,2 Lichen striatus is an enigmatic self-limiting inflammatory dermatosis with lesions following the lines of Blaschko, particularly common in children. The etiology of the eruption is unknown, but environmental factors, viral infection, cutaneous injury, hypersensitivity, and genetic predisposition have been implicated. The differential diagnosis includes epidermal nevi, linear psoriasis, linear morphea, linear lichen planus, and linear cutaneous lupus erythematosus. Lichen striatus typically presents as a nonpruritic continuous or interrupted linear band of small (1–3 mm) pink or hypopigmented papules, with a smooth surface or mild scaling. Histopathologic findings depend on the stage of evolution, with variable epidermal changes and lichenoid inflammation. Perivascular and deep lymphocytic infiltrate surrounding the hair follicles and eccrine glands are characteristic.

Lichen striatus can be associated with psoriasis or eczema. In a small case series of 18 patients with lichen striatus, Taieb et al1 described two patients with psoriasis and six patients with atopic diathesis. Another three cases of psoriasis coexisting with lichen striatus were also found in our literature search. In the first case, unilateral lichen striatus preceded unilateral eruptive psoriasis on the contralateral side, and a common trigger was suspected to be the cause. In the second case report, lichen striatus developed in a patient with chronic plaque psoriasis after ultraviolet phototherapy. However, the authors thought that phototherapy did not cause lichen striatus, because lichen striatus resolved despite continuous phototherapy.2 In the third case, lichen striatus occurred after adalimumab treatment in a patient with longstanding psoriasis.5 The extent of the eruption was more extensive than in our case, but both occurred 3 months after adalimumab treatment.

The mechanism of this unique phenomenon is unknown. It could be Th1/Th2 imbalance, cytokine imbalance between TNF-alpha and interferon-alpha, or loss of immune tolerance induced by an acquired stimulus, after TNF blockers.7,8 Lichen striatus associated with etanercept therapy in rheumatoid arthritis was reported recently. In our case, however, lichen striatus did not happen during the course of etanercept treatment. Although both are TNF blockers, etanercept and adalimumab still differ in terms of the mode of action.9,10 We speculate that the difference in the degree of immunosuppression may be one reason. Adalimumab is considered more immunosuppressive because clinical improvement of psoriasis is higher after adalimumab compared to etanercept. In addition, infection rate (in particular, tuberculosis) is also higher for adalimumab compared to etanercept. In our previous report, maximal improvement of psoriasis occurred 3 months after adalimumab treatment, coincidental with the time of onset of lichen striatus in our case.11

In summary, we report a patient with psoriasis who developed lichen striatus after adalimumab treatment. Although coincidence cannot be ruled out, we propose that lichen striatus should be

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considered in the differential diagnosis of new eruptions in patients receiving TNF blockers.

I-Chun Lin, Ting-Shun Wang
Department of Dermatology, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan

Hsien-Yi Chiu
Department of Dermatology, National Taiwan University Hospital Hsin-Chu Branch, Hsinchu, Taiwan

Tsen-Fang Tsai*
Department of Dermatology, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan

*Corresponding author. Department of Dermatology, National Taiwan University Hospital, Number 7 Chung Shan South Road, Taipei, Taiwan. E-mail address: tftsai@yahoo.com (T.-F. Tsai).

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Figure 1 (A) Whitish linear patches over the right arm, following the lines of Blaschko. (B) Hypopigmented pinpoint lichenoid papules on the right shoulder. (C) Superficial perivascular and perieccrine lymphocytic infiltration (H&E). (D) Higher power field showed perieccrine lymphocytic infiltration (H&E). (E) Follow-up picture 6 months after discontinuing adalimumab treatment. H&E = hematoxylin and eosin.