

could be involved in patients with concomitant AA and AD. Based on the cases reported to date, there appears to be a potential risk of developing AA with dupilumab rather than a remission of pre-existing AA.

We report the shortest time to onset of AA after dupilumab introduction. This is an unusual case of AA after a single injection of dupilumab. Clinicians should be aware of the risk of AA in patients treated with dupilumab. Scalp biopsies should be performed to better understand the pathophysiology of AA. ■

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TNF α inhibitor-induced urticaria: a class effect

The introduction of tumour necrosis factor alpha (TNF α) inhibitors and more recent biologics such as ustekinumab or anti-IL17/IL17R blockers have revolutionized the management of moderate-to-severe psoriasis and complete skin clearance can now be attained. However, the use of biologics such as anti-TNF α are associated with several adverse skin events including psoriasiform or lichenoid eruptions and lupus-like syndrome. We report the case of TNF α inhibitor-induced urticaria with adalimumab and infliximab, revealing a class effect reaction.

We describe the case of a 27-year-old woman who suffered from severe psoriasis since childhood with skin lesions affecting her entire body including her scalp and skin folds (*figure 1A*). Many topical and/or systemic therapies including topical steroids, PUVA therapy, methotrexate, and etanercept were unsuccessful in treating her condition (*figure 1C*). Because of the severity of her psoriasis (PASI:16 and DLQI:18), adalimumab treatment was introduced and led to temporary clinical remission. However, 18 months later, the disease flared up, warranting the discontinuation of the adalimumab and a switch to ustekinumab. Due to multiple side effects (weight gain, arthralgia, and depressive state), the patient interrupted the ustekinumab treatment after six months. Three years later, the patient returned to the dermatology clinic and presented with severe psoriasis (PASI:11 and DLQI:23). Because anti-IL-17 blockers were not yet available, adalimumab was proposed with increased injection frequencies (40 mg per week). Again, the psoriasis responded very well to the therapy but eight months later, she developed a urticarial rash with dermographism, associated chronic pruritus and insomnia. The urticarial rash did not start at the sites of injection nor did it coincide with the adalimumab injections. The papules developed on the trunk and limbs, were evanescent during the day, and were partially controlled by antihistamines. Adalimumab was stopped and secukinumab was initiated. The patient immediately noted the disappearance of the urticarial lesions, but anti-IL-17 blocker did not improve the psoriasis lesions. After six months, secukinumab was stopped and it was decided to retarget the TNF α pathway by treating with infliximab (5 mg/kg every six weeks). Despite the efficacy of infliximab as treatment for the psoriasis, a recurrence of the urticarial skin lesions associated with major pruritus was observed (*figure 1B*) two weeks after the second infliximab administration. Antihistamines and methotrexate were added to control the urticarial reaction. The patient presented no systemic symptoms. Blood analyses, eosinophil count (0.22G/L), and tryptase level (2.4 μ g/L) were normal.

Anti-TNF α -induced urticaria can be triggered by different mechanisms [1]. Firstly, an immediate IgE hypersensitivity-mediated reaction may be implicated, as has already been described in previous publications. However, urticaria should then occur within two hours [2, 3]. Delayed hypersensitivity has also been described in anti-TNF α -induced urticaria [4, 5]. However, neither of these two mechanisms were retained because the chronology of urticarial lesions did not coincide with treatment administrations and because the skin tests, performed two months after the beginning

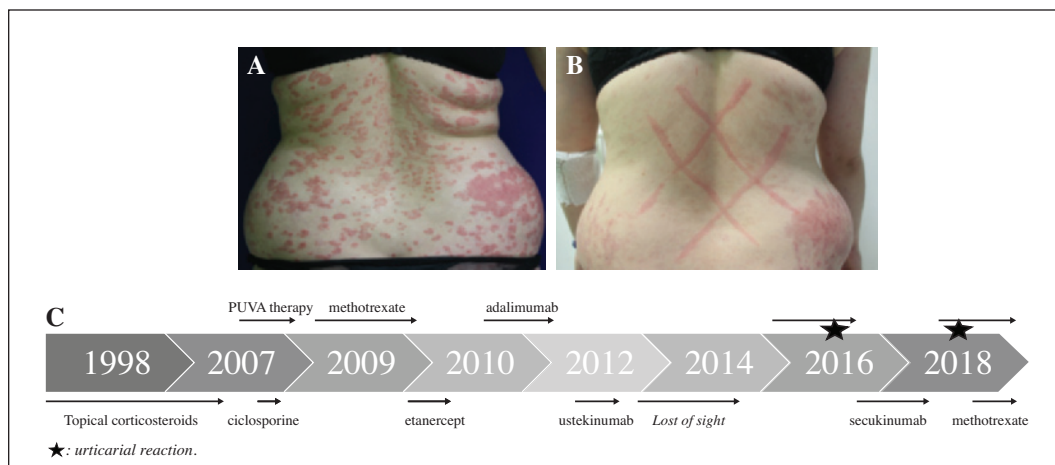


Figure 1. A) Psoriasis lesions before infliximab treatment. B) Dermographism one month after the beginning of infliximab. C) Treatment history of the patient and chronology of urticarial reaction.

of urticarial reactions, with infliximab were negative on immediate and two-day readings (prick tests and intradermoreaction [IDR]: 0.02 mg/mL, 0.2 mg/mL and 2 mg/mL). Another hypothesis could be an immunological deviation towards a Th1 immune response, such as the one observed in lupus-related disorders associated with anti-TNF α and urticarial vasculitis [6]. However, in contrast to our case, urticarial vasculitis lesions are more likely to be painful, non-blanching, not pruritic, and remain in the same location. Moreover, no increased antinuclear antibodies were observed before and after initiation of infliximab therapy. Finally, anti-TNF α -induced urticaria could be explained by a Th2 deviation, inducing the appearance of chronic spontaneous urticaria. Indeed, the decrease in TNF α can also lead to significant immune deviation due to the reduction of the expression of several Th1-Th17 cytokines and an increase of several Th2 cytokines which are under-expressed at baseline (interleukin [IL]-2, IL-4, IL-5) [7]. Urticaria is a skin disorder involving innate immune cells such as monocytes, eosinophils, basophils or neutrophils but is also characterized by an increase in IL-4, IL-5 and IFN- γ (mixed Th1/Th2 response) [8]. We retain this last hypothesis based on the modulation of the immune environment by anti-TNF α to explain the anti-TNF α -induced urticaria in our patient. To conclude, to our knowledge, this is the first case of TNF α inhibitor-induced urticaria with a relevant class effect. ■

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A toddler case of keratosis follicularis squamosa (Dohi) successfully treated with salicylic acid ointment

Keratosis follicularis squamosa (KFS), first described by Dohi in 1903, is a disorder of follicular keratosis [1]. Some treatments have been tried, but no standard treatment is currently available. Here, we report a case of toddler KFS successfully treated with salicylic acid (SA) ointment. A three-year-old Japanese girl presented with brownish scaly patches on her abdomen and posterior thighs. The eruptions had first appeared when she was six months old and had gradually increased in number. She had been treated with topical corticosteroid ointment and heparinoid ointment, but without effect. The family and medical histories were both unremarkable. Physical examination