



AUTOIMMUNE BULLOUS DISEASES

TRANSITION FROM PEMPHIGUS VULGARIS TO PEMPHIGUS FOLIACEUS: THE POSSIBLE ROLE OF ANTI-TNF- α AND VARICELLA ZOSTER VIRUS.

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Background: Pemphigus is a rare autoimmune blistering disease. Pemphigus vulgaris (PV) and pemphigus foliaceus (PF) are the most frequent subtypes of autoimmune pemphigus. The transition of PV into PF has been rarely described in the literature. Qualitative changes in the immunological profile of anti-Dsg auto-antibodies might lead to the transition. However, the underlying process is not completely known.

Observation: A 60-year-old Caucasian male patient affected by recalcitrant PV developed a toxic epidermal necrolysis (TEN) after the administration of rituximab. TEN was efficiently treated with etanercept 50 mg, leading to a complete clinical remission. However, after 2 months the patient developed a diffuse varicella zoster virus (VZV) herpetic eruption, and the patient was admitted again to our Department. He was successfully treated with anti-retroviral therapy. After discharging, the patient has been more strictly followed-up. 5 months after the resolution of the herpetic eruption and 7 months after the resolution of TEN, the patient was admitted again in our Department showing superficial erosions, crusts and scales on an erythematous background, involving upper chest and back. He showed no oral or mucosal involvement. A biopsy of the chest skin was performed, showing a sub-corneal cleft. Only anti-Dsg1 auto-antibodies were detected, whereas when the diagnosis of PV was made both anti-Dsg1 and anti-Dsg3 auto-antibodies were present.

Key message: The switch from PV to PF represents a rare event. To date, no more than 30 cases of shift have been described in the literature. In our case it could be thought that VZV caused epitope-spreading that led to transition from PV to PF. Indeed, although it has not been demonstrated a causal relationship between PV and herpetic infections, it has been postulated that viral infections could lead to autoimmune process unmasking previously sequestered epitopes.

