

CYTOTOXIC AND ANTIGEN PRESENTING CELLS, AND NON-BASEMENT MEMBRANE ZONE PATHOLOGY IN A CASE OF BULLOUS PEMPHIGOID

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As the most common autoimmune blistering disease, many dermatologists have made efforts to elucidate the mechanism of bullous pemphigoid (BP) to date. One of their most important findings is that the target antigens of BP autoantibodies are two protein components of the hemidesmosome, a 180-kDa transmembrane protein member of the collagen family (BP180/type XVII collagen/BPAG2) and a 230-kDa protein member of the plakin cytoskeleton linker family (BP230/BPAG1e) [1-4]. Although anti-BP230 autoantibodies in BP patients directly contribute to BP pathogenesis is a matter of controversy [5-6], several studies suggested that IgG autoantibodies against BP180 contribute to blister formation in BP [7-8]. Using the experimental animal models, Liu et al. showed that blister formation depends on complement activation, mast cell degranulation and neutrophil infiltration [9-11]. However, contrary to this conclusion, recent studies indicate that cultured keratinocytes treated with BP-IgG exhibit a reduction in adhesive strength and a loss in expression of BP180 [12]. Furthermore, recently, many groups pay attention to the association between BP and IgE autoantibodies against BP180 [13-15]. Most of above researches focused on the mechanism of blister and erythema formation in BP. In addition, some recent papers showed that cardiovascular and neurological diseases are associated with BP [16-19]. However, the contribution of these complications on the pathogenesis of BP is still absolutely unclear. Now Velez et al. convincingly showed autoantibody deposition and inflammation on dermal eccrine sweat glands, blood vessels and nerve, which may give us a hint of the mechanism which can cause cardiovascular and neurological diseases. Further studies are required.

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