

# LETTER TO THE EDITOR

## Management of erythematous skin lesions in bullous pemphigoid associated with atopic dermatitis

Dear Editor,

Bullous pemphigoid (BP) is an autoimmune blistering disease caused by immunoglobulin (Ig)G antibodies against type XVII collagen/BP180 (BP180).<sup>1</sup> This tensely blistering disease is characteristically accompanied by pruritic urticarial erythema, and long-lasting non-specific skin eruptions, such as eczematous papular and/or erythematous lesions, may precede the formations of blisters. In the prodromal phase, it is thus difficult to distinguish BP lesions from other inflammatory skin disorders. Here, we report a case of BP associated with atopic dermatitis (AD), in which BP shared refractory erythematous lesions with AD.

A 54-year-old Japanese woman was referred to us because erythematous lesions on the trunk and extremities

had persisted for 3 months. The patient had been treated for AD since childhood. On our first examination, eczematous papular and erythematous lesions were observed on the trunk (Fig. 1a) and four extremities (Fig. 1b). Itchy edematous erythema with a tendency to central clearing was more prominently observed on the extensor surfaces of the four extremities. We tentatively diagnosed the eruption as AD. However, tense blisters arose on the erythematous lesions of her left upper limb (Fig. 1c). Histopathological findings showed a subepidermal blister and an eosinophil infiltrate (Fig. 1d). Direct immunofluorescence showed linear IgG (Fig. 1e) and C3 (Fig. 1f) but no IgE deposition along the epidermal basement membrane. Circulating IgG anti-BP180 antibodies were detected by BP180 enzyme-linked immunoassay

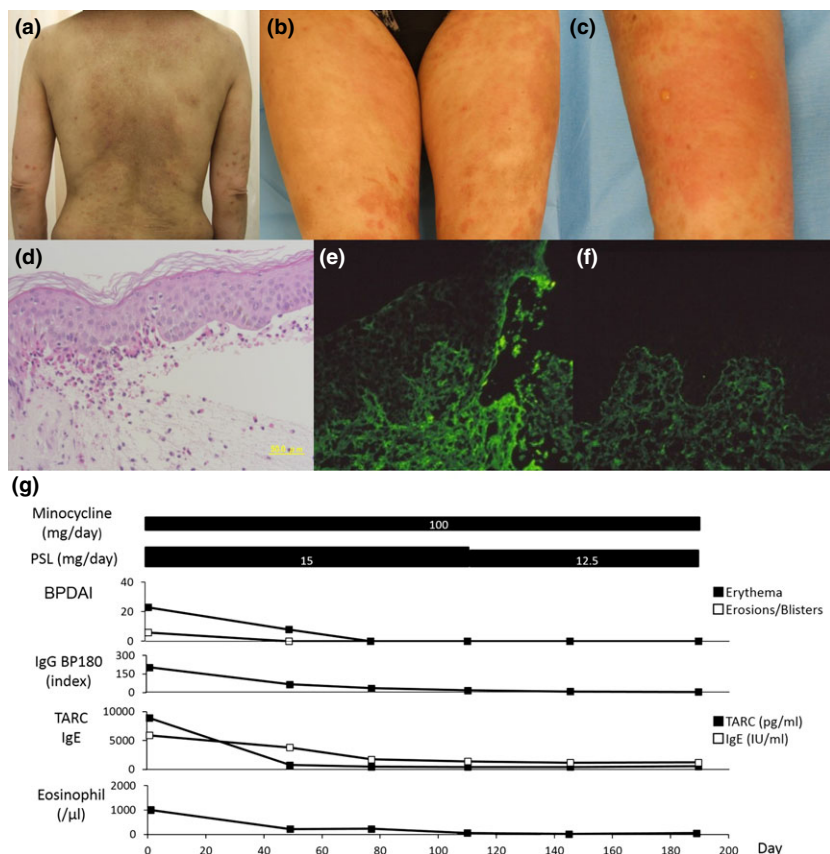


**Figure 1.** Clinical appearance, histopathological findings, direct immunofluorescence results and clinical course. Erythematous lesions were observed on the (a) trunk and (b) extremities. (c) Tense blisters were also observed on the left upper limb. (d) Histopathology, showing a subepidermal blister with eosinophil infiltration in the upper dermis (hematoxylin–eosin, original magnification  $\times 200$ ). Bar, 50  $\mu$ m. Direct immunofluorescence, showing (e) linear immunoglobulin (Ig)G deposition and (f) C3 deposition at the basement membrane zone. (g) During the clinical course, the levels of IgG anti-BP180 antibodies and CCL17/thymus and activation regulated chemokine (TARC) were decreased in association with symptomatic improvement. BPDAL, bullous pemphigoid disease area index; PSL, prednisolone.

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(ELISA; index, 202.0; positive cut-off value, >9.0). The patient was diagnosed with BP.

During the clinical course, we monitored the disease severity with a consensus BP disease area index (BPDAl) scoring, including BPDAl for erythema and BPDAl for erosions/blisters,<sup>2</sup> serum CCL17/thymus and activation regulated chemokine (TARC) and total IgE levels, and peripheral eosinophil counts as well as IgG anti-BP180 antibody levels. After treatment with oral prednisolone 15 mg/day and minocycline 100 mg/day, the clinical symptoms were rapidly improved with marked reductions of all the parameters (Fig. 1g). While AD is a T-helper (Th) 2-dominant inflammatory skin disease especially in the acute phase,<sup>3</sup> the autoimmunity of BP may be mediated by Th2 cells. Currently, we have shown that IgE anti-BP180 antibody levels correlate well with the disease severity of urticarial erythema in a BP patient.<sup>4</sup> Thus, we hypothesized that IgE anti-BP180 antibodies were associated with the erythematous eruption in this case. However, IgE anti-BP180 antibodies had not been detected by ELISA during the clinical course.

In our case, skin lesions suddenly deteriorated and became refractory to topical corticosteroid therapy, suggesting that she developed a skin disease other than AD. The patient's IgG anti-BP180 antibody levels fluctuated in parallel with the severity of erythematous lesions. The serum CCL17/TARC levels, known as a severity marker of AD, also reflected the disease severity of BP.<sup>5</sup> In our case, CCL17/TARC levels mainly reflected the disease severity of BP. Dual monitoring of serum CCL17/TARC levels and IgG anti-BP180 antibody

levels is required for evaluation of BP with severe erythematous lesions. It should be kept in mind that the elevation of serum CCL17/TARC may be a sign of the onset of BP in AD patients.

**CONFLICT OF INTEREST:** None declared.

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