Bullous Pemphigoid on Psoriasis Lesions after UVA Radiation

Hisayo Washio[1], Hiroyuki Hara[1], Hiroyuki Suzuki[1], Mariko Yoshida[2] and Takashi Hashimoto[2]
[1]*Department of Dermatology, Nihon University School of Medicine, 30-1 Oyaguchi-kamimachi, Itabashi-ku, Tokyo, 173-8610 and* [2]*Department of Dermatology, Kurume University School of Medicine, 67 Asahi-machi, Kurume, Fukuoka, 830-0011, Japan.*
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Sir,

The occurrence of bullous pemphigoid (BP) in patients with psoriasis has occasionally been reported. Most previously reported cases attributed the occurrence of BP in psoriasis to photochemotherapy, such as PUVA, UVA and UVB (1–4). We describe here a patient who initially had psoriasis vulgaris and subsequently developed BP after treatment with UVA for psoriasis. Histopathologic, direct immunofluorescence microscopy (DIF), indirect immunofluorescence (IIF) and immunoblot features fit the diagnosis of BP.

CASE REPORT

A 67-year-old woman had psoriasis vulgaris for 34 years which was controlled with etretinate 40 mg daily and topical steroids. In her past history she had diabetes, hypertension and arthritis. She complained of worsening psoriasis which was treated with UVA. After 3 weeks, she suddenly developed blisters, 5–15 mm in diameter, on the trunk and extremities. Blisters developed on the psoriatic plaques (Fig. 1a). The mucous membranes were not involved. The laboratory tests revealed leucocytosis (7.7×10^3 μl) with eosinophilia (11%). Skin biopsy specimens were taken from a blister on the psoriatic plaque.

Histopathological examination showed pronounced acanthosis with parakeratosis and elongation of the rete ridges in the epidermis, and a subepidermal blister with numerous eosinophils and nets of fibrin was seen in the upper dermis. The dermis showed mild inflammatory infiltrate composed of lymphocytes.

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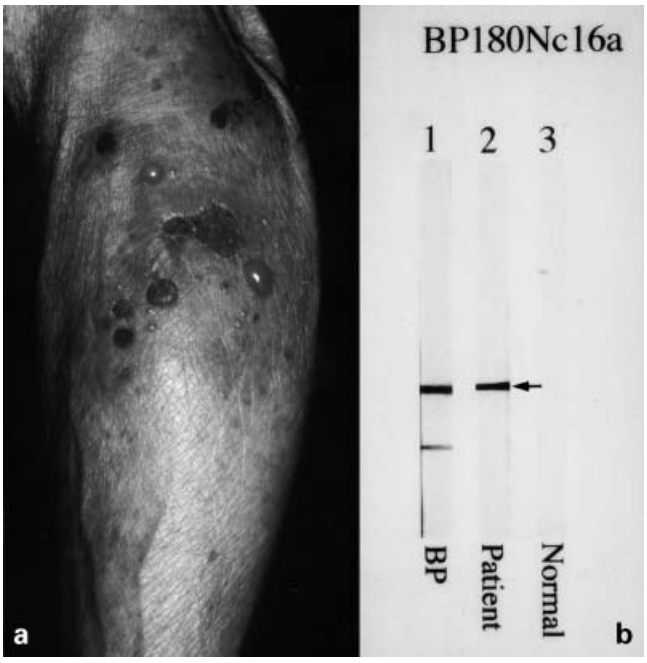

Fig. 1. (a) Tense bullae are observed on the psoriatic plaque. (b) Immunoblot of epidermal extract from a patient with bullous pemphigoid (BP), the study patient and a healthy control (Normal). Reactivity of BP180 NC16a is indicated by arrow.

DIF of both the psoriatic lesion and bullous lesion revealed a linear deposition of IgG and C3 along the basement membrane zone (BMZ). IIF of normal human skin was performed by the standard method with anti-human IgG antiserum as a secondary antibody. IIF using 1 M NaCl split skin was performed using anti-human IgG antiserum as a secondary antibody and circulating anti-BMZ antibody reacted with the epidermal side of the split with IIF of 1 M NaCl split skin. Immunoblotting of epidermal extracts and recombinant protein was performed (5). The patient's serum clearly reacted with the recombinant protein of the BP180 NC16a domain (Fig. 1b).

A diagnosis of the coexistence of psoriasis vulgaris and BP was made. As the combination of tetracycline and nicotinamide is known to be a useful alternative to systemic steroids for the treatment of BP, treatment with 200 mg of doxycycline hydrochloride and 900 mg of nicotinamide daily was initiated (6). The blisters cleared and disappeared after 10 days of the treatment. She is now in remission.

DISCUSSION

We present a patient with BP on psoriasis lesions occurring after UVA irradiation.

It is well known that the classic BP serum reacts with BP180 and BP230 antigens (7). The BP180 NC16a domain is considered to be the most immunogenic site (8) and the target epitope recognized by the autoantibodies using Western immunoblotting. We consider that the BP180 NC16a region is implicated as the pathogenic antigen in BP occurring on psoriasis vulgaris lesions. In another previously reported case, IgG autoantibodies labelled a 200-kDa epidermal protein and circulating anti-BMZ antibody reacted with the dermal side of the split with IIF (9).

Although the pathogenic mechanism of coexisting psoriasis vulgaris and BP is unclear, a common immunogenetic mechanism might be involved. Most cases of BP after UV exposure previously reported have induced the production of BP autoantibodies. Muramatsu et al. (10) suggested that BP antigen is susceptible to UVB exposure, which probably leads to configurational changes in antigen or as a secondary phenomenon. In psoriatic skin, there is expression of α-6 integrin and β_1 integrin (11). In vitro, an interaction between α-6 integrin and BP180 has been reported and epidermal integrins may play a role in the regulation of epidermal cell proliferation (12, 13). Hopkinson et al. (13) demonstrated that BP180 and α-6 integrin interaction is not only mediated by the BP epitope but is necessary for hemidesmosome formation. One possibility is that UV radiation might alter BMZ antigenicity and expose or release altered antigens that might result in the stimulation of antibody formation against the BMZ.

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