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Concurrence of Bullous Pemphigoid and Psoriasis: A Case Report

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Abstract

Bullous pemphigoid (BP) and psoriasis are chronic recurrent inflammatory skin diseases. The pathogenesis of concurrence of BP with psoriasis is still unknown. A 39-year-old male with a five-year history of chronic plaque psoriasis developed itchy large tense bullae on the trunk and upper extremities after he had been receiving narrow band ultraviolet B (NBUVB) therapy over five months. Skin biopsy from bulla on the trunk showed typical histological features of BP. Direct immunofluorescent staining showed deposit of immunoglobulin G and C3 in the basement membrane zone (BMZ) which supported the diagnosis of BP. It has been postulated that the autoimmune process responsible for BP lesions might be induced by ultraviolet light therapy and/or the inflammatory processes that occur in psoriasis.

Key words: Pemphigoid, Bullous; Psoriasis; Skin Diseases; Comorbidity; Diagnosis; Case Reports

Introduction

Bullous pemphigoid (BP) is an autoimmune disease characterized by multiple tense blisters arising on normal or erythematous skin with a predilection for flexural areas (1-3). Autoimmunity has been established as the etiology of BP, which presents as immunoglobulin G (IgG) and C3 deposits along the epidermal basement membrane zone (BMZ) (1, 2). Another common skin autoimmune disease is psoriasis, a condition characterized by erythematous plaques with silvery white scales. The most common affected areas are scalp, elbows, knees, and lumbosacral region. Environmental trigger factors such as trauma, medications, and infections, in addition to immunological and genetic factors, appear to play a role in the pathogenesis of psoriasis (4).

The pathogenesis of BP in concurrence with psoriasis is still unknown. The trigger factors responsible for the concurrence of these two diseases has been reported previously such as photochemotheraphy of psoralen ultraviolet A (PUVA) (6) and narrow band-ultraviolet B (NB-UVB). This suggests that psoriasis as a chronic inflammatory disease and the effect of the local ultraviolet radiation provides a particular predisposition of the immune system leading to autoimmune response such as developing BP lesions (5). We report a case of BP in a patient who suffers from psoriasis vulgaris that might be induced by NB-UVB.

Case Report

A 39-year-old male presented with pruritic, large tense bullae on the trunk and upper extremities which had appeared two weeks before admission. Initially, the blisters developed on the arms, then spread into other parts of the body. The patient had had chronic plaque psoriasis for five years. In addition to methotrexate and topical corticosteroids administration, the patient received NB-UVB for psoriasis. After five months of this treatment, he developed multiple bullae on his trunk and extremities. Physical examination revealed tense bullae on the abdomen, upper, and lower limbs. The Nikolsky sign was negative. There were erythematous scaly plaques on the lower extremities and the blisters also ap-

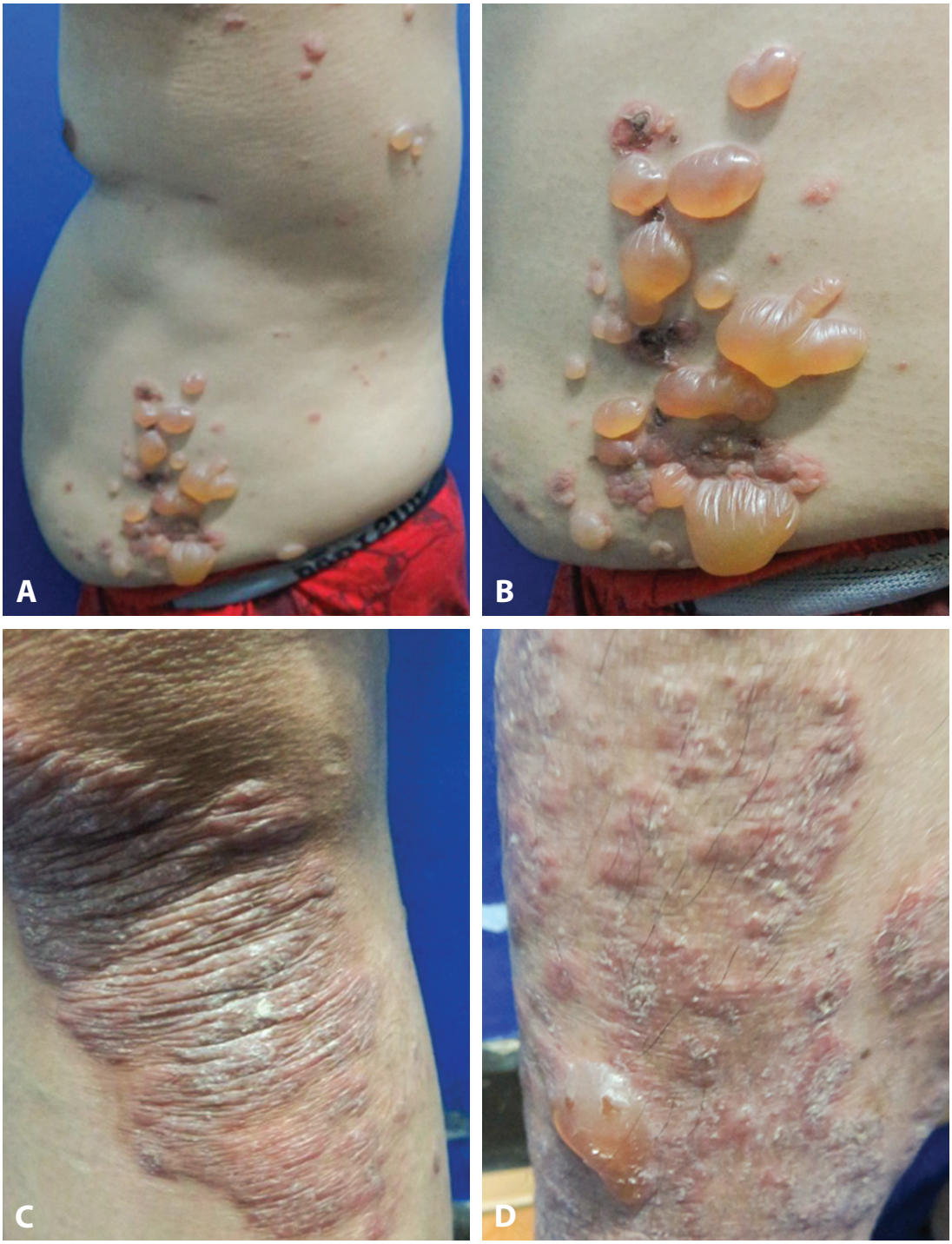

Figure 1. A, B. Blisters on the trunk. 1. C, D. Scaly plaques with blister on the lower extremity.

peared over the scaly plaques (Fgure 1. A-D). The skin biopsy from erythematous scaly plaque showed psoriasiform reaction with parakeratosis, acanthosis, and elongated rete ridges,which supported the diagnosis of psoriasis (Figure 2. A). Histopathological examination from bullae on the trunk revealed a sub-epidermal blister containing eosinophils (Figure 2.B). Direct immunofluorescence (DIF) from perilesional skin showed a linear depos-

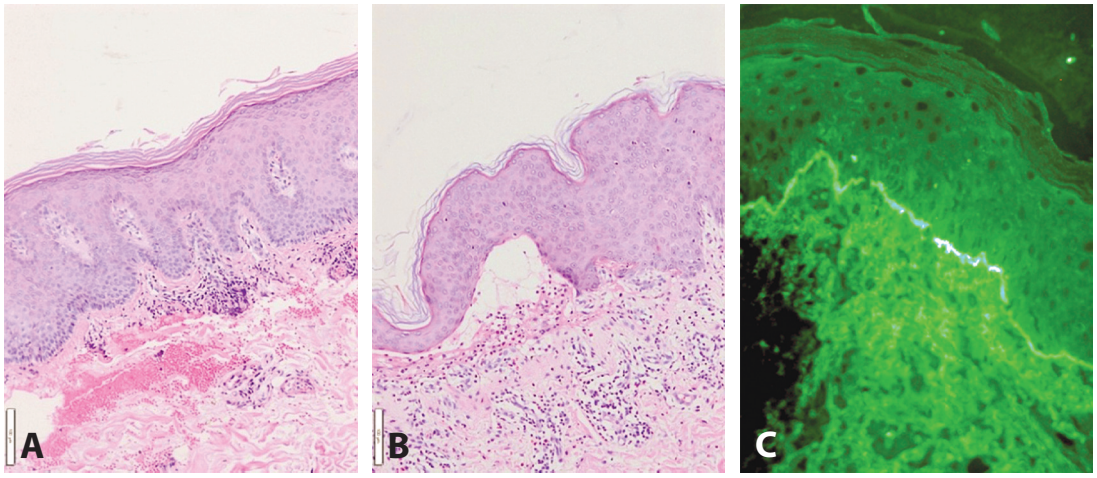

Figure 2. A). Psoriasiform reaction with parakeratosis, acanthosis, and elongation of rete ridges B). Subepidermal blisters containing eosinophils, C). Linear deposit IgG and C3 at BMZ.

it IgG and C3 at BMZ (Figure 2.C) which provided the evidence of BP.

The patient was therefore treated with 15 mg methotrexate/week orally, 64 mg of prednisone (1 mg/kgBW/day), and topical desoximethason 0.25% cream. The NB-UVB phototherapy was discontinued. At one-month follow-up, psoriatic lesion improved (Table 1) and at three-month follow-up, both conditions subsided, leaving only post-inflammatory pigmentation.

Discussion

Several cases of psoriasis and autoimmune bullous diseases have been reported; however, the relationship between two diseases remains unknown (7). In most cases, psoriasis precedes the onset of autoimmune bullous disease with an average interval of 20 years (8). In this case, psoriasis developed approximately five years before BP.

In BP patients, there is an auto-reactive response of T cells and B cells to BP antigen 1 (BPAG1/230 kDa) and BPAG2 antigens (180 kDa) (9). Several precipitating factors such as ultraviolet (UV) exposure, radiation therapy, trauma, drugs, malignancy, and autoimmune diseases have been reported to be associated with BP (10).

The mechanism of coexistence of psoriasis and BP in one patient is unclear (5). It is still the subject of controversy whether it is due to the treatment of psoriasis, pathological events at the basement membrane zone (BMZ) in psoriasis itself, common immunological or immunogenetic mechanisms, or a coincidence of multiple factors precipitating the autoimmune bullous diseases (11). A reduced barrier function of the psoriatic epidermis combined with the irritant effects of therapies administered for psoriasis may precipitate blister formation (1). Among different antipsoriatic therapies, UV radiation is most commonly suspected of triggering BP. It is suggested that UV radiation may precipitate BP by immunological alteration of the epidermal antigens. The result is formation of complement-binding anti-BMZ antibodies leading to bullous skin lesions (6). Washio, et al. (12)

Table 1. Follow up of PASI and BPDAI after treatment

Week	I	II	III	IV	V	VI	VII	VIII
PASI	5,7	4,8	2,4	1,2	0	0	0	0
BPDAI (bullae/erosion)	24	20	16	12	6	0	0	0

PASI: psoriasis area severity index, BPDAI: Bullous Pemphigoid Disease Area Index

suggested alterations of the BMZ antigenicity by UV radiation that might lead to the release of antigens and consequently stimulation of autoantibody production. The development of bullous lesions in this psoriasis patient suggested that NB-UVB radiation could have provoked subclinical BP.

The diagnosis of autoimmune bullous disease is based on clinical features, histopathological examination, and DIF (13). The clinical features of BP are large tense bullae in predilection site and negative Nikolsky sign (14). Histopathological examination of BP shows a sub-epidermal blister with characteristic infiltrates containing eosinophils (3, 14). DIF is a laboratory examination that has become the gold standard for the diagnosis of autoimmune bullous disease (7). DIF of BP lesion revealed a linear arrangement of IgG and C3 deposits in BMZ (15). In this case, the DIF examination showed a linear deposit of IgG and C3 in the dermo-epidermal junction.

The diagnosis of psoriasis is based on clinical manifestation and histopathological examination, especially in difficult cases (15). Histopathological features of advanced psoriasis are psoriasiform hyperplasia with acanthosis, elongation of rete ridges, parakeratosis, hypogranulosis, and microabses of Munro (17). In this case, the patient had suffered from psoriasis for the last five years, as confirmed by histopathological examination.

There are some alternative therapeutic modalities for patients with coexisting BP and psoriasis. Immunosupressive drugs proved to be effective in both diseases (18), such as methotrexate (19), cyclosporine (20), combination of low-dose cyclosporine and low dose systemic steroids (21), dapsone (22), azathioprine (23), mycophenolate mofetil (24), and acitretin (25). A combination of methotrexate and systemic corticosteroid for these two diseases has never been reported. The combination treatment was effective in this patient, since after the long-term follow-up there were no side effects.

Conclusion

In conclusion, the pathogenesis of concurrent BP and psoriasis remains unknown. One of the possible causes of BP in concurrence with psoriasis is that UV radiation might alter BMZ antigenicity and release altered antigens that might result in the stimulation of antibody against the BMZ. Due to the fact that BP and psoriasis are immunologically mediated, a combination of immunosuppressive regimens directed at cellular and humoral factors usually results in clinical improvement.

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Istovremena pojava buloznog pemfigoida i psorijaze – prikaz slučaja

Sažetak

Bulozni pemfigoid (BP) i psorijaza su hronične recidivne inflamatorne kožne bolesti. Još nije poznata patogeneza istovremene pojave buloznog pemfigoida i psorijaze. Tridesetdevetogodišnji muškarac, koji je imao hroničnu plak psorijazu pet godina, dobio je veliku čvrstu bulu koja svrbi na trupu i gornjim ekstremitetima nakon što je primao ultraljubičastu B-terapiju uskog opsega pet meseci. Biopsija kože iz bule sa trupa pokazala je tipične histološke odlike buloznog pemfigoida. Direktno imunofluorescentno bojenje pokazalo je deponovanje imunoglobulina G i C3 u bazi membranske zone (BMZ) što je potvrdilo dijagnozu buloznog pemfigoida. Pretpostavlja se da bi se autoimuni proces koji je odgovoran za bulozni pemfigoid lezije mogao izazvati ultraljubičastom svetlosnom terapijom i/ili inflamatornim procesima koji se dešavaju u psorijazi.

Ključne reči: Bulozni pemfigoid; Psorijaza; Kožne bolesti; Komorbiditet; Dijagnoza; Prikazi slučajeva

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