QUIZ SECTION

MJS PUBLISHING

Erythema and Blisters in a Patient with Psoriasis: A Quiz

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A 53-year-old man with a 16-year history of psoriasis developed erythema and blisters on his trunk and limbs 20 days earlier, accompanied by severe itching. Physical examination on admission revealed flaky, ring-like oedematous erythema on the trunk and limbs, with pinhead-to-green bean-sized blisters (Fig. 1). The blister wall was tense, the blister fluid was clear, Nicolsky sign was negative, and some blisters showed erosive surfaces with crusted and exfoliated epidermis. Scattered dark red patches, which were well circumscribed and overlaid with thin layers of white scales, were observed on the lower back.

What is your diagnosis?

Differential diagnosis 1: Pemphigus herpetiformis
Differential diagnosis 2: Dermatitis herpetiformis
Differential diagnosis 3: Linear IgA bullous dermatosis
Differential diagnosis 4: Erythema ultiforme

See next page for answer.

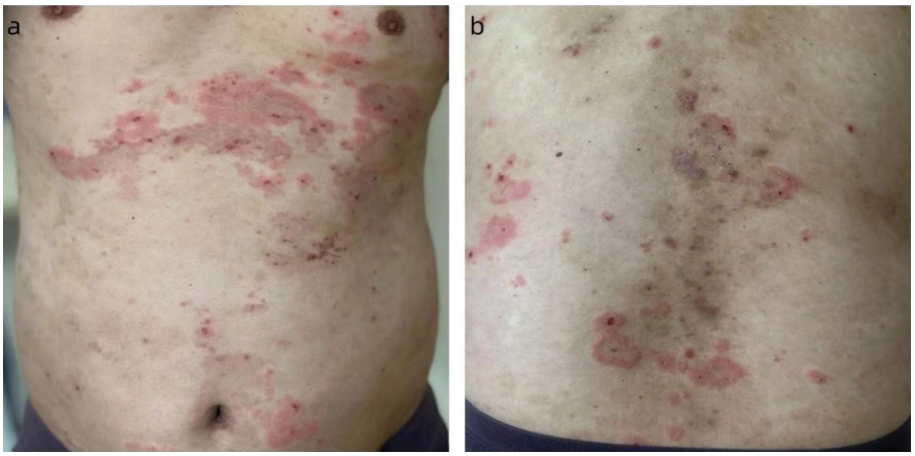

Fig. 1. Clinical image. (a, b) Oedematous erythema and blisters on the trunk. (b) Scattered dark red patches on the lower back. Written permission has been given by the patient to publish these photos.

ANSWERS TO QUIZ

Erythema and Blisters in a Patient with Psoriasis: A Commentary

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Diagnosis: Pemphigus herpetiformis

Histopathological examination of the patient's new blisters revealed blisters on the upper part of the epidermal spinous layer, with acantholytic cells and neutrophils in the blisters. There was perivascular lymphocytic infiltration with a few eosinophils in the superficial dermis (Fig. 2). Direct immunofluorescence of the skin surrounding the vesicles revealed grid deposition of C3 and IgG between epidermal keratinocytes (Fig. 3), and serum anti-Dsg1 antibodies were elevated (>150 U/mL), while anti-Dsg3 antibodies were not detected by enzyme-linked immunosorbent assay. The patient was diagnosed with pemphigus herpetiformis based on clinical, histological, and immunological findings. Methylprednisolone (30 mg once daily) in combination with mycophenolate mofetil (1 g twice daily) supplemented with topical mometasone furoate cream was used to control the disease. Notably, after 1 week, a significant improvement in clinical symptoms was observed, with subsiding blisters and erythema, albeit incomplete resolution of the psoriatic lesions. Throughout the steroid tapering phase, no recurrence of erythematous scaly skin lesions or blisters occurred, indicating ongoing disease control. The patient is still under regular follow-up.

Pemphigus herpetiformis (PH) is a rare subtype of pemphigus that is classified under the spectrum of autoimmune bullous diseases (ABDs). This variant was initially documented by Jablonska et al. (1) in 1975. PH combines the clinical presentation of dermatitis herpetiformis with the immunological characteristics of pemphigus, creating a unique diagnostic challenge. On the other hand, psoriasis is an immune-mediated chronic recurrent inflammatory systemic disease induced by interactions between multiple genetic and environmental risk factors. Both ABD and psoriasis are characterized by their propensity to invade the epidermis, with partially overlapping pathogenic mechanisms. In recent years, several studies have highlighted a correlation between psoriasis and ABD, particularly bullous pemphigoid. However, the exact underlying mechanism linking them remains unclear.

Pemphigus is an autoimmune disease that is related to the formation of autoantibodies against the epidermis, and psoriasis patients also exhibit immune abnormalities. In both diseases, the immune response targets the epidermis and induces disease following structural disruption of epidermal cells or abnormal proliferation and differentiation of epidermal cells. Recent epidemiological studies and case reports have highlighted a link between these 2 conditions. Kridin et al. (2) reported that the prevalence of psoriasis in patients with various types of pemphigus was 2.4% and noted a significant correlation between pemphigus and psoriasis. It was also observed that patients with psoriasis had a higher likelihood of developing pemphigus than normal individuals (3). In addition, Balighi et al. (4) reported 3 patients with pemphigus vulgaris who developed secondary psoriasis after symptom relief. Daulat et al. (5) reported that in a patient with psoriasis, the disease transformed to pemphigus vulgaris (PV) after the use of etanercept. Zheng et al. (6) reported a patient who had psoriasis for 20 years that was suddenly complicated by PV. Increasing evidence suggests an association between psoriasis and pemphigus, but the exact mechanisms underlying the co-occurrence of the 2 diseases remain unclear.

In recent years, epitope expansion has been found to be involved in ABD development and transformation, and this phenomenon has been confirmed in animal experiments (7). Epitope expansion exposes more epitopes and causes the original disease to aggravate, transform, or complicate other diseases. Most ABD occurs after the onset of psoriasis, and some patients have both ABD and psoriasis at the same time or after ABD, suggesting that there is some common pathogenesis between the 2 diseases. In addition, the 2

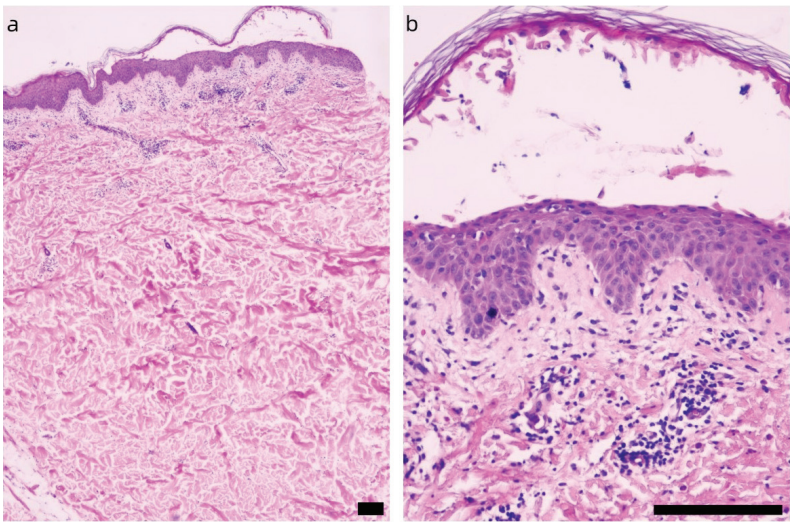

Fig. 2. Pathology of lesions. (a) Blisters on the upper part of the epidermal spinous layer (haematoxylin and eosin staining: HE, scale bar: 100 μm). (b) Acantholytic cells and neutrophils in the blisters. Perivascular lymphocytic infiltration with a few eosinophils in the superficial dermis (HE, scale bar: 100 μm).

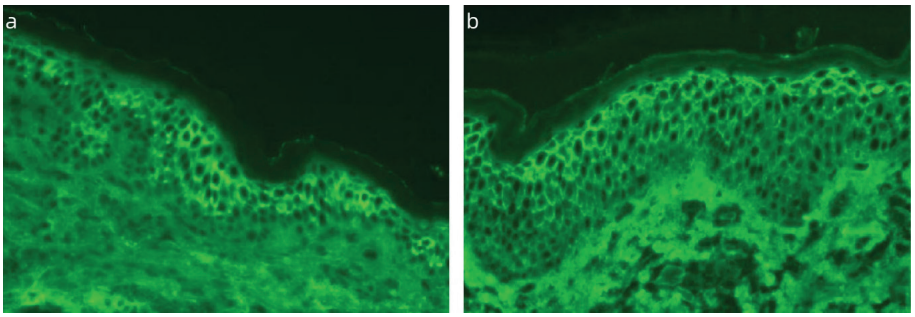

Fig. 3. Direct immunofluorescence. Grid deposition of C3 (a) and IgG (b) between epidermal keratinocytes.

diseases can also be converted to each other. Morais et al. (8) reported that PH is mainly a Th2-dominated disease, while the pathogenesis of psoriasis is mainly driven by Th1/Th17 cells in the body. Studies have shown that Th1- and Th2-dominant immune responses change correspondingly when psoriasis and ABD are transformed (9). Therefore, the immune function of the body, especially the interconversion between Th1 and Th2, may be the reason for the interconversion between the 2 diseases.

In this particular case, the patient developed pemphigus herpetiformis while his psoriasis remained stable without treatment. The severity of the 2 diseases was not significantly correlated, which may be caused by autoimmune dysfunction. However, the possibility that the 2 diseases occurred independently in the same patient cannot be completely excluded. The physicians who treated this patient before admission overlooked the potential co-occurrence of psoriasis with ABD and misdiagnosed the patient with a recurrence of psoriasis. Although psoriasis with PH is rare in clinical practice, dermatologists should consider the association of both conditions when treating psoriasis or ABD.

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