Thick stratum corneum may inhibit drug penetration, rendering the degree of keratinization, which is a possible explanation for the less impressive results on the common wart. Consequently, duct tape occlusion following the application of imiquimod may be helpful in overcoming this limitation.

In conclusion, imiquimod 5% cream and duct tape occlusion combination therapy is an effective alternative treatment modality for the treatment of the common verruca. Additional studies with larger numbers of patients including randomized double blind trials are required to establish its effectiveness.

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Selective Elevation of Antibodies to Desmoglein 1 during the Transition from Mucocutaneous to Cutaneous Type Pemphigus Vulgaris

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Dear Editor:

Pemphigus vulgaris (PV) is a group of autoimmune blistering diseases. Three types of PV have been classified including mucous PV, mucocutaneous PV (mcPV), and cutaneous PV (cPV). It has been reported that mcPV and cPV exhibit autoantibodies against both desmoglein (DSG)1 and

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DSG3, with a tendency toward predominant index values of anti-DSG3 in mcPV and with a predominance of anti-DSG1 in cPV[1-3]. A transition from mcPV to cPV has been observed[2], although the precise mechanism for the transition remains obscure. This report describes here an interesting difference in the pattern of changes in the serum levels of these autoantibodies between periods of mcPV and cPV in a case showing a transition from mcPV to cPV.

A 45-year-old female was referred in March 2002 with flaccid blisters on the trunk, anterior chest, and oral mucosa. A histologic examination of a blister on her chest demonstrated suprabasilar acantholysis and direct immunofluorescence detected significant deposition of immunoglobulin G (IgG) on the intercellular portion of the entire epidermis. An enzyme-linked immunosorbent assay revealed anti-DSG1 IgG (index value; 82) and anti-DSG3 IgG (index value; 150) autoantibodies in her serum (Fig. 1). She was treated with oral prednisolone (30 mg/d) and, additionally, mizoribine (200 mg/d). The lesions gradually disappeared. However, a highly-crusted and scaly erythe-

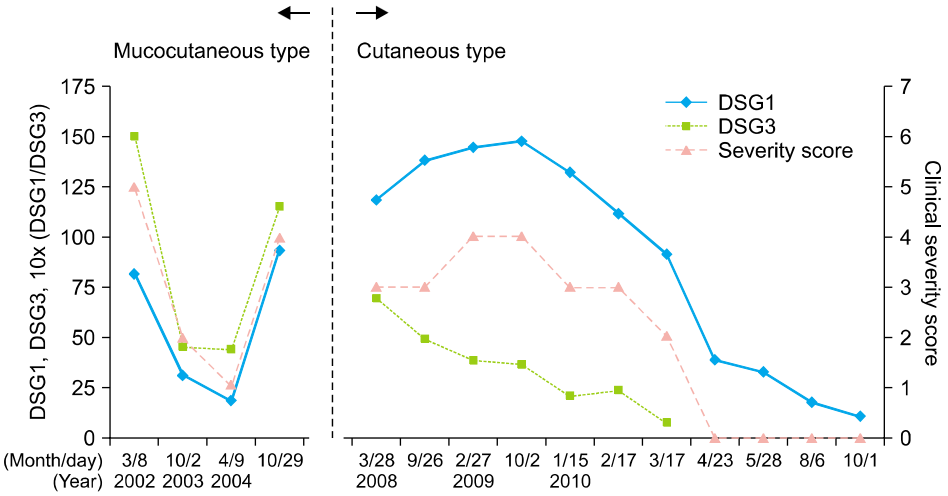

Fig. 1. Alteration of serum levels of antibodies to desmoglein (DSG) 1 and 3 and of clinical severity score. Serum levels of antibodies to desmoglein 1 and 3 fluctuated in parallel with disease severity on the stage of mucocutaneous type of pemphigus vulgaris (PV). On the stage of cutaneous type of PV, levels of antibodies to DSG1 (but not DSG3) fluctuated in parallel with disease severity. Each item in the severity index was scored from a minimum of 0 to a maximum of 3, and included: (i) the ratio of the affected area of skin to the total skin area as a percentage (0, none; 1, <5%; 2, 5~15%; and 3, >15%) (ii) the number of newly developed blisters per day (0, none; 1, occasional blisters; 2, 1~5 blisters; and 3, >5 blisters) and (iii) the presence or absence of oral lesions as a percentage (0, none; 1, <5%; 2, 5~30%; and 3, >30%). Consequently, the severity of a case was rated by the total of the scores.

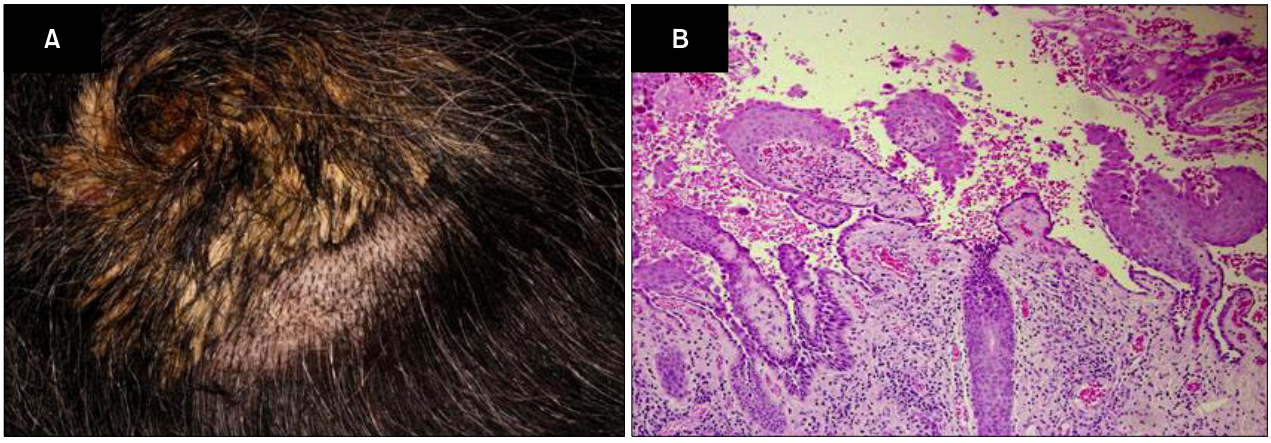

Fig. 2. Clinical and histopathological findings. (A) On the stage of cutaneous type of pemphigus vulgaris, highly-crusted and scaly erythema with partial erosions appeared on her scalp. Histologic examination of the erosion on her scalp disclosed suprabasilar acantholysis and papillomatosis without any apparent sign of superficial blister formation. (B) H&E, ×40.

ma with partial erosions appeared on her scalp in September 2005 in addition to sporadic erosions and bullae on her trunk (Fig. 2A). A histologic examination of the erosion on her scalp disclosed suprabasilar acantholysis with papillomatosis without any apparent sign of superficial blister formation (Fig. 2B). Since then, no apparent oral lesions have been found. The first examination of serum levels for autoantibodies after the relapse of the lesion on her scalp was performed in March 2008 and revealed a predominance of anti-DSG1 IgG (index value; 119) to anti-DSG3 IgG (index value; 69) autoantibodies (Fig. 1). The result of the serological examination supports the transition from mcPV to cPV. An increase in the dose of prednisolone successfully ceased the lesions.

Interestingly, when the clinical severity was assessed according to the revised severity index described previously[4], the levels of both anti-DSG1 and anti-DSG3 antibodies fluctuated in parallel with the disease severity at the period of mcPV and, in contrast, levels of anti-DSG1 alone (but not anti-DSG3) at the period of cPV (Fig. 1). This observation suggests that the selective activation of pathogenic anti-DSG1 antibody-producing cells might occur on the transition from mcPV to cPV. The selective elevation of anti DSG1 antibodies under the existence of subclinically levels of pathogenic antibodies against DSG3 could form the phenotype of cPV. A previous study demonstrated that the selective elevation of antibodies against DSG1 was observed at the relapse of mcPV in some cases[5]; however, the study did not describe whether transition from mcPV to cPV occurred at that time. The present case suggests that further studies to determine the precise relationship between the clinical manifestation and profile of serum levels for the autoantibodies should be conducted to explore the mechanism for the transition.

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