http://dx.doi.org/10.5021/ad.2016.28.5.660

A Case of Linear IgA Bullous Dermatosis Associated with Systemic Lupus Erythematosus

Ho-June Lee, Sook Jung Yun, Seung-Chul Lee, Jee-Bum Lee

Department of Dermatology, Chonnam National University Medical School, Gwangju, Korea

Dear Editor:

Linear Immunoglobulin A bullous dermatosis (LABD) is a rare, heterogeneous, acquired, subepidermal and autoimmune blistering disorder. It is characterized by linear deposits of immunoglobulin (Ig) A along the basement membrane zone (BMZ) that are seen by direct immunofluorescence (IF)[1]. The etiology of LABD is not fully understood, but some cases are reported in association with drugs, infections, malignancies and connective tissue disease[2].

A 33-year-old man presented with a 5-month history of pruritic erythematous papules and vesicles over the trunk and extremities. The skin eruptions were seen as dermatitis herpetiformis-like lesions (Fig. 1). The skin biopsy showed a subepidermal blister containing mainly lymphocytes and neutrophils infiltrated in the upper dermis (Fig.

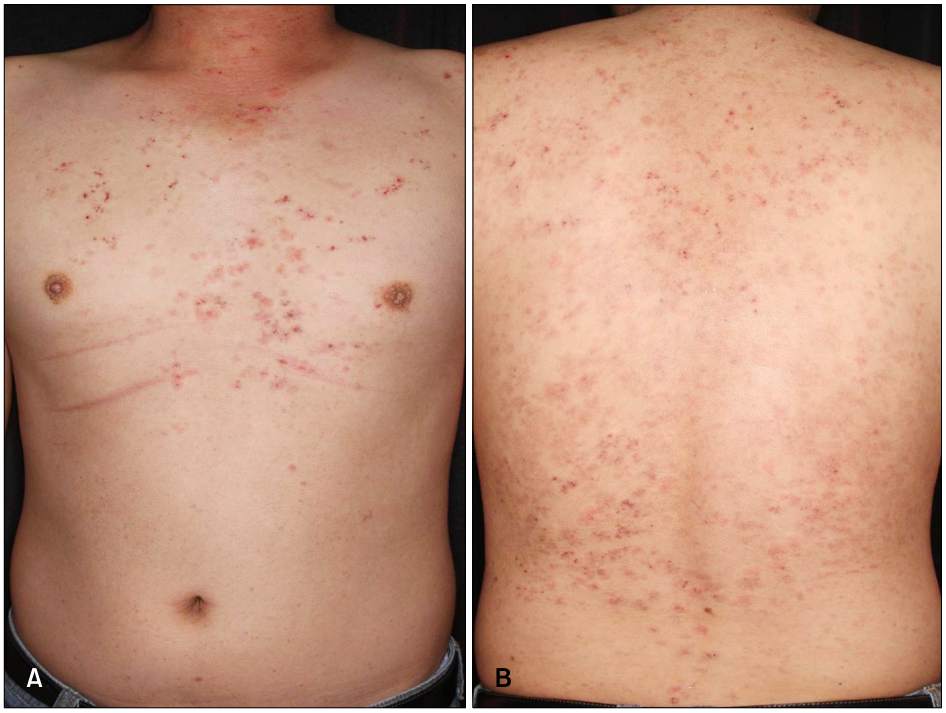

Fig. 1. Generalized erythematous vesicles and erosive patches arising on (A) chest and (B) back in a 33-year-old patient who developed linear IgA bullous dermatosis and systemic lupus erythematosus.

Received July 14, 2015, Revised September 20, 2015, Accepted for publication September 23, 2015

Corresponding author: Jee-Bum Lee, Department of Dermatology, Chonnam National University Medical School, 42 Jebong-ro, Dong-gu, Gwangju 61469, Korea. Tel: 82-62-220-6684, Fax: 82-62-222-4058, E-mail: jbmlee@chonnam.ac.kr

2A). For diagnosis, we performed additional tests including IF, immunoblotting and the enzyme linked immunosorbent assay (ELISA). The direct IF showed linear deposits of IgA and C3 in the BMZ, and the indirect IF with 1 M NaCl split-skin showed linear deposits of IgA and IgG in the epidermal side of 1 M NaCl-split skin (Fig. 2B). In immunoblotting, there are found 230 KDa and 120 KDa molecular sized polypeptides on IgA and IgG, corresponding to LAD-1 antigens, respectively (Fig. 2C). On ELISA (Medical & Biological Laboratories Co., Ltd., Nagoya, Japan), serum autoantibody level against BP180 NC1 was markedly elevated [BP 180 (38.648 U/ml), BP230 (2.498 U/ml), Dsg 1 (2.253 U/ml), Dsg 3 (9.606 U/ml)]. Most lamina lucida-type LABD sera react with the 97-kDa and 120-kDa LAD-1, truncated extracellular domains of BP180. Also in consideration our results of IF, immunoblotting and ELISA, we made a diagnosis of lamina lucida-type LABD[3]. Oral dapsone (100～200 mg/day) was started, but not responded favorably, and oral methylprednisolone (12 mg/day), mycophenolate mofetil (MMF) (250 mg/day) was added, but the skin lesions were waxed and waned.

Two months later, the patient's symptoms as malaise, fatigue, photosensitivity and multiple joints arthritis, presented mildly for several years, were aggravated. Laboratory tests revealed anemia, lymphopenia, high erythrocyte sedimentation rate and low C3/C4 levels. Anto-antibodies [(ANA;speckled(1:80), anti-dsDNA(−), anti-Scl-70(1+), antihistone(1+)] were checked. Also, proteinuria and hematuria were found. Renal biopsy revealed IgA lupus nephritis. Finally, systemic lupus erythematosus (SLE) was also diagnosed. Oral methylprednisolone (24 mg/day) and MMF (500 mg/day) were started. The skin lesions and SLE symptoms were improved and the patient has been taking low dose methylprednisolone and dapsone for maintenance since then.

LABD are known as dramatically responded to oral dapsone, inhibiting neutrophils-mediatied reaction as well as IgA adherence[4]. But our case was not responded. SLE accompanied with LABD may have altered the immune mechanism, which may in turn have resulted in unresponsiveness to dapsone.

Additionally, LABD and IgA lupus nephritis may have similar immune mechanisms related with the deposition of IgA in the BMZ and glomerular mesangium, which suggests the common immunopathogenic mechanisms might be shared in the pathogenesis of LABD associated with SLE[5].

In conclusion, our case indicates that the association of LABD and SLE is a rare and interesting event and further studies are needed to illuminate the common immunologic mechanisms between them.

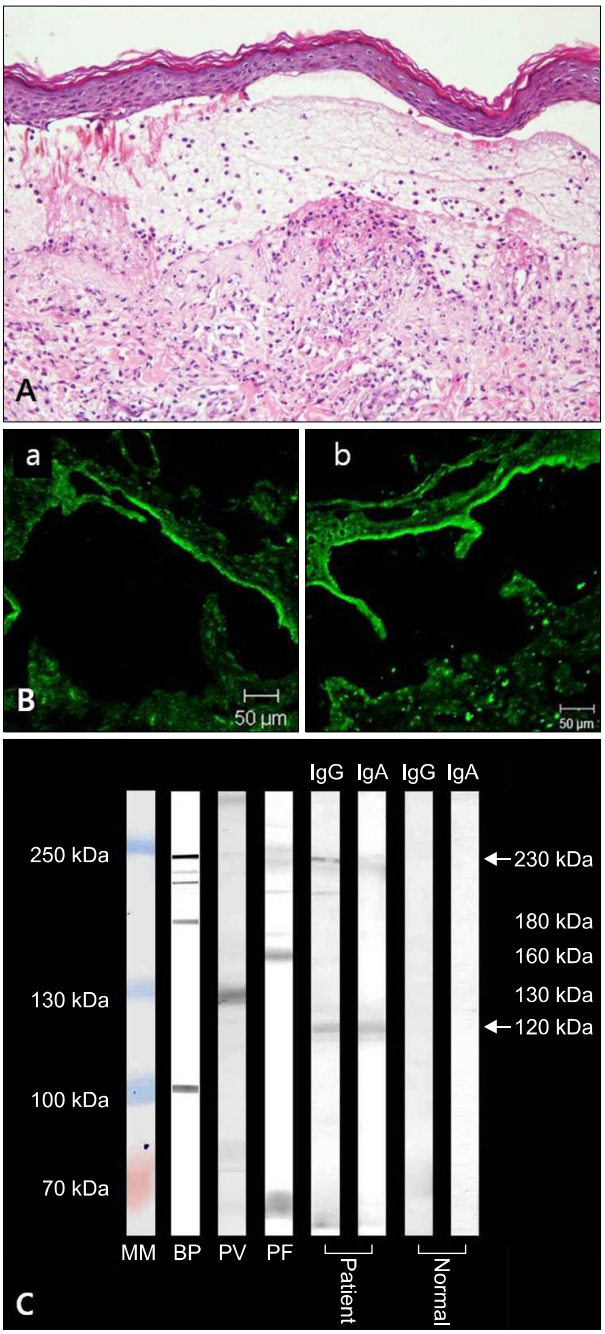

Fig. 2. (A) Histopathologic findings showing subepidermal blister and infiltration of lymphocytes and neutrophils in the upper dermis (H&E, ×200). (B) Indirect IF with 1 M NaCl split-skin showed linear depositions of immunoglobulin (Ig) A and IgG in the roof of separated skin (a: IgA, b: IgG). (C) Immunoblotting using normal human foreskin epidermal extracts showed 120 kDa, 230 kDa molecular weight bands on both IgA, IgG, respectively. MM: molecular weight marker, BP: bullous pemphigoid, PV: pemphigus vulgaris, PF: pemphigus foliaceus.

ACKNOWLEDGEMENT

This study was partially supported by the Chonnam National University Hospital Biomedical Research Institute (CRI13906-22).

REFERENCES

1. Wilson BD, Beutner EH, Kumar V, Chorzelski TP, Jablonska S. Linear IgA bullous dermatosis. An immunologically defined disease. Int J Dermatol 1985;24:569-574.
2. Venning VA. Linear IgA disease: clinical presentation, diagnosis, and pathogenesis. Dermatol Clin 2011;29:453-458.
3. Tsuchisaka A, Ohara K, Ishii N, Nguyen NT, Marinkovich MP, Hashimoto T. Type VII collagen is the major autoantigen for sublamina densa-type linear IgA bullous dermatosis. J Invest Dermatol 2015;135:626-629.
4. Wolf R, Matz H, Orion E, Tuzun B, Tuzun Y. Dapsone. Dermatol Online J 2002;8:2.
5. Ceri M, Unverdi S. IgA nephropathy coexisting with linear IgA bullous disease. Ren Fail 2011;33:101.

http://dx.doi.org/10.5021/ad.2016.28.5.662

Poor Capability of 3D-Cultured Adipose-Derived Stem Cells to Induce Hair Follicles in Contrast to 3D-Cultured Dermal Papilla Cells

Chang Hoon Seo[1], Mi Hee Kwack[1], Soo-Hong Lee[2], Moon Kyu Kim[1,3], Jung Chul Kim[1,3], Young Kwan Sung[1]

[1]Department of Immunology, Kyungpook National University School of Medicine, Daegu, [2]Department of Biomedical Science, CHA University, Seongnam, [3]Hair Transplantation Center, Kyungpook National University Hospital, Daegu, Korea

Dear Editor:

The dermal papilla (DP), a specialized mesenchymal component situated at the base of hair follicles, is thought to play a key role in controlling hair follicle development, hair growth, and hair-cyclic activity[1]. In experimental trials of hair regeneration, however, two-dimensional (2D)-cultured DP cells have been shown to lose their hair-inductive capacity (trichogenicity) during subculture[2]. Attempts have therefore been made to maintain trichogenicity of 2D-cultured DP cells by using a wide variety of methods, including supplementation with necessary factors as well as spheroid culturing[3]. Indeed, recent studies have shown that the trichogenicity of cultured human DP cells is markedly improved by the use of 3D-cultured cells (spheres) rather than 2D-cultured cells[4].

Here, we attempted to find a readily available mesenchymal cell source which can substitute for the role of trichogenic DP cells. Since adipose tissue contains a type of adult stem cells originating from the mesenchyme[5], we speculated on the potential role of adipocyte precursor cells or stem cells derived from adipose tissues. While the largest depot of adipose tissue is the abdominal subcutaneous adipose tissue under the skin, adipose tissue also exists within non-abdominal locations associated with the

Received April 3, 2015, Revised August 18, 2015, Accepted for publication September 24, 2015

Corresponding author: Young Kwan Sung, Department of Immunology, Kyungpook National University School of Medicine, 680 Gukchaebosang- ro, Jung-gu, Daegu 41944, Korea. Tel: 82-53-420-4874, Fax: 82-53-423-4628, E-mail: ysung@knu.ac.kr