**CASE REPORT**

A 53-year-old Japanese woman with a body weight of 40 kg noticed many bullae on the whole skin (Fig. 1a, 1b) and the oral mucous membrane (Fig. 1c). Histological study showed subepidermal bulla formation (Fig. 1e). DIF of a biopsy specimen from a bulla showed linear deposition of immunoglobulin (Ig) G and C3 in the basement membrane zone (Fig. 1f). IIF using normal human skin sections showed IgG anti-basement membrane zone antibodies, which reacted to the dermal side in IIF using 1 M NaCl-split skin. Immunoblot assay with extracts from normal human dermis showed that the patient's serum reacted with a 290-kDa band of collagen type VII (lane 3). In contrast, the patient's serum did not show reactivity to any antigens in the extract from normal human epidermis, p200 antigen (Fig. 1d, lane 2), or laminin 332. Antibodies to desmoglein 1 and 3, bullous pemphigoid (BP) 180, and BP 230 were not detected in the patient's serum by ELISA.

A diagnosis of EBA was made based on clinical features and histopathological findings, DIF, IIF using 1 M NaCl-split skin, or immunoblot analysis using dermal extracts. However, immunoblot analysis cannot detect autoantibodies against conformation-dependent epitopes, because denatured antigen sources are used as substrates. An enzyme-linked immunosorbent assay (ELISA) for detecting antibodies to NC1 and NC2 domains has been developed (5). We determined antibody titres to these antigens during the time-course of EBA using a commercially available ELISA kit in a patient with recurrent EBA. Antibody titres were reduced in parallel with the clinical activity of EBA.

Epidermolysis bullosa acquisita (EBA) is an acquired mechanobullous disease, characterized clinically by blisters and erosions of the skin and mucous membranes. Patients with EBA have circulating autoantibodies against type VII collagen, which is composed of 3 central identical collagenous α helical chains. Each α chain is flanked by the 145-kDa amino-terminal non-collagenous 1 (NC1) domain and the 34-kDa carboxy-terminal NC2 domain. Many epitopes in type VII collagen are located within the NC1 domain (1–3), and a few epitopes are located within the NC2 domain (4). Diagnosis of EBA is usually made by direct immunofluorescence (DIF), indirect IF (IIF) using 1 M NaCl-split skin, or immunoblot analysis using dermal extracts. However, immunoblot analysis cannot detect autoantibodies against conformation-dependent epitopes, because denatured antigen sources are used as substrates. An enzyme-linked immunosorbent assay (ELISA) for detecting antibodies to NC1 and NC2 domains has been developed (5). We determined antibody titres against these antigens during the time-course of EBA using a commercially available ELISA kit in a patient with recurrent EBA. Antibody titres were reduced in parallel with the clinical activity of EBA.

**Time-course of the Change in Titre of Antibodies Against Type VII Collagen in a Patient with Epidermolysis Bullosa Acquisita**

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with EBA, the titre was 54.9. More data are required to determine the antibody titres where EBA is clinically visible. Autoantibodies in EBA have recently been evaluated by ELISA using NC1 and NC2 domains (5), and 2% of EBA sera have been found to bind exclusively to the NC2 domain. Therefore, although some reports have described the use of an ELISA only for the NC1 domain (2, 5), an ELISA using a combination of the NC1 and NC2 domains of type VII collagen is more useful for the diagnosis and monitoring of disease activity in EBA. As there is no index for clinical disease activity of EBA, we evaluated disease activity using PDAI, which is usually used for pemphigus patients (6). The present case shows that autoantibodies measured by ELISA clearly fluctuated in parallel with PDAI. These findings indicate that an ELISA could be valuable for monitoring the disease activity of EBA, and ELISA scores may be useful for tapering corticosteroid use and for therapeutic planning.

The authors declare no conflicts of interest.

REFERENCES