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http://dx.doi.org/10.5021/ad.2014.26.2.258

Pemphigus Vulgaris in Pregnancy Associated with Herpes Virus Type 1 Infection

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Dear Editor:

Pemphigus vulgaris (PV) rarely occurs during pregnancy. We report a case of PV associated with herpes simplex type 1 virus (HSV-1) which occurred in the third trimester of pregnancy.

A 32-year-old second gravida at 37 weeks' gestation was admitted for multiple bullous skin lesions that persisted for over a month.

These vesicular and erosive lesions initiated from the periumbilical region and spread to the oral mucosa and the skin of the back (Fig. 1A, B). The diagnosis of PV was confirmed by biopsy (Fig. 1C), and direct immufluorescence detected anti-immunoglobulin G and C3 antibodies. Anti-desmoglein 1 and anti-desmoglein 3 antibodies were elevated at 82.1 U/ml (normal, <14 U/ml) and 184.9 U/ml (normal, <7 U/ml) in peripheral blood. Tzanck smear and viral polymerase chain reaction (Seeplex STD B41 Detedtion; Seegene, Seoul, Korea) were done on the base of a vesicular lesion on the trunk. Tzanck smear was negative, but, viral polymerase chain reaction (PCR) was positive for HSV-1 (Fig. 2).

Prednisolone at a dose of 20 mg/d was initiated. Foll-

Received December 6, 2012, Revised March 23, 2013, Accepted for publication April 16, 2013

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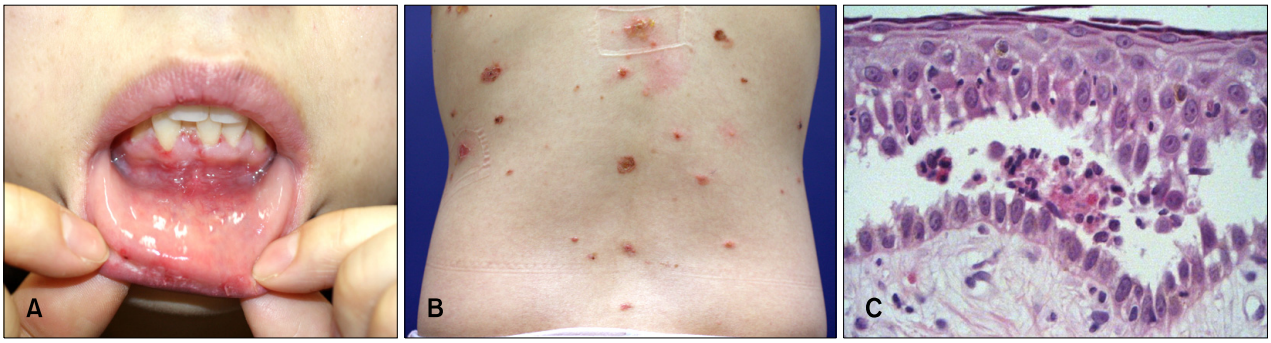

Fig. 1. (A) Multiple erosions on oral mucosa and (B) scattered crusted vesicles and erosions on the back. (C) Suprabasal blisters with eosinophils and acantholytic cells in the epidermis (H&E, ×400).

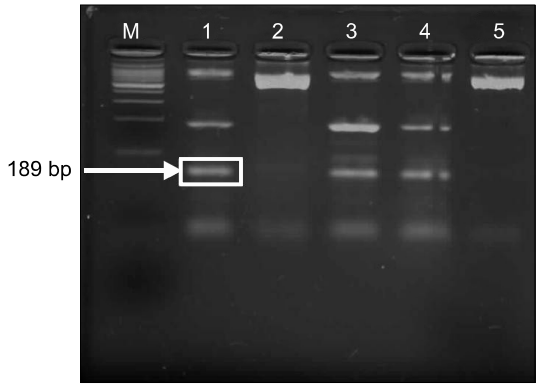

Fig. 2. Polymerase chain reaction results show a positive band on 189 bp. M: 100 bp DNA ladder, Lane 1: positive result of the patient's tissue for herpes simplex type 1 virus (189 bp), Lane 2: normal control, Lane 3: positive control, Lane 4: sensitivity test, Lane 5: negative control.

owing PCR results, antiviral agent (valacyclovir, total 4,500 mg) was also given for 5 days. Lesions started to improve after starting the medication.

Two weeks after the diagnosis the patient was transferred to the labour unit for induced labour. The baby was in a good state. After giving birth, the dose of prednisolone was increased to 25 mg/d, and mycophenolate mofetil 1.0 g/d was added. After improvement in both vesicles and general health status, the patient was discharged. During follow-up, a subtle amount of skin lesion occurred which was controlled with topical steroid, oral mycofenolate mofetil 1.0 g/d, and intermittent oral steroid at a dose of 4 mg/d.

Though it is known that pemphigus develops as a result of the interaction between endogenous (genetic) factors and exogenous factors[1], the primary stimulus for the autoimmune response of PV is still unknown.

Pregnancy may aggravate certain autoimmune diseases, such as systemic lupus erythematosus, pemphigoid gestationis, and PV[2]. The patient's vesicular eruptions developed during the period of conception and were not controlled well at that time, so we supposed her condition was related to pregnancy.

Bullous diseases that are reported to be associated with pregnancy include pemphigoid gestationis pemphigus foliaceus, and PV[2,3]. The most important diagnostic tool is histologic examination, and the two diseases mentioned above show subepidermal, subcorneal blisters. However PV, as seen in our patient, shows a suprabasal epidermal split with acantholysis and few eosinophils in the dermis, and perilesional skin shows intercelluar deposits of IgG and C3 in the epidermis on direct immunofluorescence[4]. A diagnosis of PV in this patient was made according to the histological features and clinical involvement of oral mucosa.

The possible role of viruses, especially HSV-1, has been proposed in the pathogenesis of PV. Positive viral PCR results suggest that viral factors, especially HSV and Epstein-Barr virus, are capable of inducing and/or exacerbating pemphigus in a genetically susceptible host[1].

Infectious agents can stimulate the immune response in genetically susceptible individuals or in those with immune deviating conditions such as pregnancy, leading to an increase in the production of cytokines. High levels of interferon-gamma induce the expression of human leucocyte antigen type 2 in the membranes of keratinocytes, making the structural site of PV antigen (epitope spreading) immunologically active. Chronic, recurrent viral infections can also stimulate excessive production of interleukin (IL)-4 and IL-10, which result in a shift from TH1 to TH2 response, increasing antibody production. In addition, they can also directly infect B and T lymphocytes, contributing to the production of autoreactive B lymphocytes and autoimmune antibodies[5]. When infected, keratinocytes can pass through structural changes which favor the exposure of antigens[1].

In summary, we report a case of a woman who showed

PV associated with HSV-1 in her second pregnancy, which was cured with steroids and antiviral agents.

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A Rare Case of Annular Pustular Psoriasis Associated with Pemphigus Foliaceus

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Dear Editor:

A 56-year-old Japanese woman suffered from multiple, scaly erythemas on the trunk for approximately 30 years. She had been previously diagnosed with psoriasis vulgaris in the clinic due to the clinical appearance and histological findings. She was also previously diagnosed with pemphigus foliaceus (PF) at our hospital 10 years ago, based on the clinical appearance and histological findings of a subcorneal blister, as well as on the direct immunofluorescence findings of superficial epidermal intercellular immunoglobulin (Ig) G deposition. During her clinical course, serum anti- desmoglein (Dsg)-1 IgG antibody levels were elevated to 99 index points in 2010, whereas anti-Dsg-3 antibody levels remained at <5 index points. Recently, she was treated for PF by administration of oral betamethasone (0.5 mg/d) and cyclosporine (100 mg/d). She was admitted to our hospital because the annular erythemas with pustular margins on her trunk were exacerbated and accompanied by high fever (Fig. 1). No lesions resembling PF were seen. Laboratory findings were as follows (abnormal values are underlined): white blood cell count, $21.3\times10^3/\mu l$ (neutrophils: 72%); C-reactive protein: 16.4 mg/L. Anti-Dsg1 and anti-Dsg3 antibodies were within the normal range. Because of the clinical

Received March 13, 2013, Revised April 11, 2013, Accepted for publication April 19, 2013
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