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Anti-Neutrophil Cytoplasmic Antibody-Associated Vasculitis Secondary to Human Immunodeficiency Virus Infection: A Case Report

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Dear Editor:

Vasculitides are multifactorial and often autoimmune conditions characterized by necrosis of the blood vessels induced by infiltrated leukocytes and inflammation, causing tissue injury. Various viral infections including hepatitis B virus, hepatitis C virus, cytomegalovirus, Epstein–Barr virus, and human immunodeficiency virus (HIV) are considered common causes inducing secondary vasculitides[1].

A 29-year-old male presented with erythematous patches and papules with erosions on his chest and lower legs (Fig. 1A~C). The patient was recently diagnosed with inflammatory bowel disease. Despite treatment with 5-aminosalicylic acid, the patient had recurrent mucus and/or bloody stool, with a weight loss of 5 kg during the recent 3 months. Furthermore, skin eruptions progressed into multiple atrophic whitish scar-like patches (Fig. 1D) with focal reticulated livedo reticularis-like lesions and multiple erythematous nodules evolving into necrosis, suggestive of vasculitis (Fig. 1E, F). A biopsy from the lower leg showed typical leukocytoclastic vasculitis with eosinophils involving the upper dermal blood vessels without any granulomatous lesions (Fig. 2). Direct immunofluorescence was negative. Laboratory tests showed mild leukopenia (2,800/μl), peripheral eosinophilia (14.4% of white blood cells), and thrombocytopenia (88,000/μl) with elevated erythrocyte sedimentation rate (41 mm/h). Myeloperoxidase anti-neutrophil cytoplasmic antibody (ANCA), anti-cardiolipin immunoglobulin M, and lupus anticoagulant were also detected. Under the

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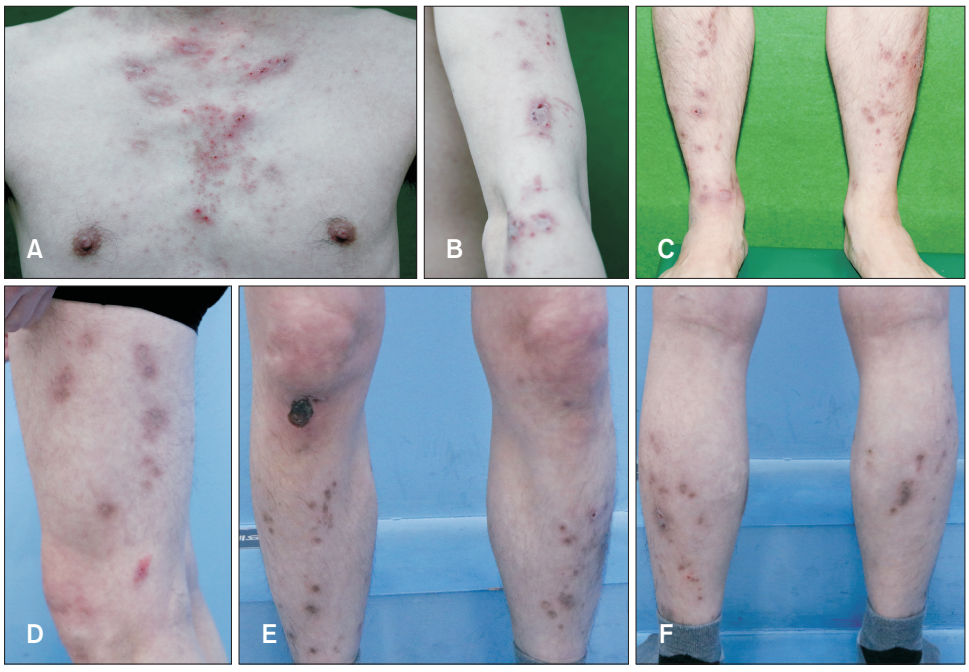

Fig. 1. Clinical presentation of anti-neutrophil cytoplasmic antibody-associated vasculitis secondary to human immunodeficiency virus infection. (A~C) Initial manifestation of skin lesions with ulcers, excoriation, erythematous patches on the chest, arm, and legs and (D~F) subsequent evolution into necrotic vasculitis-like lesions. We received the patient's consent form for publishing all photographic materials.

provisional diagnosis of P-ANCA-associated systemic vasculitis involving the skin and gastrointestinal tract, a systemic workup was performed to evaluate organs frequently involved. Thorough exams on airway and renal system, gastroenterological evaluation including sigmoidoscopy, and hematological workup for cytopenia did not show evidence of systemic involvement by primary ANCA vasculitis. However, additional workup for leukopenia revealed positivity for anti-HIV I/II antibodies and HIV antigen; HIV-RNA quantitation test revealed titer of 1.67×10^6 copies/ml. Phenotyping on blood lymphocytes showed abnormal CD4/CD8 ratio of 0.37 (CD4: 23.5% [161/μl], CD8: 63.7 [438/μl]). Thus, a combined anti-retroviral regimen including bictegravir, emtricitabine, and tenofovir was initiated. The patient reported improvement in skin lesion after treatment, indicating alleviation of vasculitis. Considering HIV infection and atypical pattern, the patient was finally diagnosed with ANCA-associated vasculitis secondary to HIV infection, which corresponds to vasculitis associated with probable etiology based on the revised Chapel Hill Consensus Conference nomenclature[2].

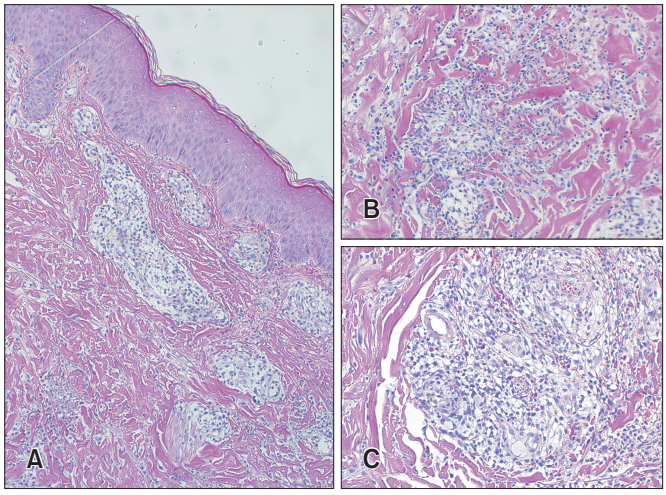

Fig. 2. Histopathological presentation of anti-neutrophil cytoplasmic antibody-associated vasculitis secondary to human immunodeficiency virus infection. (A) A biopsy sample from the lower leg showing dense perivascular inflammatory cellular infiltration (H&E, ×100). (B) A Typical leukocytoclastic vasculitis: leukocytoclasis with nuclear dust and fibrinoid change of vessels (H&E, ×200). (C) Eosinophil infiltration with red blood cell extravasation. No sign of granulomatous lesions (H&E, ×200).

HIV infection may cause diverse cutaneous eruptions. However, it is often challenging to suspect HIV infection when a patient presents rare skin manifestations such as ANCA-associated vasculitis, due to its low incidence (around 1%). In addition, ANCAs are detected in 20%~83% of HIV-infected cases; the detection of ANCAs is usually an epiphenomenon of HIV infection[3]. It is probably attributed to dysregulation of immune response, which results in expansion of B cells and CD8 T cells, increase in immune complex, and polyclonal hypergammaglobulinemia[4]. Interaction of supe-

rantigens, adhesion molecules, cytokines, growth factors, and immune complexes is hypothesized to be a causative mechanism[5]. Moreover, acquired immunodeficiency by HIV infection results in opportunistic infections that trigger autoimmunity through molecular mimicry[5].

Collectively, this case highlights that HIV infection should be suspected in cases of ANCA-associated cutaneous vasculitis with atypical systemic manifestations.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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