http://dx.doi.org/10.5021/ad.2015.27.3.347

A Case of Psoriasis Accompanied by Systemic Lupus Erythematosus

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Dear Editor:

The pathologic mechanisms of systemic lupus erythematosus (SLE) and psoriasis are different: psoriasis is caused by systemic inflammatory reaction from mainly Th1 cell activation, whereas SLE is caused by Th2 cell-related abnormalities. Cases of generalized psoriasis caused by the anti-CD20 monoclonal antibody rituximab and cases of aggravation of SLE after anti-tumor necrosis factor α biologics have been reported, suggesting the immunopathologic mechanisms of these two diseases are opposite[1,2]. However, we experienced a case of psoriasis accompanied by SLE. A 32-year-old woman had erythematous scaly macules and patches on the trunk, inguinal area, and forehead starting one year ago (Fig. 1A, B). Medical history included arthralgia progressing since one year ago, starting from the left third finger and spreading to all fingers, wrists, toes, ankles, knees, and hips (Fig. 2A). Serologic findings were as follows: anti-SSA/Ro Ab(+), anti-Smith Ab(+), fluorescent antinuclear antibody(+), and speckled pattern. She also presented with a malar rash starting 3 months ago. On the basis of clinical and serologic findings, she was diagnosed with SLE. She was prescribed methotrexate 15 mg weekly, hydroxychloroquine 400 mg, prednisolone 5 mg, and nonsteroidal anti-inflammatory drugs, and the pain seemed to abate. To evaluate skin lesions, the forehead lesion was subjected to biopsy. Histopathologic findings were consistent with psoriasis (Fig. 1C). The skin lesions responded to topical vitamin D3 analogue and topical steroid. Arthritis was evaluated by radiography; the findings of the left hip, bilateral middle fingers, and right index finger were suggestive of psoriatic arthritis, indicating that psoriasis preceded SLE (Fig. 2B). Th17 cells can be associated with the pathogenesis of various autoimmune and inflammatory diseases by producing several effector molecules. Interleukin (IL)-17 is a potent proinflammatory cytokine produced by highly activated Th17 cells and is well known to play a major role in maintaining chronic inflammation in psoriasis. Actually, skin biopsies from patients with psoriasis exhibit high IL-17, IL-22, and IL-23 expressions[3]. IL-22, one of the effector molecules produced by Th17 cells, induces antimicrobial agents and β-defensins in keratinocytes and promotes epidermal hyperplasia[4]. This cytokine is also known to be essential for the immune barrier function of epithelial cells. Recent evidence indicates IL-17 also plays a role in the pathogenesis of SLE. Patients with SLE have higher serum levels of IL-17 and IL-23 than healthy controls[3,5]. Interestingly, plasma IL-17 levels are correlated with SLE disease activity[5]. In animal models, IL-17 is related not only to T cell-mediated tissue injury, but also to the production of pathogenic autoantibodies. In the presence of IL-17, SLE-derived B cells increase anti-DNA production[5]. However, the exact function of IL-17 that leads to SLE remains unknown. Besides IL-17, IL-21 produced by Th17 cells causes CD8+ T cell proliferation and induces B cell differentiation[4]. In summary, we report a case of psoriasis accompanied by SLE. We propose the Th17 cell-mediated immune pathway is common to both diseases. This case suggests the potential utility of treating SLE with Th17 cell-targeting agents.

Received November 18, 2013, Revised March 17, 2014, Accepted for publication July 30, 2014

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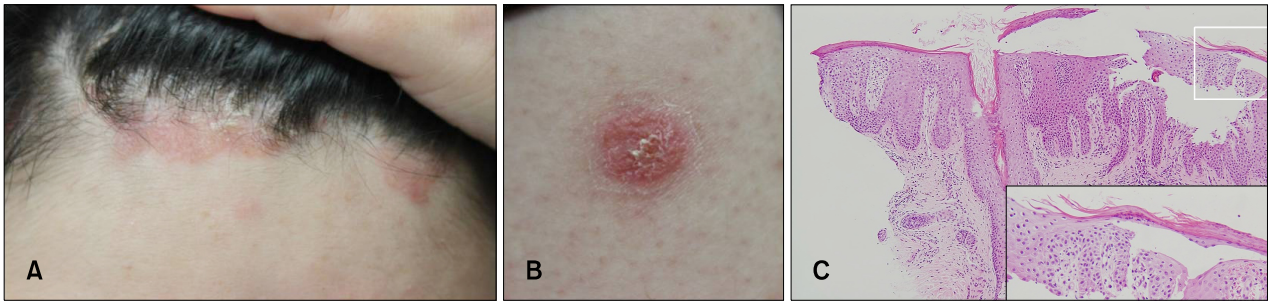

Fig. 1. (A) Erythematous scaly patches on the forehead. (B) An erythematous scaly patch on the inguinal area. (C) Regular elongation of rete ridges, parakeratosis, and mononuclear cell infiltration in the epidermis (H&E, ×40; inset: ×100).

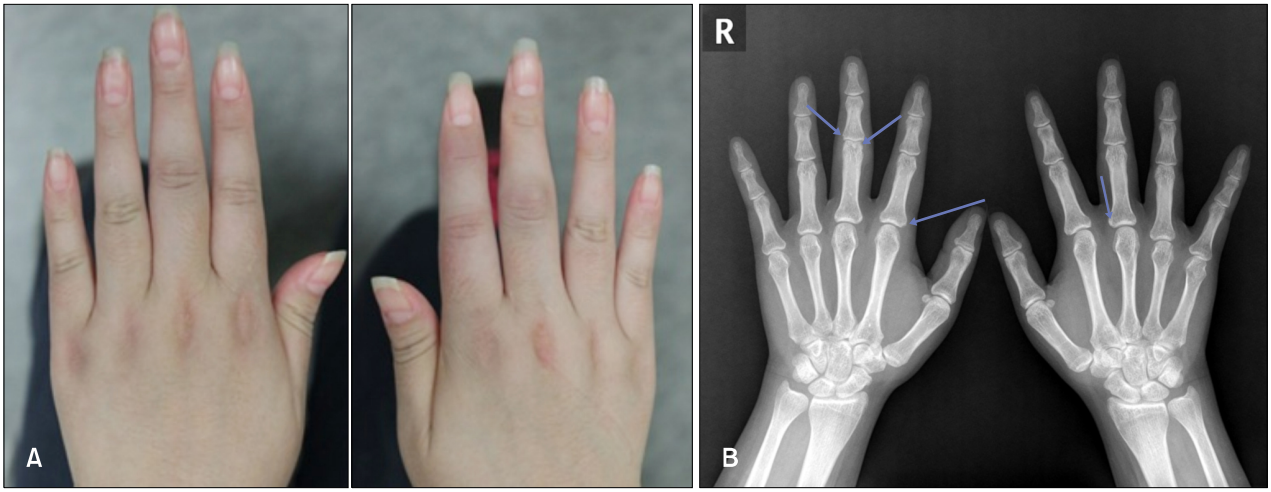

Fig. 2. (A) Swollen proximal interphalangeal joints on bilateral third fingers. (B) Radiograph showing marginal erosion at the right third proximal interphalangeal joint, small bony proliferation at the right index metacarpophalangeal joint, and suspicious erosion at the left middle proximal interphalangeal joint.

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