

CASE REPORT

Coexistence of Ankylosing Spondylitis and Systemic Lupus Erythematosus

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Coexistence of ankylosing spondylitis with connective tissue diseases is uncommon. We describe here the coexistence of ankylosing spondylitis and systemic lupus erythematosus in a 35-year-old man. He presented with a 4-year history of pain in the hip joint and lower spine, and he later developed a malar rash and discoid rash. Immunological tests revealed that antinuclear antibody and double stranded DNA antibody were positive. The human leukocyte antigen B27 antigen was also found to be positive. We propose that development of systemic lupus erythematosus in this case may have been due to low grade chronic inflammation. [*J Chin Med Assoc* 2010;73(5):260–261]

Key Words: ankylosing spondylitis, antinuclear antibody, double stranded DNA antibody, human leukocyte antigen B27, systemic lupus erythematosus

Introduction

Coexistence of ankylosing spondylitis (AS) with connective tissue diseases is very rare and only a few reports have been published.^{1–5} We describe here a rare case of coexistence of systemic lupus erythematosus (SLE) and AS.

Case Report

A 35-year-old man presented with pain in the right hip joint and lower spine that had been present for the last 4 years. In the 3 months prior to presentation, he had developed fever with a malar rash and discoid rash on the chest and ears for which he visited the dermatologist and rheumatologist. On examination, in addition to a butterfly rash on his cheek and nose, tenderness in the joints, anemia and mild hepatomegaly were also noted. Anterior lumbar flexion was limited. His laboratory examination showed

hemoglobin, 7.4 g/%; serum glutamic oxaloacetic transaminase over serum glutamic pyruvic transaminase, 43/28 IU/mL; C3, 77.6 mg/dL; and C4, 8.6 mg/dL. Urine examination showed proteinuria more than 1 g/day and granular cast. Antinuclear antibody (ratio 5.32) and dsDNA antibody (73.5 IU/mL) were strongly positive but tests for rheumatoid arthritis factor, C-reactive protein, hepatitis B surface antigen, hepatitis C virus, human immunodeficiency virus and typhoid were negative. Human leukocyte antigen (HLA) B27 was positive. An X-ray revealed class I bilateral sacroiliitis (Figure 1). The patient was diagnosed with AS based on the low back pain and class I bilateral sacroiliitis, and also SLE because he fulfilled 6 positive American College of Rheumatology criteria: malar rash; discoid rash; anemia; proteinuria; positive for anti-nuclear antibodies (ANA); and positive for dsDNA antibodies. The coexistence of SLE and AS is uncommon but is a possibility when SLE patients present with chronic inflammatory low back pain.



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Figure 1. X-ray shows inflammatory changes in the sacroiliac joint (class I bilateral sacroiliitis).

Discussion

Similar to our case, Olivieri et al reported a 42-year-old woman who was diagnosed with sacroiliitis. After 4 years, she developed full blown SLE with a malar rash, oral ulcers, alopecia and Raynaud's phenomenon.⁶ Another study from Italy also reported the coexistence of AS and undifferentiated connective tissue disease in a 45-year-old woman.⁷ Her disease course started with inflammatory low back pain and blurring of vision owing to anterior uveitis in the right eye. She also had retinal vasculitis and Tyndall's phenomenon in vitreous fluid. Four years later, she developed subcutaneous SLE on the face and neck with photosensitivity, migratory arthralgias, xerostomia and fatigue. In both of these case reports, patients were HLA B27 and ANA positive.

Recently, Perez-Garcia et al also reported a 65-year-old woman who had AS with uveitis for the last 15 years. Initially, she was given an anti-inflammatory drug and methotrexate to which she did not respond. She was then given infliximab; however, after 48 hours

of infusion, she presented with fever, myalgias, polyarthritits, morning stiffness and a positive test for ANA.⁸

We did not carry out complete HLA typing. We propose that HLA B27 antigen predisposes to certain microbial infection and later infection may trigger the development of SLE. Infection and inflammation are the most important factors in the development of autoimmune diseases.

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