Coexistence of ankylosing spondylitis and discoid lupus: A case report

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ABSTRACT

Coexistence of ankylosing spondylitis with connective tissue diseases is very rare. Here, in this study, we describe a coexistence of ankylosing spondylitis and discoid lupus erythematosus in a 35-year-old man. He presented with a 5-year history of low back pain and concurrent development of a discoid rash. Inflammatory low back pain, HLA-B27 positivity and bilateral active sacroiliitis confirmed the diagnosis of ankylosing spondylitis. Discoid lupus erythematosus was diagnosed based on a skin biopsy. There are reports of discoid lupus associated with medications, particularly with tumour necrosis factor-alpha (TNF-α) blocking drugs. However, the patient presented here had coexistence of ankylosing spondylitis and discoid lupus before starting such treatments.

Keywords: Ankylosing spondylitis; discoid lupus; systemic lupus erythematosus.

CASE REPORT

A 35-year old male patient was admitted to the outpatient clinic with low back pain who had these complaints for five years and had been diagnosed as ankylosing spondylitis two years ago. He had a history of non-steroid anti-inflammatory drugs usage that had been inadequate for the relief of the symptoms. The patient also had an erythematous plaque on the bridge of the nose for five years with a history of an increase in skin lesions in the previous year (Fig. 1). There were no peripheral arthritis, uveitis, psoriasis or inflammatory intestinal disease and also no clinical findings of systemic involvement and the family history of rheumatic diseases. In physical examination increased thoracic kyphosis and reduced cervical and lumbar range of motion were detected. Lumbar Schober was 1.5 cm, finger base distance was 25 cm, and jaw manubrium distance was 0. Sacroiliac compression tests were bilaterally pos-
itive. BASDAI score was 6. Anti-nuclear antibody and anti-dsDNA were negative. Erythrocyte sedimentation rate (ESR) was 40 mm/hr and C-reactive protein (CRP) was 2.5 mg/dl (CRP normal range 0-0.5 mg/dl). Complement tests, full blood count, liver and kidney functions, serum protein and creatinine phosphokinase levels, thyroid function tests, urine analysis and urine protein/creatinine ratio were within a normal range. Hepatitis B and C viruses, HIV and Brucella serology were negative. HLA-B27 was positive. The pulmonary radiograph was normal. On the pelvis radiograph grade 3-4 bilateral sacroiliitis were seen (Fig. 2).

A skin biopsy was performed and mononuclear inflammatory cell infiltration around the hair follicles on the dermis and increased mucin in the dermal papilla were seen (Fig. 3). After a diagnosis of ankylosing spondylitis and discoid lupus, 40 mg/2 weeks subcutaneously adalimumab, 400 mg/day hydroxychloroquine and topical tacrolimus were started. Using a high factor protective sun cream was recommended. In the follow-up, the clinical symptoms of AS were significantly improved.

DISCUSSION

Coexistence of AS and SLE is very rare, with a limited number of cases reported in the literature [3–12]. AS is frequently seen in male patients and SLE is frequently seen in female patients. Coexistence of these two diseases are predominantly reported in female patients and in the majority of the cases complaints related to AS and diagnosis of AS was before the diagnosis of SLE [3–8, 10, 12]. Our patient was a male and his complaints related to AS and DLE started at the same time. Inflammatory low back pain, HLA-B27 positivity and bilateral active sacroiliitis confirmed the diagnosis of AS based on the modified New York criteria. DLE was diagnosed according the results of a skin biopsy. In the literature, there are reports with various combinations of AS with findings of SLE such as hematological and renal involvement, malar rash, ANA, anti-dsDNA, hypocomplementemia, thrombocytopenia, and leukopenia [3–12]. In contrast, in our patient, there was only DLE and no systemic involvement was detected.

Nashel et al. [3] reported a black male patient with HLA-B27 antigen combination with DR2 and DR3 antigens affected by both AS and SLE. Combination of genetically determined markers seems to have caused an increased risk for the development of both disorders. Although HLA-B27 was positive in our patient, we could not look for DR2 or DR3 antigens. Patients with AS often use anti-TNF drugs. In patients with AS, rheumatoid arthritis (RA), and inflammatory intestinal disease who were treated with anti-TNF drugs rarely discoid lupus was reported. Stratigos et al. [13] reported
a RA patient who developed discoid lupus during infliximab therapy and the skin lesions of the patient recovered with antimalarial treatment after terminating infliximab. Unlike these reports, the findings of DLE emerged before the anti-TNF treatment in our patient. The findings were not related to the drugs and the patient had no systemic symptoms of lupus.

SLE may develop in 5%-10% of the DLE patients [14]. If DLE lesions are in a disseminated form with trunk involvement, there is an increased risk of systemic involvement [2]. Also, there is a risk of the development of squamous cell carcinoma in discoid lupus lesions [15]. Great care should be taken in this respect. Skin biopsy was reported as DLE in our patient, and no findings of malignancy were determined. Symptoms of SLE have not been detected during the follow-up of the patient.

In conclusion, here, we reported a male patient with coexistence of AS and DLE. It should be kept in mind that although they have different etiopathogenesis, genetic and clinical factors, coexistence of these diseases could be very rarely seen.

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