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Sarcoidosis Developing after Stopping Oral Prednisolone for Systemic Lupus Erythematosus

Sir.

Although neither the pathogenesis of sarcoidosis nor that of systemic lupus erythematosus (SLE) has been elucidated, both are thought to result from abnormalities of the immune system. However, association of sarcoidosis and SLE is quite rare. We report here a patient who developed sarcoidosis after oral prednisolone therapy for SLE was stopped.

CASE REPORT

A 60-year-old Japanese woman was referred to our hospital in December 1999 for examination of a subcutaneous nodule on her left forearm that had appeared 1 month before. She had a butterfly rash, photosensitivity, leukopenia, antinuclear antibodies and anti-double-stranded DNA. She had been followed since the diagnosis of SLE in 1994, and oral prednisolone therapy for SLE had suddenly been stopped 2 months prior to that. A subcutaneous tumour subsequently appeared on her left forearm. She also noticed edematous erythema on both eyelids. Physical examination revealed the existence of multiple subcutaneous nodules on the extremities and buttocks.

Laboratory examination showed the following abnormalities: antinuclear antibody titre $(320\times)$ with a homogeneous and speckled pattern, positive antibodies to double- stranded DNA and SSA antigen, and reduced white blood cell count and hemoglobin. Antibodies to RNP, Sm and SSB antigens, LE test, rheumatoid factor, serum angiotensin-converting enzyme and serological tests for syphilis were negative.

A skin biopsy from the nodular lesion on her left forearm showed noncaseating epithelioid cell granulomas throughout the dermis, and a diagnosis of sarcoidosis was made. A biopsy specimen taken from the edematous erythema on her right eyelid revealed hydropic degeneration of basal cells and a dense superficial perivascular mononuclear cell infiltrate around superficial blood vessels, indicating SLE

Chest X-ray and computed tomography demonstrated bilateral

hilar lymphadenopathy. Ophthalmic examination revealed endogenous uveitis.

The patient was diagnosed as having sarcoidosis with underlying SLE. She was treated with oral prednisolone (30 mg/day). The edematous erythema on the eyelids completely subsided in 1 week, and the subcutaneous nodules on the extremities and buttocks gradually became soft and small.

DISCUSSION

Teilum (1) first suggested that sarcoidosis and SLE might be involved in the same underlying immune mechanisms. David et al. (2) reported a patient with sarcoidosis who developed rapidly progressive SLE after steroid therapy was withdrawn. In our case, sarcoidosis occurred after cessation of oral prednisolone, and SLE was exacerbated simultaneously. Such cases suggest that once immunosuppression has occurred, steroid reduction may rapidly lead to immune dysfunction, resulting not only in recurrence of the disease but also in development of an allied disease.

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