Follicular Lymphomatoid Papulosis and Multiple Myeloma

Sir,

We report the first case of lymphomatoid papulosis associated with multiple myeloma. Although such an association may be simply coincidental, it is however intriguing. A possible mechanism explaining the coexistence of T- and B-cell proliferation in the same patient is suggested.

CASE REPORT

A 48-year-old man presented with a history of a self-healing, itching papular eruption, which had been recurrent for 6 years. Diagnosis remained pending and no treatment was given. Fatigue and pain in the bones had started 4 years earlier. At that time, the erythrocyte sedimentation rate was 80 mm/h, with normochromic normocytic anaemia. Serum protein electrophoresis showed monoclonal gammopathy of IgA-k (1.70 g/dl). A quantitative immunoglobulin study showed an elevated IgA of 6.50 mg/ml (normal 0.3 to 3.0 mg/ml) and normal IgM and IgG levels. Radiography revealed osteolytic areas of the skull, pelvis and femur. A bone marrow biopsy specimen showed a marked increase (30%) of plasma cells. These had atypical features. Renal and liver function tests were normal, and Bence Jones proteins were not detected. The diagnosis of IgA-k myeloma was made. Since January 1993, the patient has been treated with 3 MU interferon-alpha on alternate days.

On examination some erythematous, mostly follicular papules, up to 0.5 cm in diameter, were noted on his trunk and limbs. Most of them had central crusts, and several depressed scars were present. Histologically, the epidermis was normal. In the dermis there was a dense inflammatory infiltrate with an evident perifollicular distribution. The infiltrate consisted of lymphocytes, histiocytes, neutrophils, eosinophils and large, atypical mononuclear cells with hyperchromatic and convoluted nuclei. Some mitoses were seen. The follicular epithelium was hyperplastic and infiltrated, without cysts or mucin deposits. Microorganisms were neither seen nor cultivated. The infiltrate was predominantly composed of T-cells with a prominent helper/inducer phenotype (UCHL-1 and CD-4 positive). Only a few scattered cells were positive for Ki-1 (CD 30) and the B-cell marker L-26.

DISCUSSION

Lymphomatoid papulosis is a chronic self-healing eruption, in which recurring crops of necrotic papules display a cytologically malignant infiltrate (1) of activated T-helper/inducer lymphocytes. A significant amount of the infiltrating cells express the Ki-1 (CD 30) antigen (2).

An association with malignant lymphoma is reported in 10–20% of patients, but whether this represents an independent association or a transformation of one lymphoid disorder into another is unclear.

Multiple myeloma has never been reported in patients with lymphomatoid papulosis. The coexistence of T- and B-cell proliferation in the same patient may, of course, be simply coincidental. We have ruled out any influence of drugs, as our patient was not given any therapy for his lymphomatoid papulosis. A causal mechanism, therefore, may be envisaged. As lymphomatoid papulosis involves T-helper cells, a high T-helper/T-suppressor ratio may also lead to a sustained stimulation of B-cells and plasmocytes. This would cause an abnormal or mutant B-cell clone to escape the normal regulating mechanisms, and eventually to produce a second disease.

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