Rheumatoid arthritis and B cell lymphoma with pathological changes of reactive histiocytosis

M Shimada, M Kojima, G Tani, S Komatsu, M Nara

Abstract
A 67 year old woman with rheumatoid arthritis was admitted to hospital in acute renal failure. Her clinical features included increasing dyspnoea and oedema, and a computed tomogram of the abdomen showed a large mass in the retroperitoneum. Twenty six days later, she died, and a post mortem examination was carried out. The histological changes of the mass indicated B cell lymphoma of diffuse large cell type, with a reactive proliferation of erythrophagocytosing histiocytes. Immunocytochemical studies showed that the histiocytes were positive for CD68 and lysozyme, but negative for S-100 protein. Such neoplastic B cell proliferation accompanied by activation of benign looking histiocytes with erythrophagocytosis is very rare.

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Case report
A 67 year old woman who had had rheumatoid arthritis since she was 40 years old was admitted to hospital in acute renal failure. Blood chemical findings showed that the urea nitrogen concentration was 145 mg/100 ml, and the creatinine concentration 9-6 mg/100 ml. An x ray film of the chest showed pulmonary oedema. Her dyspnoea worsened and haemodialysis was begun. Computed tomography of the abdomen disclosed a large mass in the retroperitoneum, lymphadenopathy, and bilateral hydronephrosis which suggested that the tumour had spread into the ureters. An ureteric catheter was inserted into the right renal pelvis and diuresis carried out. The patient developed extreme respiratory distress, however, and 26 days after admission she died.

A post mortem examination was carried out, and the histopathological findings of the mass indicated malignant lymphoma of the diffuse large cell type, with reactive proliferation of erythrophagocytosing histiocytes. Immunocytochemical studies showed that the lymphoma cells expressed the B linkage markers CD20 and CDw75, but not the T-linkage markers CD43 and CD45RO. The histiocytes were also positive for CD68 and lysozyme, but negative for S-100 protein.

Discussion
Rheumatoid arthritis is sometimes associated with malignant lymphoma, and most of the lesions are considered to be B cell lymphomas. On the other hand, most of the malignant lymphomas associated with haemophagocytic histiocytosis have the T cell phenotype. Kadin et al1 have described haemophagocytic T cell lymphomas, suggesting that haemophagocytic T cell lymphoma and malignant histiocytosis may be closely related. Malignant histiocytosis is a syndrome characterised by a systemic, neoplastic proliferation of histologically recognisable histiocytes and their precursors.2 The clinical features of our case were not similar to those of malignant histiocytosis, but the pathological changes did show reactive histiocytosis with haemophagocytosis, which was similar to malignant histiocytosis. Jaffe et al3 reported T cell lymphoma associated with benign histiocytes, and they also suggested that the mechanism of this syndrome was the production of lymphokines by neoplastic T lymphocytes. Our case was probably B cell lymphoma, and such B cell lymphoma accompanied by activation of benign looking histiocytes with erythrophagocytosis is very rare, while that associated with rheumatoid arthritis has not been described before. The pathogenesis of the histiocytic proliferation in our case remains unclear. A viral infection such as virus-associated haemophagocytic syndrome is a possibility, although no viral antibody was detected.

Rheumatoid arthritis and B cell lymphoma with pathological changes of reactive histiocytosis.

M Shimada, M Kojima, G Tani, S Komatsumoto and M Nara

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