

Predominance of CD4+ T cells and Th2 cytokines in the pericardial fluid of a dermatomyositis patient with cardiac tamponade

Sirs,

Pericardial disease occurs in 10% of patients with idiopathic inflammatory myopathy (IIM) (1). Pericardial involvement, however, rarely evolves into cardiac tamponade (2-4). To study the immunological abnormalities of pericardial disease in IIM, the lymphocyte subpopulations and cytokines were examined in peripheral blood (PB) and pericardial fluid (PF) of a dermatomyositis (DM) patient who presented cardiac tamponade.

A 59-year-old woman was diagnosed with DM in 1997 when she developed a subacute onset of proximal muscle weakness characterized by difficulty in rising from a bed or chair, climbing, walking, lifting and combing her hair. Initially she presented heliotrope rash and Gottron's papules. She had a 5 to 10-fold elevation of serum creatine kinase (CK) and the electromyogram revealed a short duration and low amplitude polyphasic motor unit action potentials. She was treated with prednisone 60 mg po daily for 6 weeks followed by a gradual tapering over the next 6 months to 10 mg daily. She achieved normal muscle strength.

In December 2002 she was hospitalized due to chest pain, nausea and dizziness. She presented jugular venous distention and decreased heart sounds. A pulmonary examination was negative. She had no muscle weakness in the upper and lower extremities and no DM skin lesions. The electrocardiogram revealed a low voltage. Chest X-rays showed massive cardiomegaly. A large pericardial effusion with evidence of early hemodynamic compromise of the right atrium was observed on echocardiography. Cardiac catheterization showed non-obstructive coronary artery disease and diastolic dysfunction, but a normal ejection fraction at 65%.

A laboratory work-up showed a white blood cell count of 17,100/mm³ (85% neutrophils, 10% lymphocytes), hemoglobin 13.8 g/dl and platelet count 356,000/mm³. Serum BUN, creatinine, liver function tests and CK were normal. She had a positive ANA test at 1:640 with a homogeneous pattern. However, she did not present any clinical or other serologic findings of systemic lupus erythematosus (SLE).

A pericardiocentesis was performed and yielded 850 ml of yellowish fluid. Examination of PF showed a white blood cell count of 6,100/mm³ (polymorphonuclear cells 97%). Protein was 4.9 g/dl and glu-

Table 1. Lymphocyte populations and cytokines concentrations in peripheral blood and pericardial fluid.

Parameter	Peripheral blood	Pericardial fluid
Lymphocyte population*		
Total Tcells, %	86	41
CD4+ Tcells, %	48	34
CD8+ Tcells, %	38	1
B cells, %	8	20
NK cells, %	2	6
Thelper/suppressor ratio	1.2	30.1
Cytokine (pg/ml)†		
IL-2	3.7	3.5
IL-4	3.9	5.1
IL-6	39.3	4549.9
IL-10	4.6	24.4
TNF-	2.1	1.3
IFN-	1.5	5.4

*Determined by four-color flow cytometry using FACSCalibur (Becton Dickinson, San José, CA)

†Measured by cytometric bead array. Assay sensitivity (pg/ml) for IL-2 = 2.6; IL-4 = 2.6; IL-6 = 2.4; IL-10 = 2.8; TNF- = 2.8; IFN- = 7.1; negative control = 0. Values below the assay sensitivity were extrapolated. Determined using FACSCalibur (Becton Dickinson, San José, CA.).

cose 79 mg/dl. PF cultures for bacteria were negative. No acid fast bacilli were found. Cytological examination was negative for malignant cells. She was treated with methylprednisolone 60 mg IV q 12 hrs, followed by tapering. She was switched to prednisone 40 mg po qd. The pericardial effusion resolved and did not recur after 3 months of follow-up.

Before IV corticosteroid treatment, PF and PB were obtained simultaneously for immunological analysis (Table 1). Among the lymphocyte populations, the percentage of B cells and NK cells was higher in PF than in PB. Both in the PB and PF, CD4+ T cells predominated but the ratio was much higher in PF (30.1 vs. 1.2). Only 1% of CD8+ T cells were detected in PF. There was marked elevation of the IL-6 concentration in the PF when compared to plasma. IL-10 was also higher in PF. IL-2, IL-4, and TNF- and INF- were either low or undetected in the PF and plasma.

The augmented concentration of IL-6 in PF presented here is noteworthy. It seems that IL6 has an important role in the pathogenesis of pericardial involvement of autoimmune connective tissue diseases. The marked elevation of PF IL-6 found here has been reported in the PF of systemic lupus erythematosus and rheumatoid arthritis patients complicated with pericardial tamponade (5, 6).

In summary, different patterns of lymphocyte populations and cytokines were found

in PB and PF from a patient with DM, with CD4+ T cells and Th2 cytokines predominating in PF. These disparities suggest a localized inflammatory process in the pericardial tissue. Further studies are required to determine the relevance of these immunological abnormalities in IMM.

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