Cardiac Tamponade as an Initial Manifestation of Systemic Lupus Erythematosus - single case report -

We describe a case of pericardial tamponade as an initial manifestation of systemic lupus erythematosus (SLE). Although pericarditis or pericardial effusion is the common cardiac complication of SLE, tamponade is unusual. Treatment consists of pericardiocentesis, administration of high dose glucocorticoid and antimalarial drug. ***(JKMS 1997; 12: 75~7)***

Key Words : *Systemic lupus erythematosus, Pericardial tamponade*

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Received : October 2, 1996
Accepted : November 18, 1996

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INTRODUCTION

Systemic lupus erythematosus (SLE) is an multi-organ disease. Cardiac involvement is rare as an initial manifestation of the disease (1, 2). In late onset SLE (>50 years of age), pericardial involvement is a common presenting manifestation, with a 27% incidence (2, 3). But cardiac tamponade is extremely rare. Furthermore, tamponade as an initial manifestation of the illness is distinctly unusual. We describe a Korean woman whose initial manifestation of SLE was cardiac tamponade.

CASE REPORT

A 44-year-old Korean woman with no history of past medical illness was admitted to Hanyang University KuRi Hospital with a 10 day-history of left shoulder pain, shortness of breath and chest tightness. She denied oral ulcer, skin rash, photosensitivity or alopecia. She complained knee joint pain not arthritis. Examination revealed an acutely ill woman. Her blood pressure 90/65 mmHg, pulse rate 95/min, body temperature 38℃ and respiratory rate was 24/min. Cardiac examination showed notably decreased heart sounds. Breath sounds were diminished on left side at the base. There was 2 fingerbreadth, non-tender, firm hepatic enlargement. Electrocardiogram revealed low voltage, sinus tachycardia and complete right bundle-branch-block. Admission laboratory tests were as follows ; hemoglobin 8.3 g/dl, white blood cell count (WBC) 3500/mm^3, platelet count 206,000/mm^3, serum creatinine 0.4 mg/dl, serum protein 7.6g/dl, albumin 3.0g/dl, ALT 75 IU, AST 55 IU, LDH 267 U, antinuclear antibodies (ANA) >1:2560 positive with speckled cytoplasmic pattern (performed on IT-I cells), rheumatoid factor (-), anti-DNA antibody 8.9 U/ml (radioimmunoassay, normal : <7 U/ml), C4 30 mg/dl (normal 15-45), C3 129mg/dl (normal 70-150). Urinalysis was normal. Anti-Sm antibody (-), anti-nRNP antibody (+), anti-Ro antibody (-). Chest radiography revealed a markedly enlarged cardiac silhouette (Fig. 1). A 2-D echocardiogram demonstrated a large pericardial effusion and diastolic collapse of the right ventricle (Fig. 2). Valvular function and left ventricular ejection fraction was normal.

One day after admission, the patient suffered from progressive shortness of breath and markedly distended neck vein and pulsus paradoxus were noted. An immediate pericardiocentesis was performed. Intrapericardial pressure was measured 15mmHg and 1000ml of yellowish and clear fluid was aspirated. Pericardial fluid analysis revealed RBC count 26,000/mm^3, WBC count 120/mm^3 (36% neutrophils and 64% lymphocyte), protein 5.4g/dl, LDH 604 Unit, C_3 78.1mg/dl, C_4 13.3 mg/dl and ANA >1:2560 positive with speckled cytoplasmic pattern. Bacterial and mycobacterial smears and cultures were negative. Polymerase chain reaction to *Mycobacterium tuberculosis* was negative. Cytologic examination revealed no malignant cells. Prednisolone 30mg/day was administered and she improved rapidly. The diagnosis of SLE was established based on the presence of serositis, leukopenia and the presence of ANA, antibodies to dsDNA suffering 4 of 11 ACR criteria. Follow-up echocardiogram and the chest X-ray

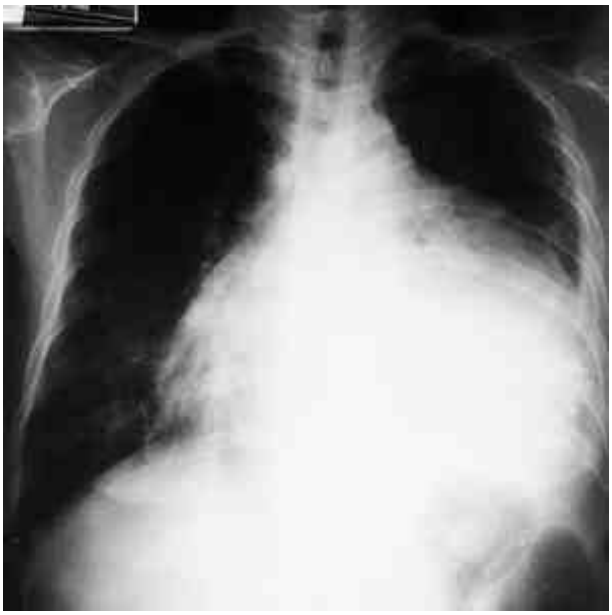

Fig. 1. Chest roentgenogram demonstrating marked cardiomegaly without lung infiltration.

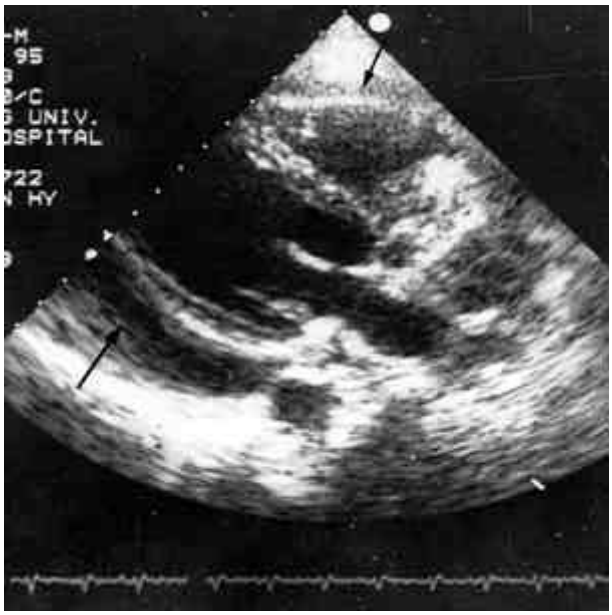

Fig. 2. Echocardiogram, long-axis parasternal view demonstrating massive pericardial effusion. The arrows point to pericardial effusion.

revealed very small pericardial effusion. Two weeks after admission, she was discharged and prednisolone was gradually tapered to 10mg/day over five months. Five months after discharge the patient has continued to do well with anti-malarial drug only.

DISCUSSION

The cardiac manifestations of SLE include pericarditis, myocarditis, endocarditis and conduction system abnormalities (4~6). Although pericarditis is common, cardiac tamponade is a distinctly uncommon. As an initial manifestation of SLE, cardiac tamponade is a strikingly unusual event. Cardiac tamponade is rare but may occur in up to 6% of SLE patients who have pericarditis (7). The low incidence of tamponade despite the high frequency of pericarditis in SLE may in part be due to the widespread use of steroid and NSAIDs which are quite effective in reducing pericardial inflammation (6). Cardiac tamponade develops when a critical amount of fluid accumulates in the pericardium resulting in diminished blood flow to the ventricles (2, 3). The clinical manifestations of tamponade are caused by diminished cardiac output and systemic venous congestion. The diagnosis is established by echocardiography (2). Typical symptoms and signs include dyspnea, orthopnea, chest pain, pulsus paradoxicus and hypotension. Pericardiocentesis and steroids are the initial treatments of choice so pericardiocentesis was performed in our patient. Pericardial fluid volumes of 1000ml were removed. Pericardial fluid typically showed leukocytosis with neutrophilia. Depressed complement levels in the pericardial fluid suggest the presence of local complement consumption. The presence of the dsDNA antibody in serum confirmd the diagnosis of SLE and low complement levels in the blood and pericardial fluid further support this diagnosis (8, 9).

In summary, this patient showed cardiac tamponade as an unusual initial manifestation of SLE and accurate and timely diagnosis of this rare manifestation of SLE may be life saving.

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