Systemic lupus erythematous: An unusual cause of cardiac tamponade in a young man

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Abstract Although pericarditis and pericardial effusion are common cardiac complications of systemic lupus erythematosus (SLE), cardiac tamponade is a very rare initial manifestation of this disease. We describe a case of a young male patient in whom cardiac tamponade secondary to a loculated pericardial effusion was the presenting symptom of SLE.

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Introduction

Systemic lupus erythematous (SLE) is a connective tissue disorder which often involves the heart, mostly the pericardium usually manifests as diffuse pericardial effusion. It is mostly of a mild degree and more common in elderly. Cardiac involvement as the initial presentation of SLE has been reported in a few patients and cardiac tamponade of the disease is rare as the first manifestation. SLE related cardiac tamponade has generally a benign evolution with proper treatment. Although pericardiocentesis associated with anti-inflammatory drugs is the treatment of choice, surgery is indicated in some cases. In this report, we describe a young male in whom cardiac tamponade secondary to localized pericardial effusion was surgically treated and the diagnosis of SLE was established.
Case report

A 26-year-old male patient was admitted to another hospital with a recent history of dyspnea, left shoulder pain, and lower leg edema. A CT scan was performed and mild pericardial effusion was found. His hemodynamic status deteriorated and he was transferred to our hospital. At arrival he had mental confusion. His past medical history was unremarkable except for smoking. The physical examination showed an arterial pressure of 80/50 mmHg, a pulse rate of 125 beats/min, a respiratory rate of 30/min and a body temperature of 38°C with distended neck veins, mild hepatomegaly, and decreased heart sounds. ECG revealed sinus tachycardia and low voltage. Chest X-ray showed cardiomegaly with clear lungs. Laboratory tests were normal except for moderate anemia. A 2-D transthoracic echocardiogram (TTE) demonstrated near total collapse of the right atrium (RA) and the right ventricle (RV) due to compression by an adjacent cystic structure covered with a thick membrane (Fig. 1). When we examined the CT scan in the light of these echocardiographic findings, we noticed that this large cystic structure was misinterpreted as RV (Fig. 2). Since the patient was unstable, emergent surgery was planned. Surgical exploration via a median sternotomy showed a loculated pericardial effusion compressing the RV and the RA antero-laterally with a thickened pericardium. The surgeon also noted the presence of laciniae fibrosae which caused a septation and compartmentation of the pericardium. After drainage of 800 cc hemorrhagic fluid, partial pericardiectomy was performed. Analysis of the fluid revealed WBC count of 110/mm³ (predominantly lymphocytes), protein level of 5.2 g/dl, LDH level of 628 U/L, and ANA > 1/2560 positive with speckled cytoplasmic pattern. Pathologic examination of the resected pericardium revealed prominent infiltration with lymphocytes and plasma cells. Bacterial smears, cultures and polymerase chain reaction to Mycobacterium tuberculosis were all negative with no malignant cell on cytologic examination. Blood analysis with radioimmune assay was positive for anti-dsDNA antibodies. Because the patient met the criteria adopted by American Rheumatism Association,5 the diagnosis of SLE was established and steroid therapy was begun. Control TTE demonstrated that the RV and the RA were free of compression. The patient had an uneventful postoperative course and was discharged on 15th postoperative day.

Discussion

The cardiac manifestation of SLE includes pericarditis, myocarditis, endocarditis and conduction system abnormalities. Although pericarditis is common, cardiac tamponade, especially as an
initial form of presentation, is unusual. Pericardial involvement usually manifests itself as diffuse pericardial effusion but the presence of fibrous bands can cause loculated effusion. Although differential diagnosis of SLE related pericardial effusion includes idiopathic, viral, bacterial, tuberculous, uremic, postmyocardial infarction, neoplastic, and traumatic pericardial effusions, in this case, hydatid cyst had been considered first because of cystic appearance of effusion and local reasons. Since SLE is more prevalent in women and its cardiac manifestation usually occurs in later years of life, cardiac tamponade caused by SLE in a young male is another striking finding in this patient.

Emergent surgery was needed in this patient before identification of the definite etiology. Because the surgeon had noticed an increased pericardial thickness during the surgery, he performed not only drainage of the fluid but also pericardiectomy at the same session. We think that this approach would be helpful to prevent future symptoms of constrictive pericarditis in addition to saving life. In the case of diffuse pericardial effusion, pericardiocentesis associated with steroid therapy is the treatment of choice.

In conclusion, SLE related loculated pericardial effusion should be kept in mind as a rare cause of cardiac tamponade even in patients without previous SLE diagnosis.

References