

A case of cardiac tamponade caused by tuberculous pericarditis

Tüberküloz perikardite bağlı kardiyak tamponad

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A 71-year-old woman was admitted with a diagnosis of cardiac tamponade. Emergency transthoracic echocardiography showed a large amount of pericardial effusion compressing the whole heart. Pericardiocentesis was performed immediately and nearly 1 liter of hemorrhagic fluid was aspirated. Pathological result of the pericardiocentesis material was benign, acid-resistant bacteria were not found in the pericardial fluid, and bacteria cultures were negative. The only parameter suggesting tuberculous pericarditis was adenosine deaminase activity in the pericardial fluid, which was measured as 76 U/l. With anti-tuberculosis therapy for six months, the patient showed complete improvement; no signs of deterioration were observed and echocardiographic findings were normal.

Key words: Adenosine deaminase; cardiac tamponade/etiology; echocardiography; pericardial effusion/etiology; pericarditis, tuberculous/diagnosis/therapy.

Tuberculosis is believed to be one of the main causes of pericarditis in developing countries. Its incidence is increasing with the overspreading of HIV disease.^[1] Tuberculosis is diagnosed in only 4% of acute pericarditis cases. Pericarditis may be seen in 1-2% of pulmonary tuberculosis cases. It may originate from the tracheobronchial tree, regional lymph nodes, and bone structures; however, hematogenous spread may also be seen.^[2] Cardiac tamponade is a common complication of tuberculous pericarditis. Tuberculosis is diagnosed in 60% of patients with cardiac tamponade.^[3] It is difficult to isolate Mycobacterium species from pericardial fluid samples and only one-third of them are diagnostic.^[4] Measurement of adenosine deaminase (ADA), a white blood cell-mediated enzyme, in the pericardial fluid increases the diagnostic accuracy. In a prospective study, it was reported that ADA levels higher than 40 U/l had a sensitivity

Yetmiş bir yaşında bir kadın hasta kardiyak tamponad tanısıyla kliniğimize yatırıldı. Acil olarak yapılan transthorasik ekokardiyografide, tüm kalpte belirgin basıya neden olan ileri miktarda perikard efüzyonu saptandı. Yapılan perikardiyosentezle 1 litreye yakın hemorajik sıvı aspire edildi. Perikardiyosentez materyalinin patolojik incelemesi benign bulundu, perikard sıvısında aside dirençli bakteri görülmedi, bakteri kültürü negatif sonuçlandı. Hastada tüberküloz perikardisine işaret eden tek gösterge, perikard sıvısında 76 U/l düzeyinde ölçülen adenozin deaminaz aktivitesiydi. Altı ay süreyle antitüberküloz tedavisi gören hastanın genel durumunda tam düzelme görüldü; diğer bulgularla beraber ekokardiyografi bulguları normal seyretti.

Anahtar sözcükler: Adenozin deaminaz; kardiyak tamponad/etyoloji; ekokardiyografi; perikard efüzyonu/etyoloji; perikardit, tüberküloz/tanı/tedavi.

of 93%, and specificity of 97% for the diagnosis of tuberculosis.^[5] Therefore, in case of any suspicion, pericardial fluid levels of ADA must be determined.

CASE REPORT

A 71-year-old woman was referred to our clinic with a diagnosis of cardiac tamponade. Her general status was serious with the following parameters: body temperature 36.5 °C, apex beat 130/min and arrhythmic, arterial blood pressure 70/50 mmHg, and respiration rate 24/min. Physical examination showed cyanotic lips, jugular venous distention, bilateral decreased respiratory sounds, extensively rough rales, muffled heart sounds, obese abdomen, and 1+ pretibial edema. Electrocardiography showed atrial fibrillation with voltage loss in all leads. Emergency transthoracic echocardiography was performed. A large amount of pericardial effusion was observed, surrounding the

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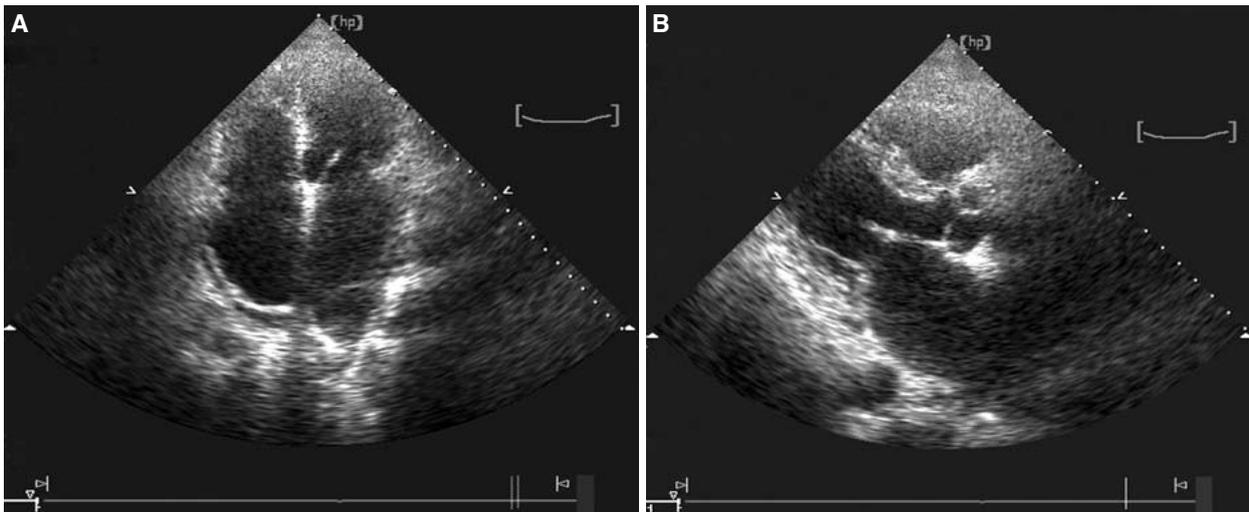


Figure 1. (A) Parasternal long-axis and (B) apical four-chamber views showing minimal pericardial effusion.

heart, reaching 4-5 cm thickness in some parts, and causing a marked compression to the whole heart. Pericardiocentesis was performed immediately and nearly 1 liter of hemorrhagic fluid was aspirated from the patient. Samples from the pericardial fluid were prepared for biochemical, microbiologic, and pathologic examinations. After pericardiocentesis, signs of respiratory distress resolved, arterial blood pressure began to increase, and heart beats decreased to normal levels. The parasternal long-axis (Fig. 1a) and apical four-chamber views (Fig. 1b) showed minimal residual effusion. The results of biochemical blood analysis were normal, with urea 136 mg/dl, creatinine 1.6 mg/dl, aspartate aminotransferase (AST) 22 U/l, alanine aminotransferase (ALT) 10 U/l, lactate dehydrogenase (LDL) 503 U/l, total protein 6.6 mg/dl, albumin 3.6 mg/dl, and C-reactive protein (CRP) 88 mg/dl. Hematological parameters were as follows: hemoglobin 13.5 g/dl, leukocyte count 11,300/mm³, and erythrocyte sedimentation rate 49 mm/hour. Because of the infiltrative appearance on the chest X-ray, consultation was obtained from the Chest Diseases Department. The patient was diagnosed as having pneumonia and oral clarithromycin and intravenous levofloxacin were initiated. Anticoagulation was avoided because of the hemorrhagic nature of the aspiration fluid even though atrial fibrillation was present. Oxygen saturations started to decrease progressively. Arterial blood gas values were as follows: pH 7.5, pO₂ 59 mmHg, pCO₂ 24 mmHg, and HCO₃ 19 mmol/l. D-dimer was measured as 4 ng/l (upper normal limit 1 ng/l at laboratory). Pulmonary embolism was considered after obtaining another consultation from the Chest Diseases Department. Anticoagulation therapy was added to treatment. After continuous

positive airway pressure (CPAP) therapy, prognosis of the patient showed improvement. Subsequent echocardiographic evaluations showed no progression in the pericardial fluid. Biochemical analysis showed exudative pericardial effusion. Pathological result was interpreted as benign. Acid-resistant bacteria were not found with Gram, Giemsa, and Ehrlich-Ziehl-Neelsen staining of the pericardial fluid samples. Bacteria cultures were also negative. Pericardial fluid ADA activity was measured as 76 U/l. The final diagnosis was made as tuberculous pericarditis. Antituberculosis treatment with isoniazid (300 mg 1x1 po), rifampin (300 mg 1x2 po), pyrazinamide (500 mg 3x2 po), and ethambutol (1x2 po) was initiated and continued for six months. Liver enzymes showed normal levels. The patient's general status improved and INR values returned to normal. She was discharged from the hospital. During a follow-up period of 12 months, no signs of deterioration were observed and echocardiographic findings were normal.

DISCUSSION

Tuberculosis is the primary cause of pericarditis in developing countries.^[1] It is detected in 1-2% of all acute pericarditis cases. Cardiac tamponade is the main presentation in 7% of these cases. Tuberculosis-related mortality rates are still 14% to 40%.^[2] Cough, shortness of breath, orthopnea, night sweats, weight loss, and ankle edema are common symptoms in tuberculous pericarditis. The most common findings are cardiomegaly, pericardial rub, fever, and tachycardia. Hepatomegaly, distended jugular veins, distant heart sounds, paradoxical pulse, and pleural effusion are common in large effusions.^[6] Diagnosis of tuberculous pericarditis is based on the follow-

ing parameters: (i) identification of *Mycobacterium tuberculosis* from the pericardial tissue or fluid cultures, (ii) histopathologic demonstration of granulomas or acid-fast bacilli in the pericardial tissue, (iii) formation of granulomas in pericardial tissues in the presence of known extracardiac tuberculosis, and (iv) response to specific antituberculosis therapy.^[6,7] Clinical progression of tuberculous pericarditis exhibits four phases, including dry phase, effusion phase, absorptive phase, and constrictive phase. Initially, effusion is frequently accompanied by pericardial fibrin formation.^[6] Based on echocardiographic findings, tuberculous pericarditis is categorized into early stage, when only pericardial effusion is present, and advanced stage, when fibrin strand formation or fibrosis are observed, resulting in pericardial thickening and suggestive of constrictive pericarditis.^[8]

It is rather difficult to isolate *Mycobacterium* species from pericardial fluid samples and it is diagnostic in only one-third of the cases.^[4] In our case, pathological result of the pericardiocentesis material was benign, acid-resistant bacteria were not found in the pericardial fluid samples, and bacteria cultures were also negative. The only parameter suggesting tuberculosis was the ADA activity in the pericardial fluid. It was reported that ADA values exceeding 70 U/l in the pericardial fluid had a 100% sensitivity and 91% specificity for tuberculous pericarditis, and that this might serve as a useful biochemical marker for tuberculous pericarditis.^[9]

In our case, echocardiographic views before pericardiocentesis could not be captured due to emergency limitations and the patient's poor clinical status.

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