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INTRODUCTION

Normal pericardium surrounds the pericardial cavity and contains an ultrafiltrate plasma. Usually, pericardial effusion is mentioned when this fluid exceeds the required amount of 15-50 cc (1). The cause of pericardial effusion and disease varies depending on the epidemiology and patient population. Most of the cases, which are difficult to diagnose and treat, are considered idiopathic. At this stage, the definitive diagnosis is significant in terms of the prognosis of the patients.

Pericardial effusions can occur acute, subacute, or chronic. The normal pericardium may stretch to accommodate volume increases, depending on how rapidly the effusion develops. Slow-growing effusions are more flexible than rapidly growing effusions. Pericardial effusion and diseases may occur due to an isolated disease affecting the heart or systemic disease manifestation. Differential diagnosis of the disease and multidisciplinary, contemporary treatment approaches are essential to reveal treatable causes. Effusion can be detected incidentally by diagnostic procedures in symptomatic patients or by echocardiography in asymptomatic patients. The first thing to do after the effusion is detected; is to determine the amount of effusion, evaluate the patient's hemodynamic status, and

determine the associated diseases that may lead to this.

CASE REPORT

A 66-year-old female patient with a known Essential Hypertension had admitted to our clinic with complaints of fatigue, constant sleepiness, headaches, and chest pains that last for about a week. The patient stated that the chest pain was intermittent, increased significantly in the lying position, was a needle-prickling style, and decreased when sitting. She stated that her headache complaint showed no specificity and continued intermittently.

She had a history of cholecystectomy 30 years ago, no history of smoking, alcohol, or herbal medicine use, and she had a history of 4 mg Benidipine and 50 mg Losartan potassium antihypertensive drug use for hypertension. There was no acute pathological finding in the systemic physical examination of the patient. Biochemical test results were as normocytic anemia, acute renal failure, increased sedimentation, and in the complete urinalysis, pyuria was found [Table 1]. Patient consent was obtained to share the biochemistry and imaging results.

A posteroanterior chest X-ray was performed on the patient who complained of chest pain. Pleural effusion and cardiothoracic index were increased in the imaging of the patient [Figure 1].

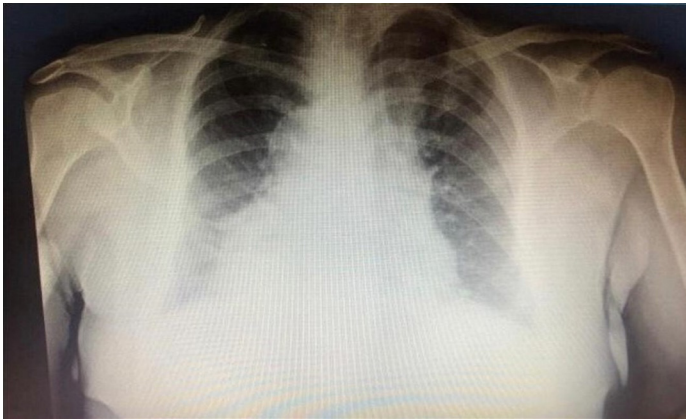


Figure 1. Posteroanterior chest radiograph of the case

Due to the current pandemic period (COVID-19), scanning with thorax computed tomography was planned for the patient whose clinical status and laboratory results were evaluated and whose infection parameters were found to be high. Echocardiographic evaluation was planned for the patient who had pleural effusion in the bilateral hemithorax and effusions in the pericardial area, whose possible covid-19 pneumonia diagnosis was excluded in the computed tomography scan and who had no known cardiac disease or heart failure diagnosis [Figure 2]. Ertapenem 1 gram/day parenteral antibiotic therapy was started for the patient who had pyuria and high infection parameters in the complete urinalysis.

In the echocardiographic evaluation of the patient, pericardial effusion and fibrin bands of 2 cm were detected in the deepest part. Pulmonary arterial pressure was measured as 35-45 mmHg. Oral treatment of 2*0.5 mg colchicine was started in the patient with pericardial effusion. Non-steroidal anti-inflammatory treatment was not started in the patient because of acute renal failure. It was planned to evaluate rheumatological autoantibodies for connective tissue diseases that may cause pericardial effusion. It was planned to investigate the possible causes and determine the etiology of the patient, whose pleural and pericardial effusions to explain chest pain complaints.

Due to the development of acute respiratory distress during the patient's follow-up in the service, control echocardiography was planned with the preliminary diagnosis of cardiac tamponade. It was observed that there was no increase in the amount of pericardial effusion, and there was no cardiac tamponade. Pulmonary CT angiography scanning was planned for the preliminary diagnosis of an acute pulmonary embolism due to evaluated arterial blood gas results; hypoxia, hypocarbia, and respiratory alkalosis. In the computerized tomography imaging of the patient, the preliminary diagnosis of pulmonary embolism was excluded, and the amount of pleural effusion was found to be increased. Parenteral diuretic therapy and parenteral steroid therapy at a dose of 1 mg/kg were started in the patient for acute pulmonary edema. After the current treatment was administered for three consecutive days, treatment continued with oral maintenance dose steroid. With these treatments, it was observed that the patient's complaints

regressed, there was a significant improvement in her clinical condition. During the follow-up, the patient was scheduled for diagnostic pericardiocentesis with control echocardiography four days later. After diuretic, steroid, and colchicine treatments, the patient's pleural and pericardial effusions regressed significantly. However, due to insufficient effusion diagnostic, interventional procedures could not be performed.

Pre-diagnosis of malignant neoplasm of the breast was excluded with breast ultrasonography and mammography scanning of the female patient, who was over 50 years old. No thoracoabdominal mass or gynecological pathology was detected in the thoracic and abdominal scanning of the patient who had no smoking history. Rheumatological parameters evaluated for connective tissue diseases were negative. The preliminary lymphoma diagnosis was excluded in the patient who did not have symptoms of fever, sweating, weight loss, pathological lymphadenopathy in the systemic physical examination and superficial tissue ultrasonography, and atypical cells did not detect in the peripheral smear evaluation. Serum Adenosine deaminase enzyme level results were average in the patient. The patient had no previous tuberculosis, history of contact, and had no cavitory lesion or fibrosis in lung scanning. The submitted Quantiferon test was indeterminate, and the test was repeated. The pre-diagnosis of tuberculosis infection was excluded in the patient whose test result was negative. All pre-diagnoses reviewed in the differential diagnosis were excluded, C-reactive protein and sediment levels in the control blood biochemistry, active chest pain, and effusions regressed. The patient was evaluated as an idiopathic pericardial effusion case and was discharged with the recommendations for outpatient control.

Table 2. Laboratory variables of the patient

Lab variables	Values at the time of admission
Urea (mg/dl)	68
Creatinine (mg/dl)	1.69
WBC /ne (103/ μ l)	12.9
Hemoglobin (gr/dL)	8.2
Glucose (mg/dL)	107
TSH (mUI/L)	0.41
Sodium (mmol/L)	136
Potassium (mmol/L)	5.3
Sediment (mm/h)	92
C-reactive protein (mg/L)	219
Alanine aminotransaminase (U/L)	11
Aspartate aminotransferase (U/L)	18
Lactate dehydrogenase (U/L)	292
Uric acid (mg/dL)	8.2
INR	1.18

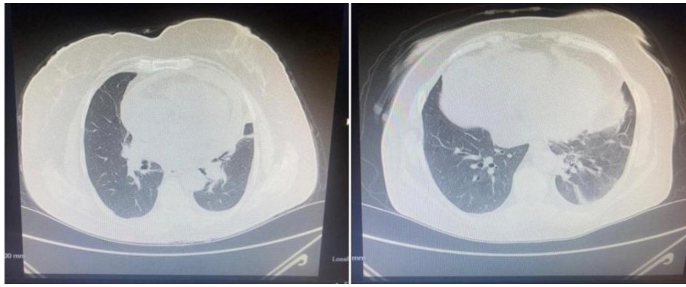


Figure 2. Thorax computed tomography image of the case, pericardial and pleural effusion

DISCUSSION

Among the most common causes of pericardial effusion; infections (viral, bacterial, tuberculosis infection especially in endemic areas), malignancies, connective tissue diseases, autoimmune diseases, pericardial injury syndromes, post-myocardial infarction or cardiac surgery, metabolic causes, hypothyroidism, mediastinal radiation, uremia and renal failure, myxedema, side effects of some drugs effect (procainamide, phenytoin, methyl dopa, penicillins, doxorubicin, tyrosine kinase inhibitors), myopericarditis and aortic diseases. The frequency of these causes varies according to epidemiology, hospital setting, and conventional diagnostic approaches. While many cases are considered idiopathic in developed countries, tuberculosis is the predominant cause in developing countries (2-4). These reasons are revealed with the symptoms and biochemical values of the patients, electrocardiogram, and echocardiographic findings. Our case is a case that we evaluated as idiopathic after various diagnostic stages.

Most patients without hemodynamically significant pericardial effusion do not have signs and symptoms of effusion, but there could be symptoms of the underlying disease. For this reason, pericardial effusions are usually detected incidentally during the evaluation of other diseases; patients with significant pericardial effusion present with impaired hemodynamic status, cardiac tamponade, and secondary symptoms.

The presence of pericardial effusion can be demonstrated by the patient's history, systemic physical examination, electrocardiogram, echocardiography, and radiographic findings. Echocardiography should be performed in every patient suspected of pericardial disease. Echocardiography is a non-invasive method that is both specific and sensitive for the detection of pericardial disease (1,2). Systematic approaches are recommended in standardized practice guidelines. Thus, it aimed to reduce the number of idiopathic cases and identify treatable causes. In our case, differential diagnoses were made with algorithmic approaches until the last stage, and the case was evaluated as idiopathic. Additional imaging modalities such as computed tomography or magnetic resonance imaging may be needed in non-diagnostic echocardiographic findings with a high clinical suspicion of pericardial effusion. In our case, who had pleural effusion with pericardial effusion, we also supported our

diagnosis with thoracic computed tomography imaging. After detecting pericardial effusion, the first thing to do is to evaluate the hemodynamic status of the patient. The clinical situation varies according to the amount of pericardial fluid and the rate of accumulation. If pericardial fluid accumulates rapidly, it can cause cardiac tamponade within minutes. If it accumulates slowly, it can reach large amounts without causing signs and symptoms (5,6). Classic symptoms include orthopnea, dyspnea after exertion, and chest pain. Additional symptoms due to local compression; nausea (diaphragm), dysphagia (esophagus), hoarseness (laryngeal nerve), and hiccups (phrenic nerve) may develop. Nonspecific symptoms include cough, weakness, fatigue, anorexia, and palpitations. Hypotension and sinus tachycardia indicates the compressive effect of pericardial fluid on adjacent anatomical structures. Our case also had complaints of fatigue and chest pain at the time of admission. A patient's fever is a nonspecific sign that may be associated with infectious or immune-mediated pericarditis (7-9).

The development of cardiac tamponade without inflammatory manifestations is associated with a higher risk of neoplastic etiology, while cardiac tamponade and a severe effusion without inflammatory manifestations are usually associated with a chronic idiopathic etiology (10,11).

Pericardial effusion management aims to treat the underlying disease. In approximately 60% of cases, the effusion is associated with a known disease. When the cases with pericardial effusion are symptomatic, and when medical treatments fail, patients with impaired hemodynamic stabilization should be considered for surgical drainage of effusion (2,8,12). In idiopathic chronic effusions, non-steroidal anti-inflammatory drugs, colchicine, and corticosteroids can be administered, but there is no proven study on these treatments (13-15). Invasive treatment options should be considered in large amounts of effusions unresponsive to medical treatment.

CONCLUSION

Pericardial effusion prognosis is related to the etiology of the disease. In the studies performed, infectious and idiopathic cases were found to have a good prognosis (16). Although the etiology of most cases is unknown, etiologies can be revealed with long-term follow-up. At this point, the important thing is to continue the follow-up of the cases that we consider as idiopathic, taking into account the hemodynamic status of the patient, the amount of effusion, and the rate of development, in order not to miss the treatable causes of pericardial effusion. In the long-term follow-ups, it has been revealed that the cases may have tuberculosis infection or malignancy (10). However, the optimal follow-up period has not been defined for the cases. In a study conducted by Imazio et al. in three Italian centers between 2000 and 2015, it was observed that cardiac tamponade could develop in a small number of cases, while the amount of effusion regressed in some (11). Therefore, it should be kept in mind that asymptomatic patients may experience deterioration in their hemodynamic

status during their follow-up. Our pericardial effusion case with idiopathic etiology benefited from oral maintenance dose steroid and colchicine treatments; the amount of effusion regressed and was followed up for a long time.

Conflict of interests

The authors declare that they have no competing interests.

Financial Disclosure

All authors declare no financial support.

Informed Consent

Written consent was obtained from the patient and his parents..

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