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Diagnosis and treatment of constrictive pericarditis: From chest X-ray to pericardiectomy or anti-inflammatory therapy

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Introduction

Constrictive pericarditis (CP), a serious, often a difficult diagnosis that causes heart failure, is associated with thickening and calcification of the pericardial layers, impairing the diastolic functions of the heart (1). Chest X-ray, transthoracic echocardiography (TTE), cardiac computed tomography (CT) and magnetic resonance imaging (MRI), and left-right cardiac catheterization are used for diagnosis (1).

The standard treatment for chronic CP is pericardiectomy (1). In the transient form of the disease, anti-inflammatory therapy is the primary treatment option (2). In these two case reports, we want to pay attention to the diagnostic process and the importance of distinguishing the chronic form of the disease from the transient form, and the difference between the two treatment modalities.

ABSTRACT

Constrictive pericarditis (CP) consists of pericardial fibrotic thickening and calcification and causes the impaired diastolic filling of the ventricles. Echocardiography and other imaging modalities and left-right cardiac catheterization are used for diagnosis. The standard treatment for chronic CP is pericardiectomy. However, in the transient form of the disease, anti-inflammatory therapy should be the primary treatment option. In two cases of CP, the diagnostic process and the importance of distinguishing the chronic form of the disease from the transient form, and the difference in the two treatment modalities have been highlighted.

Case Presentations

Case 1

A 32-year-old male patient was admitted with shortness of breath. Physical examination revealed pretibial edema, and increased jugular venous pressure. On admission, the electrocardiogram showed atrial fibrillation (AF), and the chest X-ray revealed pericardial calcification (Figure 1A). TTE examination showed normal biventricular function, moderate to mild mitral and tricuspid regurgitation, the diastolic bounce of the interventricular septum, and >25% variation in mitral inflow with respiration, mitral annular septal-lateral é reversal, and increased pericardial thickness (Figure 1B, 1C). Cardiac CT showed diffuse calcification of the pericardium, and the calcific plaques were 6 mm at the thickest part (Figure 1D). The patient was diagnosed with CP, but he refused surgery. On a phone

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visit 6 months later, the patient was learned to have undergone pericardiectomy and was clinically better.

Case 2

A 50-year-old female patient was admitted with dyspnea and palpitations. We learned that the patient was a refugee and had a history of pericarditis and pericardiocentesis 6 months ago in his own country. In laboratory examination. hepatic and renal function and albumin levels were normal but brain natriuretic peptide 950 pg/mL, C-reactive protein 58 mg/L, white blood cell count 12.000/µL. The TTE of the patient revealed normal biventricular function, moderate atrial dilatation, marked dilation of the inferior vena cava (25 mm), and increased thickness of the pericardium (Figure 2A). Also, diastolic bounce of the interventricular septum, >25% variation in mitral inflow with respiration, and mitral annular septal-lateral é reversal, as indicators of ventricular interdependence, were observed (Figure 2B, 2C). Cardiac CT showed increased pericardial thickness and minimal calcification (Figure 2D). In the catheterization, left ventricular and right ventricular end-diastolic pressures equalized and increased, right atrial pressure increased, X and Y descent became evident, and there was respiratory discordance (Figure 2E, 2F). Since the patient had a previous history of pericarditis and pericardiocentesis

and pericardial calcification was more limited, a follow-up was planned with anti-inflammatory therapy to rule out a transient constriction. In addition to standard medical therapy, the patient was prescribed oral corticosteroid. The patient showed clinical and TTE improvements at the 3-month follow-up.

Discussion

In these two case reports, we intended to draw attention to some significant differences in the diagnosis and treatment of CP. In particular, we emphasized that, after the diagnosis of CP is made, the transient form of the disease should be distinguished. In addition, CP can successfully be treated with anti-inflammatory therapy without the need for pericardiectomy.

CP is characterized by abnormal diastolic filling patterns and low cardiac output. The most common clinical findings are shortness of breath, edema, and increased jugular venous pressure. Also, hepatomegaly, pleural effusions, ascites, and hypoalbuminemia may occur in more advanced cases.

Chest X-ray, TTE, cardiac CT and MRI, and left-right cardiac catheterization are used for the diagnosis of CP (1). Chest X-ray can be an initial diagnostic test that can detect pericardial calcifications (3). The essential pathophysiologic mechanism in CP is the dissociation of intrathoracic and intracardiac pressures and ventricular interdependence within a fixed space.



Figure 1. A-C: Chest X-ray, computed tomography, and echocardiographic images of increased pericardial thickness and calcification. D: >25% variation in E mitral inflow wave with respiration on echocardiography



Figure 2. A, B: Transthoracic echocardiography showed >25% variation in mitral inflow with respiration, dilation of the inferior vena cava, and diminished collapse. C: Mitral annular septal-lateral é reversal. D: Thorax computed tomography showed increased pericardial thickness and minimal calcification. E, F: In the left and right heart catheterization, left ventricular and right ventricular end-diastolic pressures equalized and increased, right atrial pressure increased, X and Y descent became evident, and respiratory discordance

Through these pathophysiologic mechanisms in TTE, the abnormal septal motion might have increased the respiratory variation of mitral inflow velocity, and also reversed the mitral annulus septal-lateral velocity (4). In addition to TTE, CT is a valuable imaging modality, especially in the evaluation of pericardial calcifications (5). With the development of newer imaging methods, there has been a shift to invasive imaging tools, particularly when CP is suspected. However, cardiac catheterization remains a diagnostic tool, particularly when other imaging tools are inconclusive (6).

While it is critical to distinguish the disease from restrictive cardiomyopathy before the diagnosis of the disease, it is important to distinguish the disease in its transient form after the diagnosis of CP. The transient constriction is a typical clinical course that implies the presence of acute inflammatory pericarditis with constriction due to inflammation, which resolves once the inflammatory process is treated. Thus, conservative treatment may be conducted for 2 to 3 months before recommending pericardiectomy in the absence of signs that the problem is chronic (for example, cachexia, AF, hepatic dysfunction, or pericardial calcification) (2). Also, CT and MRI can be used to detect pericardial inflammation to diagnose transient constriction (5).

The standard treatment for chronic CP is pericardiectomy (1). However, pericardiectomy has significant perioperative mortality ranging from 6 to 12% (1,7). Therefore, pericardiectomy should be carefully evaluated in patients with left ventricular systolic dysfunction, severe renal and hepatic failure, cachexia, hypoalbuminemia, and patients with radiation-induced CP (1,2). Considering this information, we recommended pericardiectomy in our first case because the patient was young, did not have cachexia, and had low comorbidities.

In the transient form of the disease with concomitant evidence of pericardial inflammation and increased inflammatory markers, empiric anti-inflammatory therapy can be considered (1). While the combination of non-steroidal antiinflammatory drugs and colchicine is recommended as initial treatment, corticosteroids can be chosen in resistant cases (1). In our second case, corticosteroid treatment was started due to increased inflammatory markers in the blood tetsts, resistance of the disease to the fşrst line treatment and the absence of findings indicating the chronic form of the disease such as hypoalbuminemia and pericardial calcification.

Conclusion

The diagnosis and treatment steps of two different cases of CP are discussed in this case report. The evaluation of patients

for transient constriction, especially before pericardiectomy can be recommended, which could prevent unnecessary and highrisk surgery.

Ethics

Informed Consent: Informed consent forms were obtained from all patients for the case reports here.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: S.A., S.E., Design: S.A., M.Ç., E.M., Data Collection, or Processing: S.A, E.M., Analysis, or Interpretation: S.A., M.Ç., H.K.K., Literature Search: S.A., S.E., Writing: S.A.

Conflict of Interest: No conflict of interest was declared by the authors.

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