Delayed Diagnosis of Constrictive Pericarditis Resulting in Recurrent Heart Failure: A Case Report

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ABSTRACT: Constrictive pericarditis can lead to compromised diastolic ventricular filling due to pericardial inflammation and fibrosis. A diagnosis of constrictive pericarditis was established by identifying structural and hemodynamic features through echocardiography. We present a case of constrictive pericarditis, which manifested in the form of gradually worsening dyspnea and lower-extremity edema over a 7 years period. The patient was diagnosed with constrictive pericarditis using echocardiography, and underwent a pericardiectomy.

KEYWORDS: Constrictive pericarditis, echocardiography, respirophasic ventricular septal shift, recurrent heart failure

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Introduction

Constrictive pericarditis (CP) is caused by a noncompliant pericardium lacking elasticity, which prevents both ventricles from properly filling and ultimately results in diastolic heart failure.[1] Hemodynamic abnormalities in CP are characterized by the decoupling of intrathoracic and intracardiac pressures during respiration as well as heightened ventricular interdependence.[2] Unfortunately, CP patients are often initially misdiagnosed with restrictive cardiomyopathy (RCM), severe tricuspid regurgitation, liver cirrhosis, or severe chronic obstructive pulmonary disease.[2] However, CP should be distinguished from these and other causes of heart failure because it can be cured with pericardiectomy.[3] As a result, we present a case of recurrent heart failure that resulted from the delayed diagnosis of constrictive pericarditis.

Presentation of the Case

A 67-year-old man was admitted to the hospital with progressively worsening dyspnea and lower-extremity edema for 7 years, at which time, he was diagnosed with chronic right heart failure due to lower limb edema. To alleviate his symptoms, he would intermittently take loop diuretics. However, this medication failed to control his edema, which continued to gradually increase, and even resulted in pleural effusion and ascites. Five years ago, he was diagnosed with heart failure resulting from coronary artery disease, which manifested as generalized edema and dyspnea. He was treated with diuretics and other related medications in an attempt to improve prognosis. However, once he was forced to discontinue diuretics due to hypotension, his dyspnea became acutely exacerbated and he experienced a relapse of lower extremity edema. During this 5-year period, responding poorly to diuretics, the patient underwent high saphenous vein ligation and stripping to treat his leg edema. He also underwent multiple drainage procedures for pleural and abdominal effusions. About a month ago, a transthoracic echocardiography performed at another hospital demonstrated bi-atrial enlargement, mild mitral and tricuspid valve insufficiency, and a left ventricular ejection fraction of 57%. At that time, the patient denied a history of diabetes or hypertension and reported no inherited heart disease.

On admission, the patient's blood pressure was 108/82 mmHg, and his heart rate was approximately 124 beats/min and demonstrated an irregular rhythm. Deep breathing elicited Kussmaul's sign and pulsus paradoxus. Auscultation detected a mild pericardial knock. Breath sounds were absent in the right lower lung, shifting dullness was present, and both lower extremities exhibited pitting edema. Blood tests indicated a brain natriuretic peptide level of 652 pg/mL (normal value <100 pg/mL). Yet, the results of the other blood tests were normal. An electrocardiography revealed atrial fibrillation, low voltage in limb leads, and nonspecific minor ST-T abnormalities. Computed tomography demonstrated increased pericardial thickness with calcification, a large amount of ascites, and a right pleural effusion (Figure 1). The assays of pleural effusion and ascites indicated transudative fluid in both cases. To alleviate dyspnea and edema, the patient received furosemide and underwent drainage procedures for the ascites and pleural effusion.

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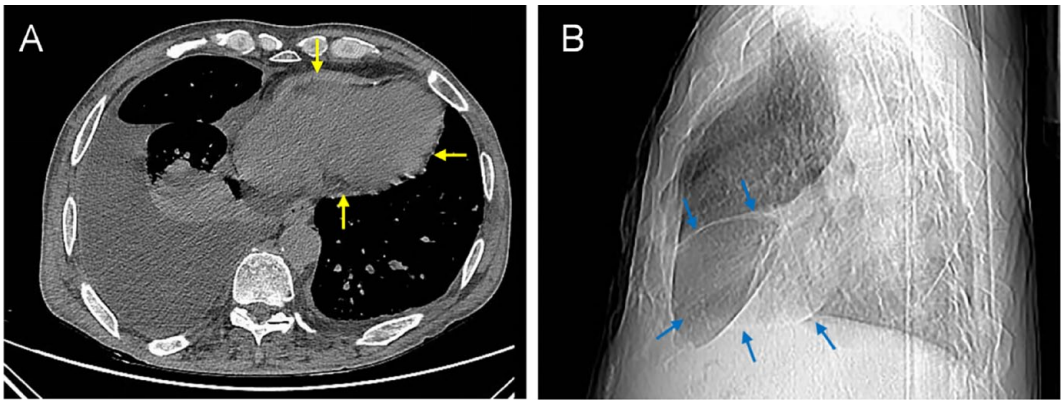

Figure 1. Computed tomography revealed increased pericardial thickness with calcification. Panel (A): Thickening and calcification of the pericardium (yellow arrow). Panel (B) Circular calcification of the pericardium (blue arrow).

To provide a more accurate assessment of structure and function of the patient's heart, we repeated echocardiography for this patient again. Two-dimensional and M-mode imaging revealed a shift of interventricular septum: to the left during inspiration, and to the right during expiration (Figure 2; Supplemental Materials, Video 1). Additional findings included a septal bounce, dilation of the inferior vena cava (3.18 cm) and hepatic veins, along with the absence of inspiratory collapse (Figure 2; Supplemental Materials, Videos 1 and 2). The tricuspid annular plane systolic excursion, however, was normal. Doppler echocardiography demonstrated that there was mitral and tricuspid regurgitation, and that hepatic venous flow reversal was accentuated during expiration (Supplemental Materials, Video 2). Tissue Doppler imaging revealed a mitral annular e′ velocity of 11 cm/s (Figure 2).

Coronary angiography findings indicated 50% to 60% stenosis in the mid-distal segment of the left anterior descending artery, as well as sporadic plaques in both the right and left circumflex coronary arteries. Based on a combination of the above-described clinical manifestations and transthoracic echocardiographic findings, the patient was conclusively diagnosed with chronic constrictive pericarditis. Once his heart failure had improved, the patient underwent a pericardiectomy at another hospital, and postoperative pathology confirmed chronic pericardial fibrosis with calcification. However, biopsy studies of the pericardial tissue did not provide definite insight into the etiology. Since the patient was diagnosed with tuberculosis 32 years ago, constrictive pericarditis due to tuberculosis may be the final diagnosis. After being discharged from the hospital, the patient underwent cardiac rehabilitation therapy. For the past 6 months, the patient has been able to perform general physical activities without experiencing recurrent heart failure.

Discussion

CP is a rare disease. There is no definitive epidemiological data available on the disease, despite the fact that it has a reported incidence rate of 0.2% to 0.4% following cardiac surgery.[4] As a result, it is often overlooked by clinicians. The noncompliant pericardium resulting from inflammation and fibrosis can hinder the filling of both ventricles, leading to diastolic heart failure.[1] CP exhibits numerous clinical features that resemble other diseases such as RCM, severe tricuspid regurgitation, liver cirrhosis, and severe chronic obstructive pulmonary disease. As a result, CP is frequently misdiagnosed for these diseases and countless others, moreover, the patient underwent both cardiac and non-cardiac tests and interventions.[2] This CP patient, for example, diuretic therapy failed to sufficiently relieve diastolic dysfunction of heart. On the contrary, due to insufficient ventricular filling, it is possible to induce hypotension on the basis of diuretic therapy. Unfortunately, because of his initial misdiagnosis, over the course of a 7-year period, the patient underwent great saphenous vein surgery and multiple drainage procedures performed instead of seeking etiology. However, CP should be distinguished from other causes of heart failure because it can be cured with pericardiectomy.[3] Therefore, establishing an accurate diagnosis is crucial to eliminating unnecessary patient suffering.

Part of the key to making an accurate diagnosis is recognizing the clinical manifestations of CP, which include dyspnea, the presence of kussmaul sign and pulsus paradoxus. However, these are nonspecific to differentiate from RCM and severe tricuspid regurgitation, particularly in the presence of atrial fibrillation.[2] However, unexpectedly low B-type natriuretic peptide in the context of significant volume overload should prompt consideration of CP.[5] As for this patient, severe dyspnea, general edema and atrial fibrillation are inconsistent with the mild elevation of B-type natriuretic peptide. Given this, and in the light of the mild pericardial knock, we began to suspect CP.

With CP, the noncompliant pericardium resulting from inflammation and fibrosis can hinder the filling of both ventricles, leading to diastolic heart failure. The hemodynamic consequence of CP is that the interaction of respiratory intrathoracic and intracardiac pressure is isolated by the inelastic pericardium, which then increases ventricular interdependence during respiration.[6] Such changes may be detected through echocardiography. The Mayo Clinic has recommended the following echocardiography diagnostic criteria for CP: respirophasic ventricular septal shift (VSS), medial mitral e′

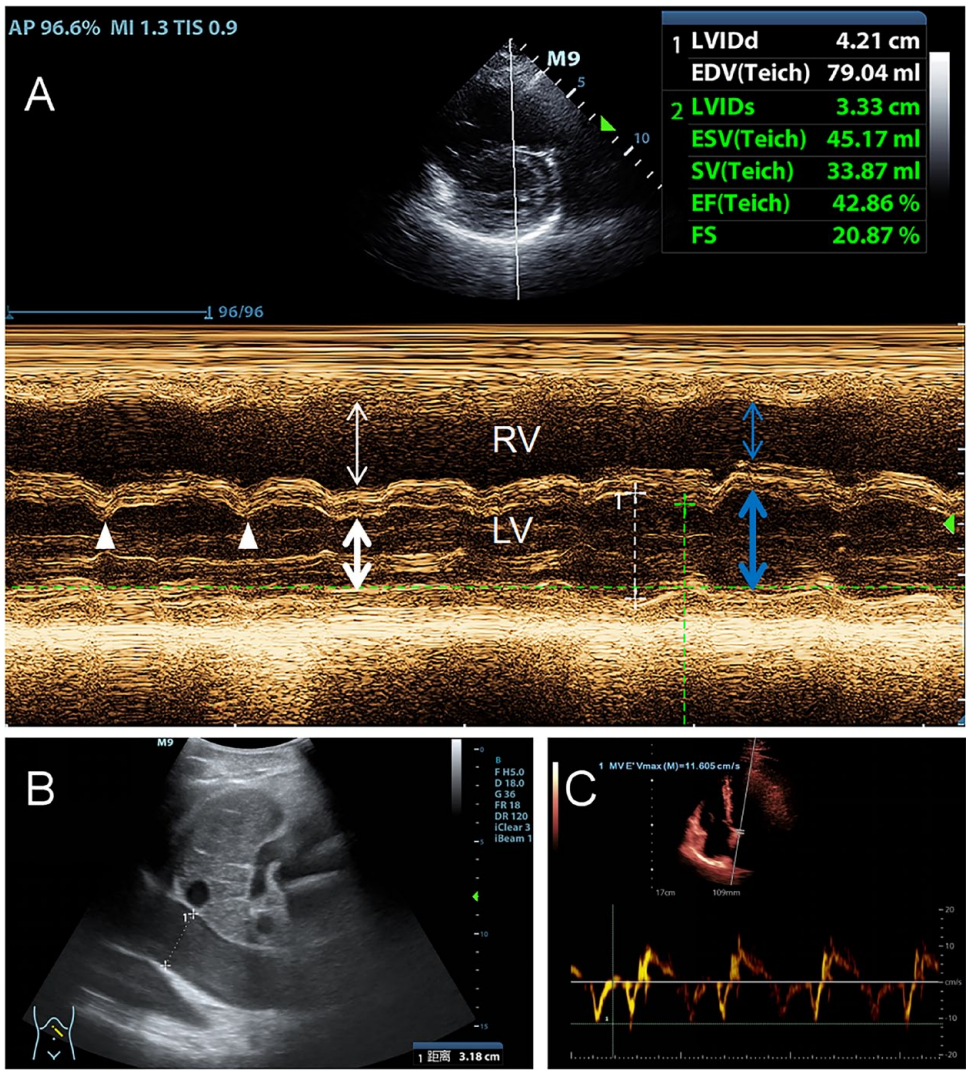

Figure 2. The echocardiography findings. Panel (A): espirophasic ventricular septal shift and septal bounce. The interventricular septum shifts to the left during inspiration (white arrow), while right during expiration (blue arrow). Septal bounce is characterized by a shape that resembles the square root sign (white triangle). Panel (B): Dilation of the inferior vena cava (3.18 cm) and hepatic veins. Panel (C): The mitral annular e′ velocity was 11 cm/s.

⩾9 cm/s, and hepatic vein expiratory diastolic reversal ratio ⩾79%.[7] The sensitivity and specificity of respirophasic VSS combined with either medial mitral e′ ⩾9 cm/s or hepatic vein expiratory diastolic reversal ratio ⩾79% were 87% and 91%, respectively.[7] Respirophasic VSS refers to the interventricular septum shifting toward the left ventricle during inspiration and toward the right ventricle with expiration. It is a key echocardiographic feature used to diagnose CP.[8] Additionally, medial mitral e′ ⩾9 cm/s on tissue Doppler is another important finding in CP.[9] RCM, on the other hand, is characterized by stiffness and noncompliant myocardium, resulting in a medial mitral e′ decrease below 8 cm/s.[10] In our case, the diagnosis of CP was established based on respirophasic VSS and medial mitral e′ ⩾9 cm/s . While the Mayo Clinic criteria suggested the reversal ratio was greater than 0.79,[7] accurately recording the hepatic vein reversal velocity can be challenging.[11] In this case, bedside ultrasound revealed diastolic hepatic venous regurgitation, but precise velocity measurement was hindered by intestinal gas interference.

Although echocardiography is effective in detecting pericardial thickness or calcification, computed tomography provides more precise structural details of the heart and pericardium, therefore supplying information in the preparation for pericardiectomy.[8] If the pericardium thickness exceeds 4 mm or is accompanied by calcification, further evaluation of CP should be performed using echocardiography.[11] In our case, computed tomography revealed an increase in pericardial thickness as well as calcification, thus indicating the need to consider CP as a potential diagnosis. Findings from this case are consistent with the data on CP, which indicates that pericardial thickening occurs in approximately two-thirds of CP patients, and that a quarter of cases exhibit pericardial calcification.[8]

Conclusion

The clinical manifestations and hemodynamic characteristics of CP exhibit significant similarities to other causes resulting in heart failure, such as RCM and severe tricuspid regurgitation.[2]

Therefore, it is essential to establish an accurate diagnosis. Despite its relatively low incidence, CP can be effectively treated through pericardiectomy.[3] Physicians should consider the possibility of CP in all patients with symptoms of heart failure, particularly when left ventricular ejection is preserved. Echocardiography provides crucial structural and hemodynamic features of the heart.[7] Therefore, it is essential that cardiologists possess sufficient knowledge about the utilization of echocardiography, especially when CP is suspected.

Author Contributions

Chunliang Wang and Yuzhu Fan were responsible for drafting the article. Chunliang Wang and Yu Chen were responsible for acquisition of data and analysis of interpretation of data. Yuzhu Fan and Guiting Liang were responsible for conception and design of the manuscript. Chunliang Wang, Yuzhu Fan, Tian Tu and Juan Du were responsible for critically revising the article, final approval of the article.

Availability of Data and Materials

The data for this case report are available from the corresponding author on reasonable request.

Informed Consent

The patient of the case report has given informed consent in order to publish the details of his medical history.

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Supplemental Material

Supplemental material for this article is available online.

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