

Calcific pericarditis strangling the heart, an answer to unexplained heart failure- diagnostic modalities

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Abstract

Pericardial calcification is often found incidentally from imaging studies and may be a clue to constrictive pericarditis. Constrictive pericarditis often mimics other causes of heart failure, pulmonary, or liver disease, making it hard to diagnose. Tuberculosis is the most common infectious aetiology of Constrictive Pericarditis. Living in developing countries, such as Indonesia, should warn us of the possibility of tuberculous constrictive pericarditis as a differential diagnosis of unexplained heart failure. The presented case came with complaints of shortness of breath, especially on exertion for five years, which worsened in the last 6 months. The past history of pulmonary Tuberculosis with the Cardiac CT findings confirmed the diagnosis of Constrictive Pericarditis.

Keywords: Pericarditis, Tuberculosis, Heart Failure, Liver Diseases

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Introduction

Pericardial calcification is often detected incidentally and may be a clue to constrictive pericarditis.¹ Constrictive pericarditis is acquired from a stiff pericardium that inhibits adequate diastolic filling. Finding constrictive pericarditis as the aetiology of heart failure is vital, as pericardiectomy can cure the disease.² Constrictive pericarditis often mimics other causes of heart failure, lung, or hepatic disease, making it hard to diagnose. However, this disease must become a differential diagnosis, particularly in heart failure preserved ejection fraction. Tuberculosis is the most common infectious aetiology of Constrictive Pericarditis.³ Living in Indonesia should warn us of the possibility of tuberculous constrictive pericarditis as a differential diagnosis of unexplained heart failure. We report pericardial

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calcification in a case of heart failure with unknown cause and no risk factors with a history of tuberculosis infection.

Case Report

A forty-nine years old man was referred in January 2021, for coronary angiography from the local hospital to our hospital (Dr. Soetomo General Hospital Surabaya) with the diagnosis of atypical chest pain. He complained of shortness of breath, especially on exertion for five years, which worsened in the last 6 months, limiting patient activity. His medication history was ramipril 5mg, bisoprolol 2,5mg, and furosemide 40mg daily. However, he stopped taking the drugs as the symptoms worsened after consumption. His past medical illness was tuberculosis. There was no history of hypertension, diabetes, vascular disease, or smoking. He had normal vital signs with bilateral pedal oedema and splenomegaly on physical examination which was confirmed by abdominal ultrasonography.

Transthoracic echocardiography revealed Right Ventricular (RV) failure (TAPSE 1,3cm) with an abnormal septal motion which was concluded as coronary artery disease. The patient underwent coronary angiography which revealed normal coronary arteries. However, there was a ring-like appearance encircling the heart, which is a sign of pericardial calcification (Figure 1). The suspicion of



Figure-1: X-ray (LAO49 CAU28 view) showing a calcification ring.

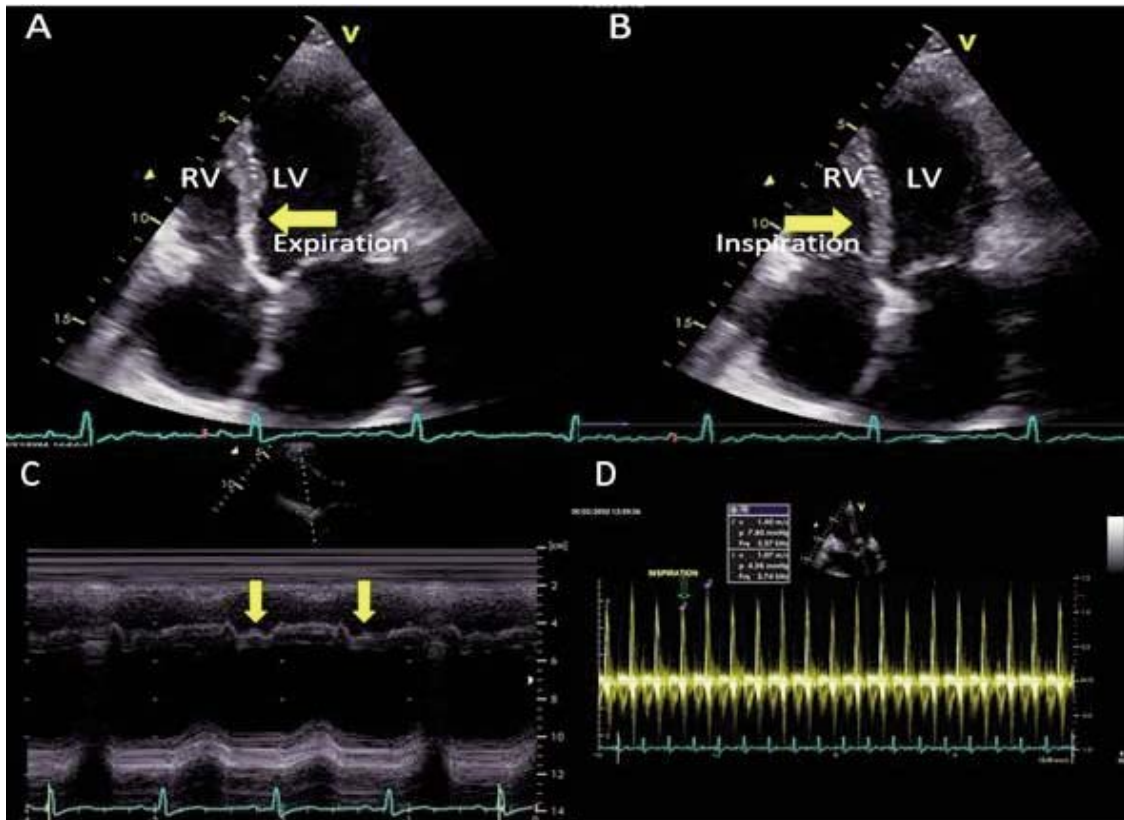


Figure-2: Apical four-chamber view: the interventricular septum (IVS) moving towards the right ventricle (RV) during expiration (A) and towards the left ventricle (LV) during inspiration (B). M-mode (parasternal long-axis view): IVS movement towards the LV with each inspiration (arrows) (C). Pulsed-waved doppler: mitral inflow velocity variation with respiration (D).

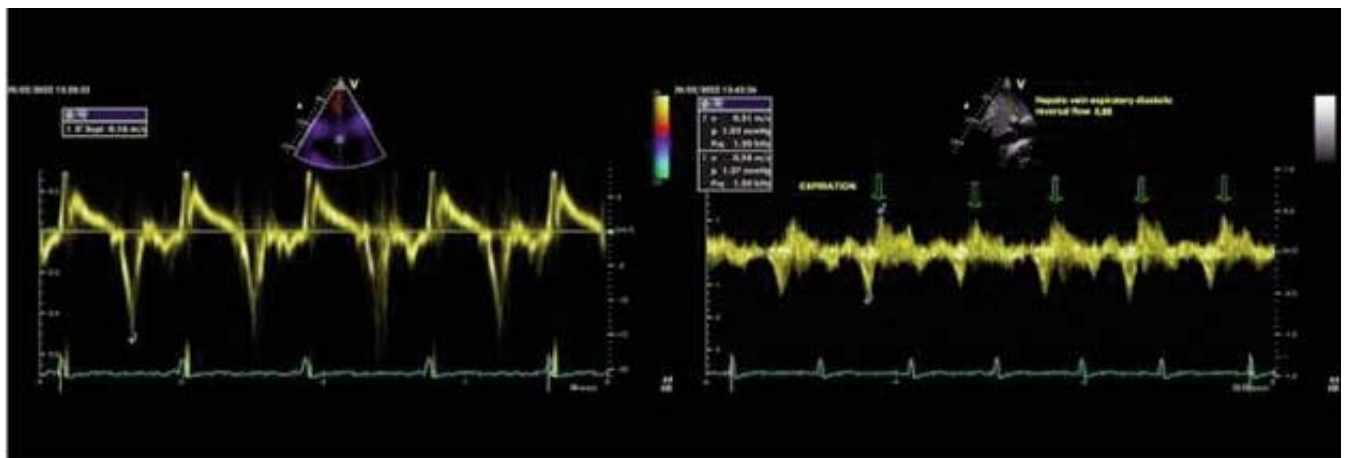


Figure-3: Doppler echocardiography shows medial annulus tissue velocity 16cm/s (A) and prominent hepatic vein flow reversals (arrow) with a ratio 0,88.

CP called for a re-examination of the echocardiography. It was observed that abnormal septal motion was related to respiration, which was a vital component of the subsequent diagnostic work-up. CP protocol echocardiogram was performed which confirmed the

diagnosis. Cardiac CT was done that found a calcific-thickened pericardium strangling the heart at the AV groove.

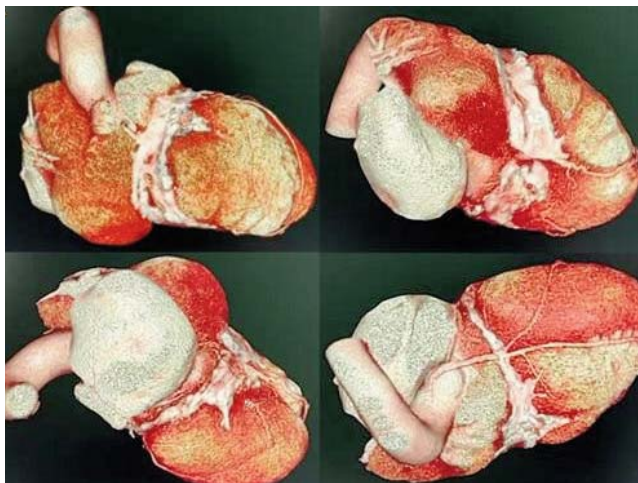


Figure-4: CT 3-D image showing pericardial calcification along the atrioventricular groove.

Discussion

The normal pericardium is 1-2mm thick, an avascular sac that encloses the heart and the roots of the great vessels. Constrictive pericarditis (CP) is caused by decreased pericardial compliance due to loss of normal elasticity, which results in diastolic heart failure.¹ It results from multiple injuries or inflammation, which could have taken place several days or years before the clinical manifestation.² The spectrum of CP's aetiology ranges from sequelae of prior cardiac surgery and chest irradiation to tuberculosis.^{1,3} Tuberculosis (TB) is the most common aetiology of calcific constrictive pericarditis in the developing world. Reviews of pericardiectomy series in the last 15 years showed the proportion of tuberculosis CP in Asia's countries is up to 80% of cases.² As Indonesia was also endemic to TB, we asked more about the patient's medical history and found he had tuberculosis 12 years ago.

The classic presentation of constrictive pericarditis consists of clinical signs of chronic right heart failure, with the most commonly reported dyspnoea on exertion and oedema.¹ The dyspnoea on effort results from raised filling pressures, whereas fatigue and weakness are due to decreased cardiac output.⁴ This clinical presentation mimics many other disorders. Patients may undergo numerous noncardiac and cardiac procedures and remain unsuspected until diagnostic evaluations have been exhausted.^{2,5,6} As the constrictive, rigid pericardium is typically thickened, fibrotic and calcified,¹ our finding of pericardial calcification presents as a ring appearance in the X-ray which is the first clue to the diagnosis. Unfortunately, it happened after five years of many diagnostic procedures.

The consequence of rigid non-pliable pericardium causes markedly restricted filling of the heart.³ The dissociation between intracardiac and intrathoracic pressures during respiration is a key to the pathophysiology, causing an enhanced interventricular dependence. During inspiration, there is an increase in RV filling, which makes a leftward interventricular septal shift, while in expiration, the opposite occurs. This phasic movement of the interventricular septum can be recognized from two-dimensional and M-mode echocardiography.³ The haemodynamic change was also present as mitral inflow velocity variation with respiration.^{1,2} These findings were detected in our patient (Figure 2).

Echocardiography is a vital imaging modality to diagnose CP. A routine echocardiogram examination will allow the exclusion of possible reasons for heart failure, such as cardiomyopathy or valvular heart disease. However, a 'CP protocol' echocardiogram is required to assure constriction and exclude restriction.¹ There are three specific parameters from echocardiography that are able to diagnose pericardial constriction. The combination between ventricular septal shift with one of these markers, either hepatic vein expiratory diastolic reversal flow ratio $\geq 0,79$ or medial mitral annulus $e' \geq 9$ cm/s was 87% sensitive and 91% specific for the diagnosis of CP.^{1,7} We performed this protocol in our patient, and from the algorithm, we met all the positive criteria: IVC plethora, tissue doppler of mitral medial e' velocity of 16cm/s, and hepatic vein diastolic reversal flow ratio 0,88 that confirmed constriction (Figure 3).

Computed tomography (CT) is a necessary adjunct to echocardiography examination in evaluating pericardial morphology. CT is useful for detecting a small amount of pericardial calcification and is the most reliable method for measuring pericardial thickness.⁸ Patients' cardiac CT discovered a calcific-thickened pericardium (3-12mm) encircling the heart at the AV groove (Figure 4). The evidence of impaired diastolic filling and pericardial thickening, especially calcification, is diagnostic of constriction in this patient.³ Furthermore, the site of pericardial calcification seems to be related to the etiology of CP. In the tubercular origin, calcification in the AV groove is a common occurrence.⁸ Finding the answer to unexplained heart failure in this patient is changing the management direction, as surgical pericardiectomy is the only curative option.²

Conclusion

In tuberculosis endemic areas, heart failure of unknown cause and absence of specific risk factors should be suspected due to pericardial calcification, especially in the

presence of a history of tuberculosis infection. A multimodality imaging approach is required for the diagnosis of CP. In the presented case, a past history of Pulmonary Tuberculosis with Cardiac CT confirmed the diagnosis of CP.

Informed consent: The consent of patient was obtained prior to the writing manuscript

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