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When the Pericardium Turns to Stone: Calcified Constrictive Pericarditis Presenting as Chronic Right Heart Failure

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Abstract | **Case Report**

Background: Constrictive pericarditis (CP) is an uncommon but potentially reversible cause of chronic right heart failure. It results from fibrous thickening and calcification of the pericardium, which restricts diastolic ventricular filling. Timely diagnosis relies on multimodality imaging, as clinical presentation overlaps significantly with other causes of heart failure. ***Case Presentation:*** We report the case of a 45-year-old woman with no history of cardiac surgery, presenting with progressive dyspnea (NYHA class III) and clinical signs of right-sided heart failure, including jugular venous distension, hepatomegaly, and bilateral lower limb oedema. Chest radiography demonstrated dense, curvilinear pericardial calcifications outlining the cardiac silhouette associated with a large right pleural effusion. Non-contrast thoracic computed tomography (CT) confirmed diffuse circumferential pericardial thickening exceeding 4 mm, extensive calcifications predominantly along the right ventricular free wall and diaphragmatic surface, a large pericardial effusion, right atrial enlargement, and dilatation of the inferior vena cava and hepatic veins. The diagnosis of calcified constrictive pericarditis complicated by right heart failure was established. The patient was managed with intensive medical therapy and underwent surgical pericardiectomy. ***Conclusion:*** This case highlights the pivotal role of multimodality imaging — particularly chest radiography and CT — in the diagnosis and management of calcified constrictive pericarditis. Early recognition is essential to ensure timely surgical intervention and improve long-term patient outcomes.

Keywords: Constrictive pericarditis; pericardial calcification; right heart failure; chest radiography; computed tomography; pericardiectomy.

1. INTRODUCTION

Constrictive pericarditis (CP) is a form of chronic pericardial disease characterized by a rigid, fibrotic, and sometimes calcified pericardium that restricts diastolic ventricular filling, ultimately leading to elevation and equalization of diastolic filling pressures across all cardiac chambers [1]. The resulting impairment in cardiac compliance manifests clinically as signs and symptoms of systemic venous congestion, including ascites, hepatomegaly, peripheral oedema, and dyspnea — a presentation frequently confused with restrictive cardiomyopathy or other causes of right heart failure [2].

The etiology of constrictive pericarditis is diverse. In developed countries, cardiac surgery, mediastinal irradiation, and viral pericarditis account for the majority of cases, whereas in developing regions, tuberculous pericarditis remains a leading cause [3]. Pericardial calcification, a hallmark of chronic disease, is most commonly associated with tuberculous or idiopathic etiologies and is identified in approximately 25–50% of constrictive pericarditis cases overall [4].

Although echocardiography is typically the first-line imaging modality for the assessment of pericardial disease, it has limitations in visualizing calcified pericardium and assessing pericardial thickness with precision. Chest radiography and, especially, computed tomography (CT) play a crucial complementary role by identifying pericardial calcifications, measuring pericardial thickness, and delineating downstream effects on cardiac morphology and the systemic venous system [5].

We present a case of calcified constrictive pericarditis in a 45-year-old woman who presented with chronic right heart failure, diagnosed through

multimodality imaging and treated with surgical pericardiectomy.

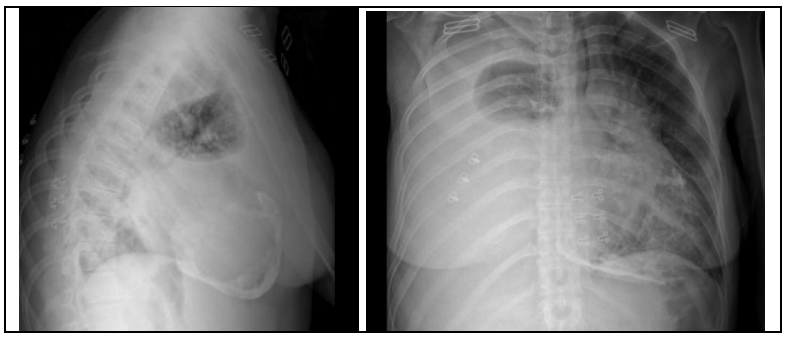
2. CASE PRESENTATION

A 45-year-old woman with no prior history of cardiac surgery, tuberculosis, or thoracic irradiation presented to the cardiology department with a one-year history of progressive dyspnea, classified as New York Heart Association (NYHA) functional class III. She reported worsening exertional breathlessness, abdominal distension, and bilateral leg swelling. There was no history of fever, chest pain, or recent respiratory infection.

Physical examination revealed elevated jugular venous pressure with prominent x and y descents, hepatomegaly on palpation, bilateral pitting oedema of the lower limbs extending to the knees, and reduced breath sounds at the right lung base consistent with pleural effusion. Heart sounds were muffled. No pericardial knock was documented at initial assessment. Vital signs were stable, with a blood pressure of 110/70 mmHg, heart rate of 90 beats per minute, and oxygen saturation of 94% on room air.

Chest radiography (Figures 1 and 2) demonstrated an enlarged cardiac silhouette with dense, curvilinear calcifications outlining the cardiac contour, consistent with extensive calcification of the pericardium. A large right pleural effusion was also identified, contributing to right basal opacification.

[Figures 1 & 2 — Chest radiography (PA and lateral views)]



Figures 1 and 2: Posteroanterior (PA) and lateral chest radiographs demonstrating dense curvilinear calcifications outlining the cardiac silhouette, consistent with extensive pericardial calcification, associated with a large right pleural effusion and cardiomegaly

Non-contrast thoracic CT (Figure 3) was subsequently performed and revealed the following findings:

- Diffuse, circumferential pericardial thickening exceeding 4 mm in all cardiac segments.
- Extensive pericardial calcifications, predominantly involving the right ventricular free wall and diaphragmatic pericardial surface.
- A large pericardial effusion adjacent to the calcified pericardium, suggesting ongoing active inflammation or chronic hemodynamic remodeling.
- Right atrial enlargement consistent with chronic right-sided pressure overload.
- Marked dilatation of the inferior vena cava (IVC) and hepatic veins, indicating elevated systemic venous pressure.

[Figure 3 — Non-contrast axial chest CT]

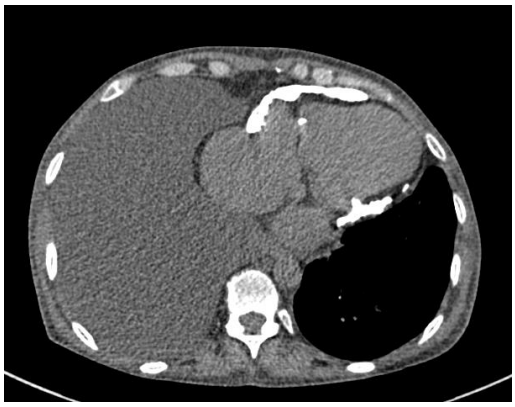


Figure 3: Axial non-contrast thoracic CT image demonstrating extensive circumferential pericardial calcifications with associated pericardial thickening exceeding 4 mm. Note the right atrial enlargement and dilatation of the inferior vena cava, consistent with chronic right-sided pressure overload secondary to constrictive pericarditis

The integration of clinical and multimodality imaging findings was consistent with the diagnosis of calcified constrictive pericarditis complicated by chronic right heart failure.

The patient was admitted to the intensive cardiac care unit. Medical management included diuretic therapy with furosemide and spironolactone for decongestion, alongside sodium restriction. Following hemodynamic stabilization, the patient underwent surgical pericardiectomy. The intraoperative findings confirmed a markedly thickened and heavily calcified pericardium with dense adherences to the epicardial surface. Surgical decortication was completed, and the postoperative course was uneventful. At three-month follow-up, the patient reported significant symptomatic improvement, with resolution of oedema and dyspnea at rest, and functional class upgraded to NYHA class II.

3. DISCUSSION

Constrictive pericarditis remains a diagnostic challenge owing to its insidious onset and clinical presentation, which closely mimics restrictive cardiomyopathy and other forms of diastolic heart failure. The coexistence of dyspnea, jugular venous distension, hepatomegaly, and peripheral oedema — as observed in our patient — constitutes the hallmark syndrome of constrictive physiology but lacks specificity when considered in isolation [2].

Pericardial calcification, while not universally present, is a pathognomonic finding when identified and strongly supports the diagnosis of chronic constrictive pericarditis. It is estimated to occur in 25–50% of cases and is most frequently associated with tuberculous or long-standing idiopathic etiologies [4]. In our patient, the extensive circumferential calcification visible on both chest radiography and CT represented the key diagnostic clue.

Computed tomography is currently regarded as the gold standard imaging modality for the assessment of pericardial calcifications and for the precise measurement of pericardial thickness. A pericardial thickness exceeding 4 mm is considered the threshold for diagnosis, though values above 2 mm are already considered abnormal [5]. CT also enables comprehensive evaluation of associated downstream effects, including dilated systemic veins, atrial enlargement, and hepatic venous congestion — all of which were present in our case and corroborate the constrictive physiology [3].

The differential diagnosis of constrictive pericarditis must include restrictive cardiomyopathy, a distinction that carries critical therapeutic implications, as pericardiectomy is curative for CP but contraindicated in restrictive cardiomyopathy. While CT accurately defines pericardial anatomy, cardiac MRI provides complementary functional information — particularly the demonstration of ventricular interdependence and abnormal septal motion — and may be warranted in equivocal cases [6]. Invasive hemodynamic catheterization, demonstrating diastolic pressure equalization and the characteristic square root sign, remains the definitive diagnostic tool when non-invasive imaging is inconclusive [2].

Surgical pericardiectomy is the definitive and only curative treatment for symptomatic constrictive pericarditis. Published series report 5-year survival rates of 78–83% after complete pericardiectomy, with hemodynamic and symptomatic improvement in the majority of patients. Operative mortality ranges from 5% to 12%, being highest in patients with heavily calcified pericardium, radiation-induced disease, or severely impaired myocardial function [7]. In our patient, pericardiectomy resulted in significant clinical improvement, confirming the diagnosis and the appropriateness of the surgical strategy.

Medical therapy with diuretics, as administered in our patient prior to surgery, plays a bridging role in managing symptoms of systemic congestion but does not address the underlying constrictive pathophysiology. Therefore, it should not delay referral for surgical evaluation in eligible candidates [1].

4. CONCLUSION

Calcified constrictive pericarditis should be included in the differential diagnosis of any patient presenting with chronic progressive dyspnea and clinical signs of right heart failure, particularly when the etiology is unclear. Chest radiography provides a readily accessible initial clue by demonstrating pericardial calcifications, while non-contrast CT offers definitive anatomical characterization of pericardial thickening, calcification extent, and hemodynamic consequences. A multimodality imaging approach, integrating radiography, CT, and echocardiography — supplemented by cardiac MRI or invasive hemodynamics when necessary — is essential for accurate diagnosis. Surgical pericardiectomy represents the definitive therapeutic intervention and can achieve substantial improvement in functional status and long-term prognosis when performed in a timely fashion.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest related to this manuscript.

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