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A CASE OF CIRCUMFERENTIAL CALCIFIC CONSTRICTIVE PERICARDITIS WITH A COMPRESSIVE COLLECTION

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Summary

Calcific constrictive pericarditis is an uncommon, yet severe manifestation of chronic pericardial inflammation, leading to diastolic heart failure through lower myocardial compliance secondary to pericardial stiffening. Its etiologies are diverse, including infectious, iatrogenic and autoimmune causes, with tuberculosis remaining a primary offender in endemic regions.

We present the case of a 67-year-old male with a history of chronic obstructive pulmonary disease (COPD) and permanent atrial fibrillation, admitted with decompensated global heart failure. Chest radiography revealed extensive, circumferential pericardial calcifications, resembling a plate armour, and a large, calcified saccular collection. Echocardiography confirmed features of constriction, including septal bounce and annulus reversus. Computed tomography further delineated the "armored heart" morphology and a large, compressive, calcified collection over the right ventricle. Diagnostic pericardiocentesis yielded a smooth, "café au lait" coloured fluid. While microbiological analysis for Mycobacterium tuberculosis was negative, the pericardial fluid Adenosine Deaminase (ADA) level was exceptionally high at 952 U/L. The diagnosis of tuberculous effusive-constrictive pericarditis in its late, burnt-out fibrocalcific stage was thus established.

This case provides a striking visual correlation between classic clinical signs of constriction and their underlying anatomical pathology, underscoring the diagnostic utility of ADA in culture-negative cases.

Keywords: *Echocardioraphic Parameters of Constrictive Pericarditis, Tuberculous Pericarditis, Pericardial Calcification, Adenosine Deaminase.*

Internal Medicine

Clinical Cases

Rezumat

Pericardita constrictivă calcificată este o formă rară dar severă a inflamației cronice pericardice, conducând la insuficiență cardiacă diastolică prin scăderea complianței miocardice secundar rigidizării pericardului. Etiologiile sunt diverse, incluzând cauze infecțioase, iatrogene și autoimune, tuberculoza rămânând un agent cauzal principal în regiunile endemice.

Prezentăm cazul unui pacient de 67 de ani, cu antecedente de boală pulmonară obstructivă cronică (BPOC) și fibrilație atrială permanentă, internat pentru clinică de insuficiență cardiacă globală decompensată. Radiografia toracică a relevat calcificări pericardice extensive, circumferențiale, cu aspect de platoșă, și o colecție saciformă calcificată de mari dimensiuni. Ecocardiografia a confirmat elemente de constricție, inclusiv septal bounce și annulus reversus. Tomografia computerizată a detaliat morfologia de „inimă în carapace" și colecția compresivă calcificată imprentând ventriculul drept. Pericardiocenteza diagnostică a extras un lichid cremos, de aspect „café au lait". Deși analiza microbiologică pentru Mycobacterium tuberculosis a fost negativă, nivelul adenozin deaminazei (ADA) din lichidul pericardic a fost extrem de înalt, la o valoare de 952 U/L. S-a stabilit diagnosticul de pericardită tuberculoasă efuziv-constrictivă în stadiu tardiv, consumat, fibrocalcific.

Acest caz oferă o corelație vizuală izbitoare între semnele clinice clasice de constricție și patologia anatomică subiacentă, subliniind utilitatea diagnostică a ADA în cazurile cu culturi negative.

Cuvinte cheie: *Parametri Ecocardiografici Pericardită Constrictivă, Pericardită Tuberculoasă, Calcificare Pericardică, Adenozin Deaminază.*

Introduction

Constrictive pericarditis is a relatively uncommon condition, characterized by a thickened, fibrotic, and often calcified pericardium, leading to venous congestion through impaired myocardial compliance and diastolic filling of the heart secondary to pericardial stiffening. While its incidence has declined in developed nations, it remains a significant cause of heart failure in regions where tuberculosis (TB) is and/or was endemic. Tuberculous pericarditis progresses through a multi-year evolution, from an initial fibrinous exudate to a chronic fibrotic and calcified state, often termed the "burnt-out" phase[1], [2]. We report a case of advanced effusive-constrictive tuberculous pericarditis with striking imaging findings that links pathophysiology and clinical presentation together.

Case Report

A 67-year-old man heavy smoker (45 pack years smoking history), with known comorbidities of permanent atrial fibrillation and GOLD stage II COPD, presented with clinical signs of decompensated heart failure. A year prior, a computed tomography (CT) scan had incidentally noted pericardial calcifications. More recently, he was hospitalized for stable angina, where an echocardiogram revealed a thickened pericardium, an encysted pericardial effusion, and hemodynamic features of constriction, prompting referral to a specialized pneumology center for suspected tuberculous etiology.

On examination, the patient was cachectic (BMI 18.3 kg/m^2). Cardiovascular assessment was notable for distended jugular veins with a positive Kussmaul's sign (a paradoxical rise in jugular venous pressure during inspiration). Auscultation revealed arrhythmic heart sounds and a prominent pericardial "knock" (a high-pitched sound in early diastole corresponding to the abrupt cessation of ventricular filling).

Chest radiography was remarkable for extensive, circumferential pericardial calcifications creating an "armored heart" appearance, with a large, calcified saccular collection overlying the cardiac silhouette (Figure 1).

Thoracic CT scan confirmed the massive, near-circumferential pericardial calcifications and a large, well-defined, calcified collection with maximum transverse and craniocaudal diameters measuring 92 x 77 mm, causing compression of the right ventricle (Figure 2).

Transthoracic echocardiography showed preserved left ventricular ejection fraction (55%) but confirmed the diagnosis of constrictive pericarditis, revealing trademark physiopathology features consisting of septal bounce (a paradoxical motion of the interventricular septum during early diastole towards left ventricle initially, followed by an opposite motion, signifying ventricular interdependence)[3], [4] and annulus reversus (lateral e′ < septal e′, meaning that early diastolic velocity of the medial mitral annulus is greater than that of the lateral mitral annulus, due to the limitation of the lateral wall motion)[3] (Figure 3).

A large, encapsulated fluid collection was visualized compressing the right ventricle (Figure 4).

Laboratory investigations are summarized in Table 1. Notably, N-terminal pro-B-type natriuretic peptide (NT-proBNP) was elevated, consistent with a heart failure diagnosis. Sputum microscopy, cultures, and molecular testing (GeneXpert) for *Mycobacterium tuberculosis* were negative. An ultrasound-guided pericardiocentesis was performed, evacuating approximately 15 mL of a thick,

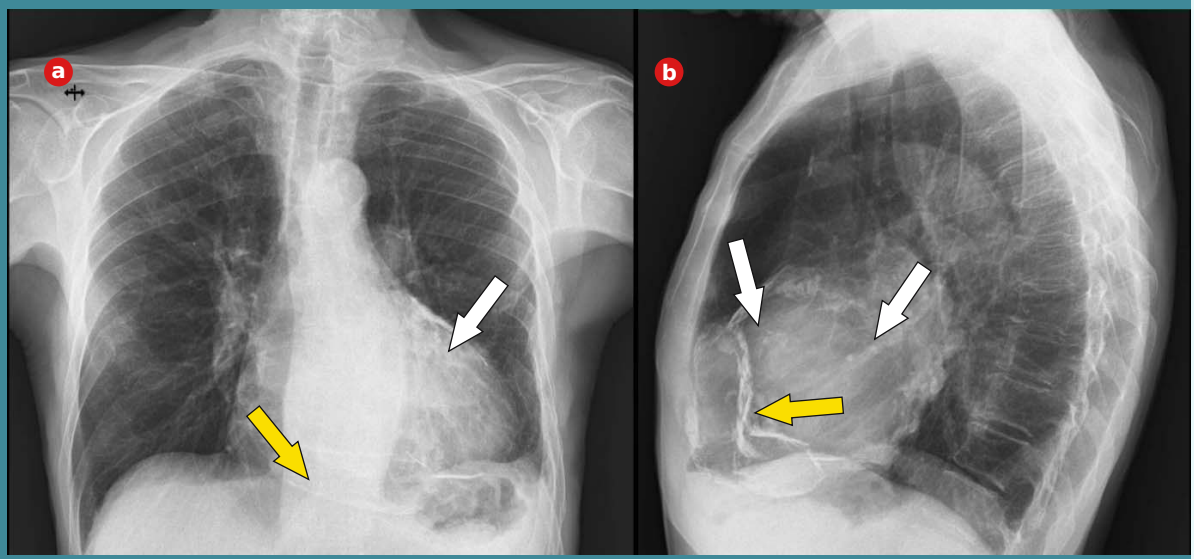

Figure 1. *Chest Radiography. (A) Posteroanterior and (B) Lateral view, demonstrating extensive, circumferential calcification of the pericardium (white arrows), creating an "armored heart" appearance. A large, calcified saccular collection is visible precordially (yellow arrows).*

creamy, non-fetid, *café au lait* colored fluid (Figure 4). Analysis of the pericardial fluid revealed a sterile exudate with an extremely high lactate dehydrogenase (LDH), very low glucose, and an exceptionally elevated Adenosine Deaminase (ADA) level of 952 U/L (Table 1). Despite the high suspicion, Ziehl-Neelsen staining, GeneXpert, and mycobacterial cultures of the fluid were negative.

Discussion

This case illustrates the severe, end-stage anatomical and physiological consequences of tuberculous pericarditis. The diagnosis was established based on a constellation of findings: the patient's origin in a TB-endemic country, the classic clinical signs of constriction (Kussmaul's sign, pericardial knock), the pathognomonic imaging of an "armored heart", and the biochemical signature of the pericardial fluid.

The central diagnostic challenge was the absence of microbiological confirmation. In the advanced fibrocalcific stage, viable mycobacteria are often absent, making culture-based diagnosis difficult. Nucleic acid amplification tests, such as GeneXpert, also

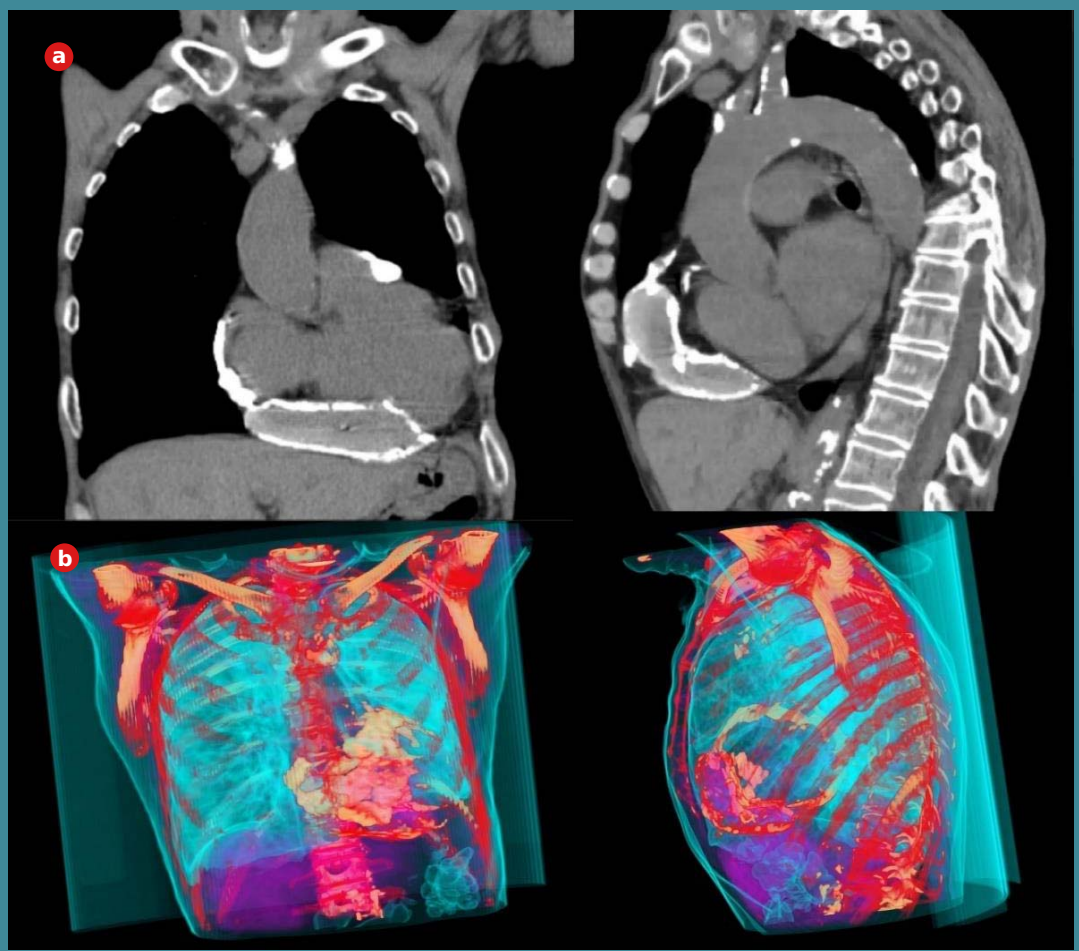

Figure 2. *Computed Tomography. (A) Classical CT Scan and (B) 3D Volume-Rendered Computed Tomography with Density Color Code, vividly illustrating the extent of the pericardial calcification (rendered in beige), which encases the heart like a shell. The images highlight the "armored heart" aspect.*

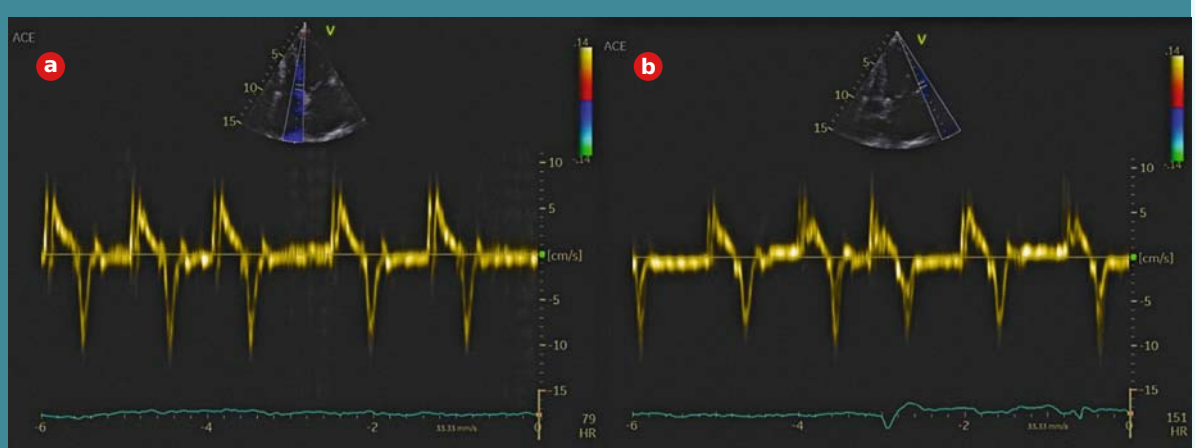

Figure 3. *Transthoracic Echocardiography. (A) Septal e' and (B) Lateral e' meassured through Tissue Doppler Imaging from an apical four chamber view.*

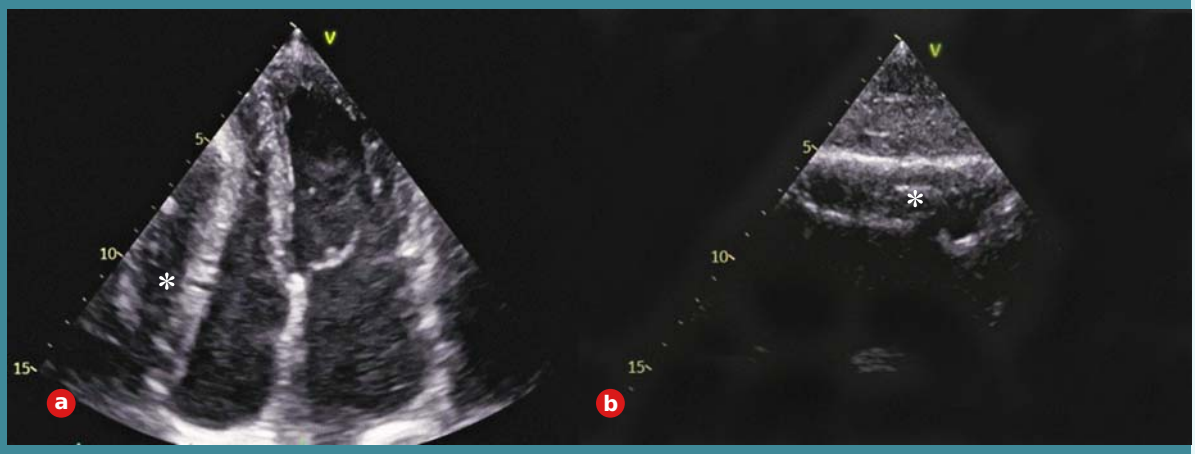

Figure 4. *Transthoracic Echocardiography. (A) Apical four-chamber view and (B) Subcostal view, showing a large, encapsulated pericardial collection (asterisk) lateral to and compressing the right ventricle (RV). The calcified pericardial layers create a posterior acoustic shadow.*

have reduced sensitivity in paucibacillary pericardial fluid specimen compared to tissue samples[1]. In this context, biomarkers like Adenosine Deaminase (ADA) become critically important. ADA is an enzyme involved in lymphocyte proliferation, and its levels are markedly elevated in tuberculous effusions due to the intense T-cell mediated immune response. A pericardial fluid ADA level >40 U/L has a high sensitivity (87-93%) and specificity (89-97%) for tuberculous pericarditis[5]. The value of 952 U/L seen in our patient is exceptionally high and, in this clinical setting, is considered virtually diagnostic of a tuberculous etiology.

The management of advanced calcific constrictive pericarditis is primarily surgical. Pericardiectomy, the stripping of the thickened and calcified pericardium, is the definitive treatment to relieve the mechanical constriction and restore normal diastolic function.

While anti-tuberculous therapy is crucial for active disease, its role in the "burnt-out" stage without evidence of active infection is debated, though often administered peri-operatively to prevent reactivation.

Conclusions

This case provides a dramatic visual representation of calcific constrictive pericarditis, the terminal stage of a chronic

Figure 5. *Pericardiocentesis and Fluid Aspect. (A) The procedure being performed after ultrasound guidance. (B) The aspirated fluid, showing its characteristic creamy, purulent, "café au lait" appearance.*

Parameter	Result	Reference range
	Blood	
Hemoglobin	11.7 g/dL	12.6 - 17.2 g/dL
C-Reactive Protein	5.76 mg/L	0 - 5 mg/L
NT-proBNP	3800 pg/mL	0 - 125 pg/mL
Total protein	8.79 g/dL	6.4 – 8.7 g/dL
Glucose	75 mg/dL	82 - 115 mg/dL
	Sputum	**Pericardial Fluid**
AFB Smear	Negative	Negative
GeneXpert MTB	Negative	Negative
Mycobacterial Culture	Negative	Negative
	Pericardial Fluid	
LDH	11,840 U/L	N/A
Glucose	5.34 mg/dL	N/A
Total Protein	19.94 g/dL	0 - 3 g/dL
ADA	952 U/L	0 - 33 U/L
Cytology	Rare neutrophils and lymphocytes	N/A

Table 1. *Laboratory Findings. Abbreviations: ADA, Adenosine Deaminase; AFB, Acid-fast bacilli; LDH, Lactate dehydrogenase; NT-proBNP, N-terminal pro-B-type natriuretic peptide.*

tuberculous infection of the pericardium. It highlights the importance of recognizing the classic, albeit increasingly rare, clinical signs of pericardial constriction.

Furthermore, it demonstrates the pivotal role of pericardial fluid Adenosine Deaminase as a diagnostic marker in TB-endemic areas, especially when conventional microbiological investigations are negative. For clinicians, this prominent aspect of the heart might serve as a memorable reminder of this disease.

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