CARDIOLOGIE TUNISIENNE

CASE REPORT

Asymmetrical constrictive pericarditis in a tuberculous endemic area: Diagnostic, Etiological, and Therapeutic Challenges

Péricardite constrictive asymétrique en zone d'endémie tuberculeuse : enjeux diagnostiques et thérapeutiques

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Summary

Introduction : Constrictive pericarditis is a rare and potentially life-threatening condition. Diagnosis can be challenging, especially when constriction is asymmetrical, limiting the sensitivity of invasive hemodynamic assessments. Tuberculosis remains a leading cause in endemic areas, but its presentation can be subtle, and systemic signs may be absent.

Case Presentation: We report the case of a 45-year-old patient presenting with signs of right heart failure and a moderate-to-large loculated pericardial effusion. Imaging revealed predominant constriction of the right ventricular free wall, without classic features of tuberculosis on thoraco-abdomin-pelvic CT, and microbiological studies (including sputum and urine cultures). Given the high epidemiological risk and inability to safely perform pericardiocentesis due to the loculated nature of the effusion, empirical anti-tuberculosis therapy (HRZE regimen: isoniazid, rifampicin, pyrazinamide, and ethambutol) was initiated.

Discussion: This case underscores several challenges: the diagnostic challenge of recognizing constrictive pericarditis, particularly in asymmetrical forms; the etiological challenge of tuberculosis when classic clinical and microbiological signs are absent; and the therapeutic challenge of initiating empiric treatment in high-risk patients. Multimodal imaging and careful clinical judgment were pivotal in guiding management.

Conclusion: In endemic regions, tuberculous pericarditis should remain a key consideration even in the absence of systemic symptoms or positive cultures. Asymmetrical constriction may limit the completeness of invasive hemodynamic assessments. Empirical anti-tuberculosis therapy, guided by epidemiological context and imaging findings, can be life-saving, highlighting the need for individualized and multidisciplinary management strategies.

Keywords

Asymmetrical constriction; Tuberculous pericarditis; Right heart failure; Multimodality imaging

Résumé

Introduction : La péricardite constrictive est une affection rare et potentiellement mortelle. Son diagnostic peut être difficile, en particulier lorsque la constriction est asymétrique, ce qui limite la sensibilité des évaluations hémodynamiques invasives. La tuberculose demeure une cause majeure dans les zones d'endémie, mais sa présentation peut être subtile et les signes systémiques parfois absents.

Présentation du cas : Nous rapportons le cas d'un patient de 45 ans présentant des signes d'insuffisance cardiaque droite et un épanchement péricardique cloisonné de taille modérée à importante. L'imagerie a révélé une constriction prédominante de la paroi libre du ventricule droit, sans caractéristiques classiques de tuberculose au scanner thoraco-abdomino-pelvien, ni à l'analyse microbiologique (y compris cultures d'expectorations et d'urines). Compte tenu du risque épidémiologique élevé et de l'impossibilité de réaliser une péricardiocentèse en raison du caractère cloisonné de l'épanchement, un traitement antituberculeux empirique (schéma HRZE : isoniazide, rifampicine, pyrazinamide et éthambutol) a été instauré.

Discussion : Ce cas met en évidence plusieurs défis : le défi diagnostique lié à l'identification d'une péricardite constrictive, notamment dans ses formes asymétriques ; le défi étiologique posé par la tuberculose lorsque les signes cliniques et microbiologiques classiques sont absents ; et le défi thérapeutique d'instaurer un traitement empirique chez des patients à haut risque. L'imagerie multimodale et une évaluation clinique minutieuse ont été déterminantes dans la prise en charge.

Conclusion : Dans les régions d'endémie, la péricardite tuberculeuse doit rester un diagnostic clé, même en l'absence de symptômes systémiques ou de cultures positives. La constriction asymétrique peut limiter la valeur des évaluations hémodynamiques invasives. Le traitement antituberculeux empirique, guidé par le contexte épidémiologique et les données d'imagerie, peut être vital, soulignant la nécessité d'une prise en charge individualisée et multidisciplinaire.

Mots-clés

constriction asymétrique, péricardite tuberculeuse, insuffisance ventriculaire droite, imagerie multimodale

Correspondance

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HISTORY OF PRESENTATION

Constrictive pericarditis (CP) remains a challenging clinical entity, characterized by impaired diastolic filling due to a non-compliant pericardium. Although typically described as a diffuse process, localized or asymmetric forms may present with subtle, atypical, or discordant multimodality imaging findings. In regions where tuberculosis remains endemic, CP continues to be an important diagnostic consideration, yet its presentation may be heterogeneous and occasionally misleading.

CASE PRESENTATION

A 45-year-old man with no known cardiovascular risk factors presented with progressive dyspnea accompanied by right upper quadrant abdominal pain.

Physical examination revealed clear signs of right-sided heart failure, including jugular venous distention, a positive hepatojugular reflux, and hepatic congestion, without evidence of left-sided heart failure. Laboratory evaluation showed mild hepatic enzyme abnormalities, with total and direct bilirubin levels of 15 and 5 mg/L, respectively, alkaline phosphatase at 90 U/L, gamma-glutamyl transferase at 39 U/L, and AST/ALT at 49/58 U/L. Hematologic, renal, and electrolyte parameters were otherwise normal. C-reactive protein was negative at 2 mg/L, and the troponin level was also negative.

Transthoracic echocardiography demonstrated a non-dilated left ventricle with preserved systolic function (LVEF 55%) but impaired global longitudinal strain (-14.3 %). Right ventricular systolic function was reduced, with S' at 6 cm/s, TAPSE at 10 mm, and markedly decreased lateral RV strain at –5.8%, while septal strain was preserved. Three-dimensional RV ejection fraction was reduced to 32.2% (Figure 1).

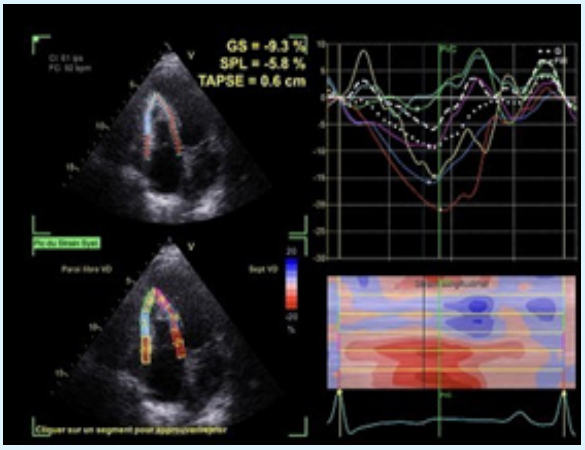
Despite a preserved LVEF of 50%, cardiac output was low, as reflected by a reduced pulmonary and LV outflow tract VTI of 9.5 cm. Cardiac chambers were of normal size, and there was no significant valvular pathology.

A loculated pericardial effusion was visualized along the right ventricular free wall, containing heterogeneous echo densities and producing mass effect on the RV free wall (Figure 2). The inferior vena cava was dilated at 22 mm with reduced respiratory variation. The mitral inflow pattern showed an E/A ratio of 1.14 without respiratory variations (Figure 3).

Echocardiographic features suggestive of constrictive pericarditis included a septal bounce, annulus reversus with medial E' velocity (13.3 cm/s) exceeding lateral E' (11.8 cm/s), annulus paradoxus with preserved lateral E' at 11.8 cm/s despite diastolic dysfunction (Figure 4), and a "hot septum" sign with strain reversus (decreased lateral strain compared with septal strain as mentioned in figure 5A). The myocardial work index was reduced at 1111 mmHg, with an efficiency of 97%, but exhibited marked asymmetry between the septal and lateral walls, characterized by higher constructive work in the septal wall (Figure 5 B-C).

Respiratory flow variation and hepatic vein expiratory diastolic reversal were absent (Figure 6). Cardiac MRI showed a moderate-to-large, loculated pericardial fluid collection along the inferior and free walls of the right ventricle, causing significant RV compression. The collection exhibited low T2 signal intensity and high T1 signal intensity, indicative of a chronic, proteinaceous, or partly organized effusion. Mild flattening of the interventricular septum was noted. Overall, biventricular systolic function was mildly reduced (LVEF 47%, RVEF 42%) with no regional wall-motion abnormalities. There was no late gadolinium enhancement; both T2 mapping (53 ms) and native T1 mapping (1080 ms) were within normal ranges, suggesting no active inflammation or fibrosis. Cardiac CT confirmed these findings, showing a spontaneously hyperdense pericardial effusion with linear and punctate calcifications, consistent with a chronic, organized, partially calcified pericardial collection. Given the absence of significant respiratory variation in transvalvular Doppler flows and the absence of ventricular interdependence on imaging, invasive hemodynamic assessment was pursued. Right heart catheterization demonstrated a classic dip-and-plateau pattern in the right ventricle, confirming constrictive physiology. Respiratory ventricular interdependence was absent, supporting the hypothesis of asymmetrical constriction mainly affecting the RV free wall, rather than uniform circumferential pericardial involvement (Figure 7).

Etiological workup excluded HIV infection, purulent pericarditis, neoplastic causes, autoimmune or systemic disease, and post-surgical or post-radiation pericarditis. Tuberculosis remained the leading suspected etiology given the epidemiological context.



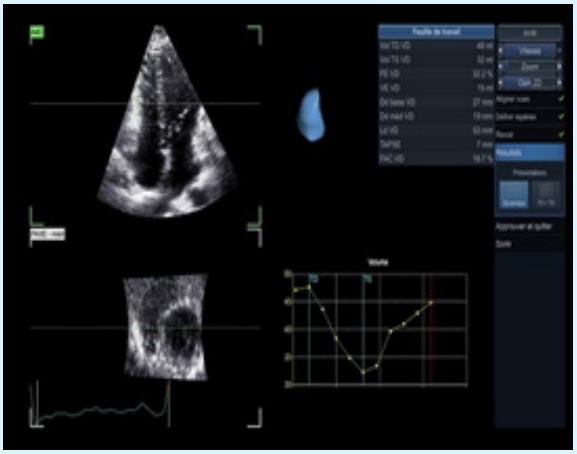


Figure 1 (A-B). Reduced right ventricular systolic function

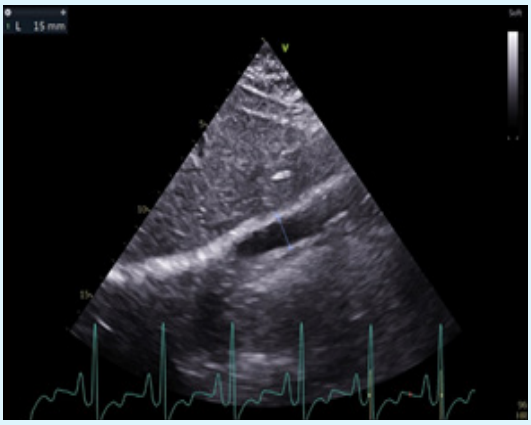
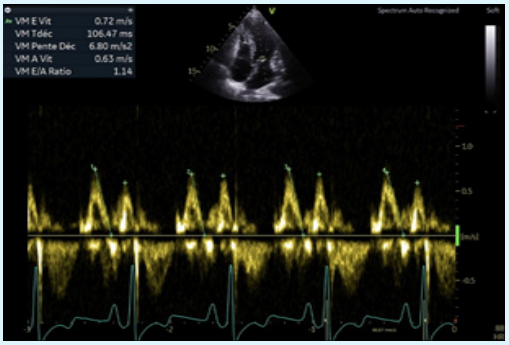


Figure 2. Loculated pericardial effusion along the right ventricular free wall



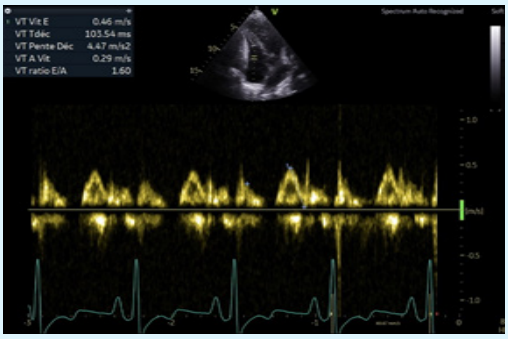
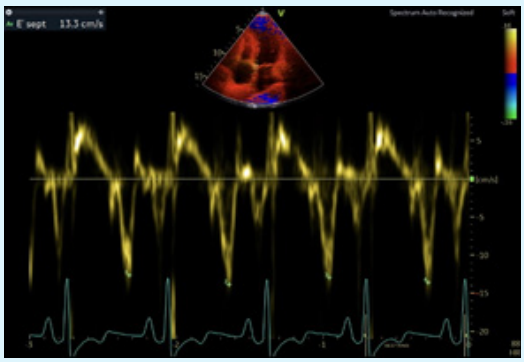


Figure 3 (A-B). Transmitral and Tricuspid Inflows: Type II Pattern Without Respiratory Variation



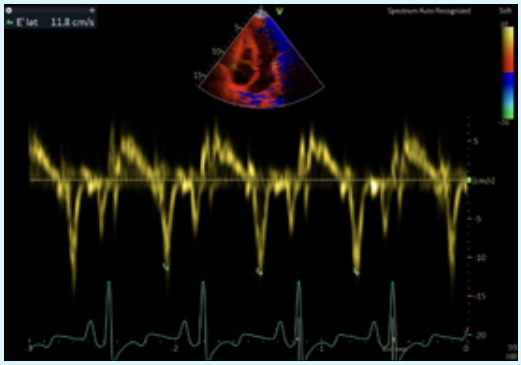


Figure 4. Mitral Annular Velocity Abnormalities: Annulus Reversus (A) and Annulus Paradoxus (B)

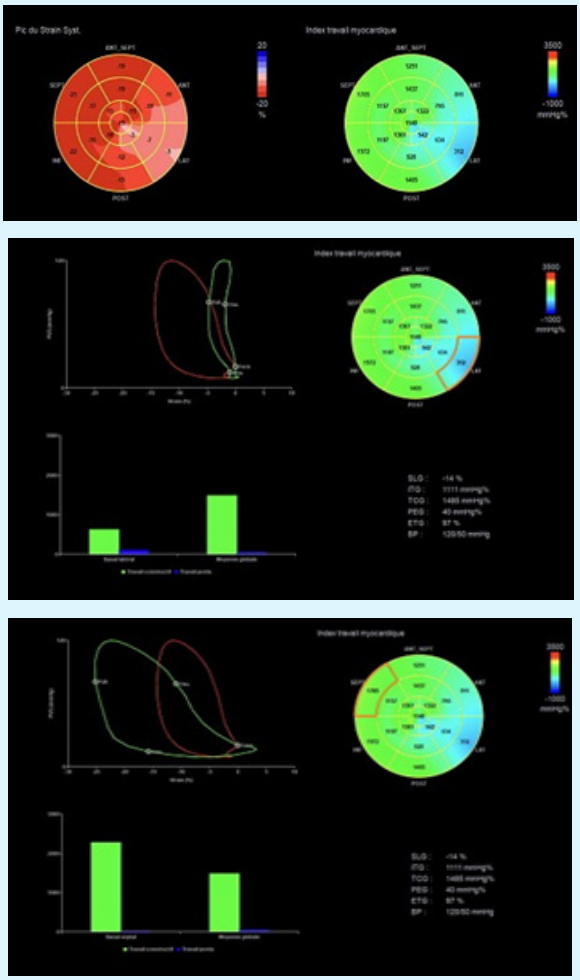


Figure 5 (A). Hot septum sign and strain reversus; (B-C) Reduction of myocardial work in lateral segments versus preserved septal work.

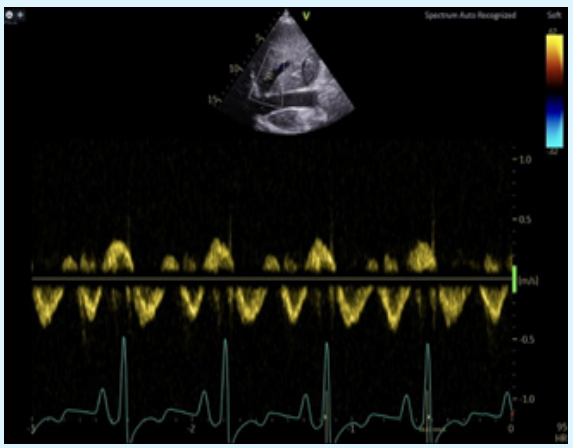


Figure 6. Triphasic sus-hepatic vein flow without expiratory diastolic reversion

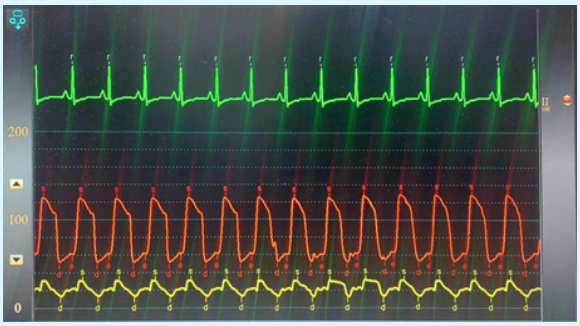


Figure 7. Right heart catheterization demonstrated a classic dip-and-plateau pattern in the right ventricle, without respiratory ventricular interdependence

In our patient, there were no clinical features suggestive of tuberculosis, such as fever, night sweats, or weight loss. Thoraco-abdominal-pelvic CT imaging did not reveal any lymphadenopathy or other findings indicative of tuberculosis. Microbiological investigations, including sputum and urine cultures for acid-fast bacilli, were negative, and both the tuberculin skin test and IFN-γ release assay (IGRA) were also negative.

Pericardiocentesis was not performed due to the deeply loculated, organized, and partially calcified nature of the effusion, making percutaneous access unsafe. The patient was started empirically after excluding other causes on a standard anti-tuberculous regimen (HRZE: isoniazid, rifampicin, pyrazinamide, and ethambutol, 4 Pills per day), but no clinical or echocardiographic improvement was observed after one month. As a result, the patient was referred for surgery: pericardiocentesis.

DISCUSSION

Definition and physiopathology

Pericardial constriction is a chronic condition characterized by a thickened, fibrotic, and sometimes calcified pericardium, resulting in impaired diastolic filling . (1)

Constrictive pericarditis results in a fixed intrapericardial volume, which restricts diastolic filling and leads to equalization of right- and left-sided filling pressures. The key feature is significant ventricular interdependence: during inspiration, decreased transmission of intrathoracic pressure to the heart reduces left-sided filling and shifts

the septum toward the LV, while increased venous return enhances RV filling. The opposite occurs during expiration. This marked respiratory variation explains Kussmaul's sign, hepatic vein flow reversals, and the characteristic discordance of ventricular systolic pressures. Reduced pericardial compliance results in rapid early filling followed by abrupt cessation, producing the classic "square root" sign. Despite high diastolic pressures, stroke volume is decreased due to impaired preload.(2)

Etiological diagnosis

The probability of developing constriction varies according to the underlying pathology. Bacterial pericarditis (particularly purulent forms) shows the highest rate of progression, estimated at 20%–30%. Immune-mediated and malignant pericardial diseases carry an intermediate risk of evolution (approximately 2% - 5%), whereas progression from viral or idiopathic pericarditis remains uncommon, with reported rates below 1%. Other major contributors include sequelae of cardiac surgery (11%–37%) and late complications of mediastinal radiotherapy, most often administered for breast cancer or Hodgkin lymphoma (9%–31%). Less frequently, connective tissue diseases (3%–7%). (1)

In the developed world, idiopathic and post-cardiac surgery etiologies are most common, followed by radiation pericarditis. Tuberculosis remains the most common cause of CP in the developing world, with a poor prognosis and a high mortality rate. (3)

Tuberculosis should therefore remain a key consideration in the differential diagnosis of effusive or constrictive pericarditis, especially in individuals with predisposing conditions or epidemiological risk factors. This includes patients from regions with high tuberculosis endemicity, those receiving immunosuppressive or biologic therapies, individuals living with HIV, and patients with chronic renal failure, particularly those on dialysis. (4)

Tuberculous pericarditis (TBP) is a rarely seen form of extrapulmonary TB, accounting for 1–2% of all TB infections. Isolated pericardial tuberculosis is extremely rare in the literature. (5) 6)

In patients with atypical clinical presentation, it can lead to misdiagnosis and delayed treatment. A negative laboratory test cannot exclude tuberculosis infection. The mortality rate of TBP 6 months after diagnosis is 17–40%. The current treatment for TB pericarditis is a "quadruple" regimen of rifampin, isoniazid, pyrazinamide, and ethambutol for 2 months, followed by isoniazid and rifampin for 4 months. (7) (8)

Etiological diagnosis

Transthoracic echocardiography (TTE) is the imaging modality of choice for the initial evaluation of constrictive pericarditis (CP) and effusive–constrictive pericarditis (ECP). (9)

Differentiation of constrictive pericarditis (CP) from restrictive cardiomyopathy (RCM) is a complex and often challenging process. Although different regarding etiology, prognosis, and treatment, CP and RCM share a common clinical presentation of predominantly right-sided heart failure, in the absence of significant left ventricular systolic dysfunction or valve disease, due to impaired ventricular diastolic filling. (10)

Pericardial thickening can be detected on transthoracic echocardiography, although interpretation may be challenging. Assessment of ventricular septal motion in M-mode and two-dimensional imaging demonstrates ventricular interdependence, with leftward septal motion during inspiration and rightward shift during expiration, often accompanied by a "septal bounce," reflecting rapid early filling abruptly halted by pericardial constraint. (11)

Doppler evaluation is central for distinguishing CP from RCM. Mitral and tricuspid inflows show early diastolic predominance with short deceleration times, but respirophasic flow variation is characteristic of CP and absent in RCM. Hepatic vein Doppler often demonstrates expiratory diastolic flow reversal. Mitral annular tissue Doppler imaging provides a highly discriminative parameter: lateral early diastolic velocity (e') is normal or paradoxically increased despite elevated filling pressures known as "annulus paradoxus", while medial e' often exceeds lateral e', a phenomenon known as "annulus reversus." (11) (12)

A typical echocardiographic global longitudinal strain (GLS) pattern has been described in in patients with constrictive pericarditis (CP): a characteristic reduction in the regional longitudinal strain of the left ventricular free wall and relative sparing of the septal longitudinal strain value which creates the so-called "hot-septum sign" at the bullseye plot and is described by some authors as "strain reversus". This

reduction in the free wall strain appears to correlate with pericardial thickness, and it is likely explained by pericardial adhesions as well as involvement of adjacent myocardium. (13) (14)

Cardiac magnetic resonance (CMR) represents a valuable complementary imaging modality in the evaluation of constrictive pericarditis, offering high-resolution anatomical characterization as well as precise assessment of pericardial inflammation. Late gadolinium enhancement (LGE) is frequently observed and reflects chronic inflammatory activity, including fibroblast proliferation and neovascularization. Both qualitative and quantitative analyses of LGE provide incremental diagnostic information beyond clinical evaluation and inflammatory biomarkers, and have been shown to predict the likelihood of response to anti-inflammatory therapy in constrictive pericarditis.(15)

Inflammatory constrictive pericarditis (LGE+, T2-STIR+, high CRP) is potentially reversible with anti-inflammatory therapy, whereas non-inflammatory fibrotic constriction (LGE–, calcifications, normal CRP) generally requires pericardiectomy. (15)

Cardiac computed tomography (CT) serves as a complementary imaging technique, particularly useful for evaluating pericardial thickness and detecting pericardial calcifications. In the preoperative setting, it also provides valuable information regarding the pericardium's anatomical relationships with adjacent structures before pericardiectomy. (9)

In our case, CT imaging was particularly valuable because isolated cardiac tuberculosis is uncommon. The scan allowed us to search for additional extracardiac tuberculous lesions, thereby reinforcing the etiological work-up and supporting the suspicion of tuberculous pericardial involvement.

Cardiac catheterization

Still considered the gold standard diagnostic test for constrictive pericarditis, cardiac catheterization with hemodynamic assessment is performed when noninvasive studies are nondiagnostic or are considered insufficiently conclusive. (16)

Expected 'classic' findings include (16):

- Elevated central venous pressure and intracardiac pressures, with near equalisation of right and left heart diastolic filling pressures.
- A 'dip and plateau' or 'square root' sign in the RV pressure tracing, both signifying rapid early diastolic filling of the RV with abrupt cessation due to pericardial constraint.
- RV systolic pressure that is < 50 mmHg
- RV end-diastolic pressure that is at least one-third of the RV systolic pressure.
- dissociation of intrathoracic and intracardiac pressures and enhanced ventricular interaction
- Simultaneous recordings of the RV and LV pressure waveforms reveal discordant variation of the waveforms during the respiratory cycle

Compared to generalized constrictive pericarditis, localized constrictive pericarditis is less common and more difficult to identify. (17)

In our case, the absence of respiratory variation in ventricular filling can be attributed to the non-uniform distribution of pericardial constriction. (17) Unlike classic constrictive pericarditis, where the rigid pericardium restricts all cardiac chambers uniformly, here the constrictive process is asymmetrical, predominantly affecting the right ventricle. As a result, ventricular interdependence and equalization of diastolic pressures are not apparent, since the left ventricle remains relatively free to expand during respiration. The diastolic compression is localized to the right ventricle, producing hemodynamic limitation and a "dip and plateau" pattern on catheterization, while the typical respirophasic septal shift and mitral inflow variation are absent.

Treatment

Initial therapy should target the underlying etiology, where applicable. Where pericardial inflammation is identified (C-reactive protein elevation or pericardial enhancement on CMR), potent anti-inflammatory therapy should be initiated before consideration of surgical intervention. Pericardiectomy should be considered in symptomatic patients without evidence of pericardial inflammation or who fail anti-inflammatory therapy, and performed by experienced pericardial surgeons. (18)

In a pilot study of 29 patients with constrictive pericarditis (CP) were treated with anti-inflammatory therapy following CMR evaluation. After a median follow-up of 13 months, CP resolved in 14 patients, whereas 15 showed persistent constriction. Reversible CP was

characterized by more pronounced inflammatory features at baseline, including greater pericardial thickness on LGE sequences, higher qualitative LGE intensity, and elevated systemic inflammatory markers (C-reactive protein and erythrocyte sedimentation rate). An LGE-derived pericardial thickness ≥3 mm demonstrated good diagnostic performance for predicting reversibility. (19)

Tuberculous pericarditis should be strongly suspected in patients presenting with pericardial disease in an endemic area.

According to the 2025 ESC recommendations, diagnostic pericardiocentesis is advised for all patients with suspected tuberculous pericarditis when non-invasive investigations fail to establish the diagnosis, in order to identify the etiological agent directly from the pericardial fluid. In endemic regions, empirical antituberculosis therapy is recommended in patients presenting with an exudative pericardial effusion once alternative causes have been reasonably excluded, as tuberculosis remains the most likely etiology. Furthermore, pericardiectomy should be considered in tuberculous pericarditis when clinical status fails to improve or deteriorates despite 4–8 weeks of adequate antituberculosis treatment, aiming to alter the disease course. (1)

In our patient, pericardiocentesis was not performed because the pericardial effusion was loculated and partially calcified, making percutaneous drainage technically difficult and potentially hazardous. In such settings, the risk of incomplete drainage or procedural complications outweighs the diagnostic benefit, particularly when the effusion is compartmentalized and not freely accessible. This anatomical configuration, therefore, limited the role of invasive sampling and justified relying on clinical, imaging, and epidemiological features to support the suspicion of tuberculous pericardial involvement.

Adjunctive corticosteroid therapy is generally considered in HIV-negative patients with tuberculous pericarditis to reduce inflammation and prevent progression to constrictive disease. (1) In our case, however, this therapeutic window had already passed, as the patient presented with an established constrictive physiology at the time of evaluation. The presence of fixed, non-compliant pericardial thickening and calcifications indicated a chronic, irreversible stage in which steroids are unlikely to modify the disease course.

In a cohort of 97 patients undergoing surgery for constrictive pericarditis, early mortality was independently associated with impaired preoperative cardiac function, specifically reduced left ventricular ejection fraction and right ventricular dilatation. Long-term outcomes were primarily influenced by comorbid conditions, with coronary artery disease, chronic obstructive pulmonary disease, and preoperative renal insufficiency emerging as significant predictors of late mortality. Notably, concomitant tricuspid valve repair, if indicated, appeared to confer a protective effect on long-term survival. (20)

Figure 8 illustrates an algorithm outlining the diagnostic approach, etiological assessment, and therapeutic strategy in constrictive pericarditis.

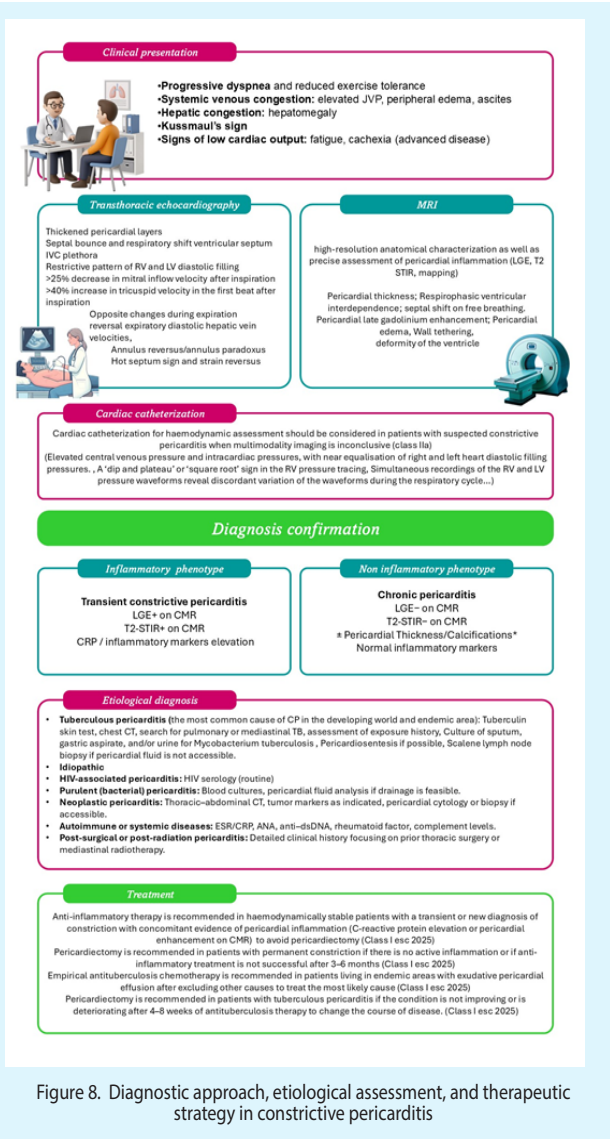


Figure 8. Diagnostic approach, etiological assessment, and therapeutic strategy in constrictive pericarditis

CONCLUSION

This case illustrates the multiple challenges encountered in managing pericardial disease. First, the diagnostic challenge lies in recognizing constrictive pericarditis, especially when clinical signs are subtle or atypical. Second, the etiological challenge requires careful evaluation to exclude infectious causes such as tuberculosis, particularly in endemic regions, as well as other inflammatory or neoplastic etiologies. Third, the therapeutic challenge involves timely decision-making regarding medical treatment and interventions and, ultimately, pericardiectomy for definitive management. Awareness of these challenges and a multidisciplinary approach are essential to optimize outcomes in patients with complex pericardial disease.

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