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Large Pericardial Effusion in Transthyretin Cardiac Amyloidosis without Hemodynamic Compromise: A Case Report

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Abstract **Case Report**

Background: Pericardial effusion is recognized in cardiac amyloidosis, although a large collection raises questions about its cause and clinical significance. ***Case presentation:*** A 69-year-old man with transthyretin cardiac amyloidosis investigated by bone-avid tracer scintigraphy showing grade 3 myocardial uptake and a reportedly negative AL amyloidosis work-up, atrial fibrillation treated with rivaroxaban, and a history of surgically managed traumatic subdural hematoma presented after six weeks of worsening exertional dyspnea, fatigue, and weight loss. He was clinically stable (blood pressure 100/67 mmHg; heart rate 70 beats/min), despite jugular venous distension. Echocardiography showed increased left ventricular wall thickness, an ejection fraction of 30–35%, grade III diastolic dysfunction, right-sided chamber enlargement, and a predominantly posterior pericardial effusion measuring 30 mm with right atrial indentation. Approximately 300 mL of straw-colored fluid was drained, and a pericardial biopsy was obtained. Histology revealed chronic changes without malignancy; fluid cytology was reactive and GeneXpert was negative. A small residual effusion and persistent dyspnea were noted after the procedure. ***Discussion:*** Neither the size of the effusion nor right atrial indentation alone establishes tamponade. Although the effusion occurred in the setting of ATTR cardiomyopathy, direct pericardial amyloid involvement was not demonstrated. ***Conclusion:*** Large pericardial effusion in ATTR cardiomyopathy warrants careful hemodynamic assessment and an independent search for treatable causes.

Keywords: transthyretin amyloidosis; cardiac amyloidosis; pericardial effusion; echocardiography; case report.

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INTRODUCTION

Transthyretin amyloid cardiomyopathy (ATTR-CM) is increasingly recognized among patients with unexplained ventricular wall thickening, heart failure, and atrial arrhythmias. Pericardial fluid may be present, but a substantial effusion presents a different clinical question: is it a consequence of the underlying cardiomyopathy, an unrelated process, or a contributor to the patient's symptoms? The distinction matters because an infiltrative, poorly compliant heart may tolerate additional constraints on filling poorly, even before classical signs of tamponade appear. We describe a patient with established ATTR-CM and a large posterior effusion in whom drainage and tissue sampling clarified some, but not all, aspects of the diagnosis.

CASE PRESENTATION

A 69-year-old man with transthyretin cardiac amyloidosis diagnosed in December and atrial fibrillation treated with rivaroxaban presented with progressively worsening dyspnea over six weeks (NYHA functional class III), fatigue, and weight loss. He denied fever and chest pain. His history included surgically treated traumatic subdural hematoma in March 2025. Reported maintenance medications were tafamidis 61 mg/day, an SGLT2 inhibitor, rivaroxaban, colchicine, spironolactone, carvedilol, and furosemide. Bone-avid tracer scintigraphy had shown grade 3 myocardial uptake on the Perugini scale, strongly supporting ATTR-CM. The clinical team reported negative testing for AL amyloidosis; individual assay results were not available for review.

At presentation, blood pressure was 100/67 mmHg, heart rate 70 beats/min, and oxygen saturation 95% breathing ambient air. Jugular venous distension and a positive hepatojugular reflux were noted without clinical shock or respiratory distress. Electrocardiography showed atrial fibrillation with a ventricular rate of approximately 70 beats/min and diffuse low QRS voltage, without reported repolarization abnormalities.

Transthoracic echocardiography demonstrated a non-dilated, thick-walled left ventricle, left ventricular ejection fraction 30–35%, and grade III diastolic dysfunction. The right atrium and right ventricle were enlarged with restrictive filling physiology. A predominantly posterior pericardial effusion measured up to 30 mm, with right atrial indentation. Detailed echocardiographic indices of tamponade physiology were unavailable.

Laboratory findings included creatinine 11.08 mg/L (estimated glomerular filtration rate 65 mL/min), sodium 131 mmol/L, potassium 4.36 mmol/L, C-reactive protein 10.3 mg/L, and thyroid-stimulating hormone 3.29 µIU/mL. Complete blood count was reported as normal. Given the sizeable posterior effusion and the need for etiologic evaluation, drainage was undertaken; approximately 300 mL of straw-colored fluid was evacuated, with concomitant pericardial biopsy. The procedural approach and periprocedural anticoagulation strategy were not specified.

Pericardial histopathology showed chronic remodeling without malignancy. Fluid cytology demonstrated a reactive mesothelial inflammatory response; GeneXpert testing was negative. These findings did not demonstrate amyloid deposits in the pericardium and do not, by themselves, exclude all infectious causes, including tuberculosis. Following drainage, the patient remained hemodynamically stable, with a thin residual effusion on echocardiography but persistent NYHA class III dyspnea. Longer-term follow-up was unavailable.



Figure 2A: Echocardiographic view of the pericardial effusion

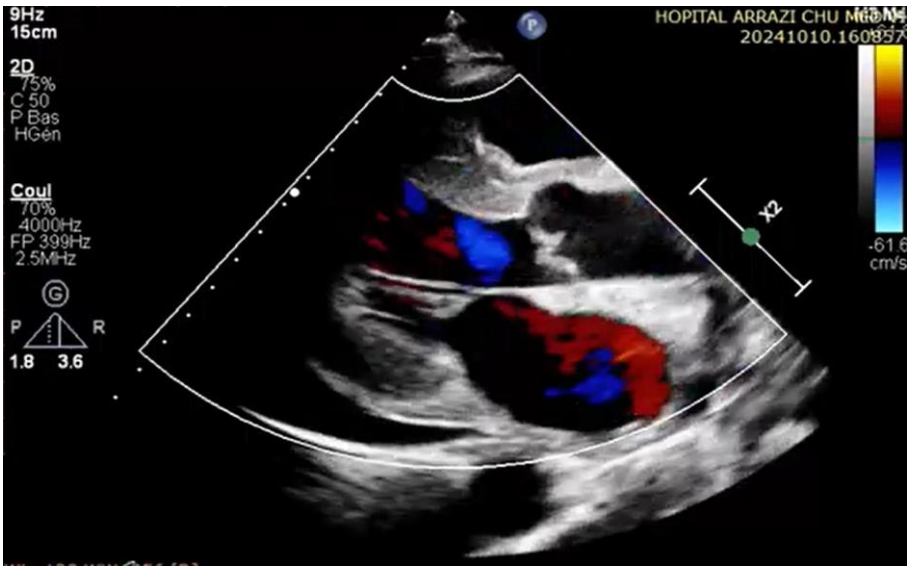


Figure 2B: Additional echocardiographic view with color Doppler

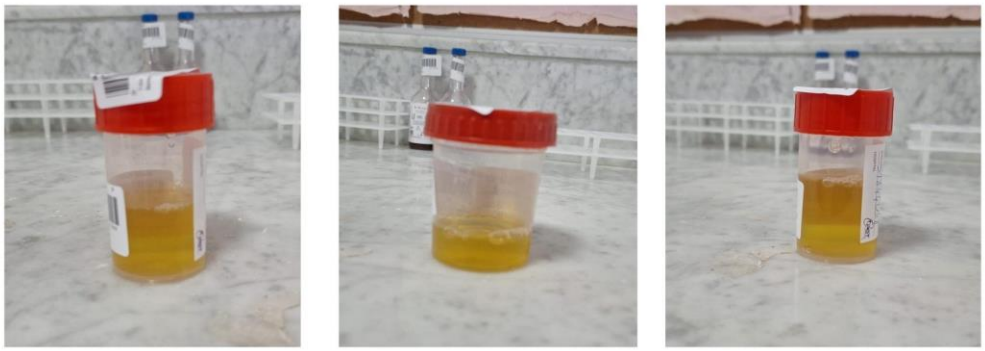


Figure 3: Pericardial fluid samples after drainage

DISCUSSION

The striking feature of this case was the discrepancy between the size of the effusion and the absence of overt circulatory collapse. The maximal echocardiographic separation of 30 mm is consistent with a large effusion [1]. Nevertheless, size is only one component of risk. The speed of fluid accumulation, pericardial compliance, and intracardiac filling pressures determine its hemodynamic consequences. Right atrial indentation warrants attention, but without documented respiratory Doppler variation, inferior vena cava findings, or right ventricular diastolic collapse, tamponade cannot be inferred from this observation alone.

Attributing the effusion to ATTR-CM requires particular caution. Amyloid-related myocardial stiffness and elevated right-sided pressures may contribute to fluid accumulation, and pericardial amyloid deposition is another possible mechanism. Yet these explanations remain hypotheses in the present patient. Cytology and biopsy did not identify malignancy, while the reported negative GeneXpert result provides only limited evidence against tuberculous disease. The absence of reported pericardial Congo red staining also prevents a claim of direct amyloid infiltration. Other infectious, inflammatory, metabolic, and neoplastic causes therefore remain relevant to the differential diagnosis.

The decision to drain a stable patient should be guided by symptoms, suspected physiology, the location and accessibility of the collection, and the need for diagnostic material rather than by an isolated measurement [1]. In this case, the posterior distribution and the use of rivaroxaban were clinically relevant procedural considerations, although the precise drainage approach and periprocedural anticoagulation strategy were not available in the source record. The removal of approximately 300 mL permitted analysis and was followed by a small residual effusion. Persistent NYHA class III dyspnea suggests that the underlying cardiomyopathy and ventricular dysfunction continued to contribute substantially to symptoms.

Finally, the diagnosis and treatment of ATTR-CM must be distinguished from the evaluation of the effusion. Current ACC guidance emphasizes exclusion of a monoclonal protein disorder before a positive bone-avid tracer scan is accepted as noninvasive evidence of ATTR-CM; genetic testing subsequently differentiates variant from wild-type transthyretin disease [2]. The clinical team reported a negative AL amyloidosis work-up (individual serum/urine immunofixation and free light-chain results were not provided). The patient was receiving tafamidis, and bone-avid tracer scintigraphy demonstrated Perugini grade 3 myocardial uptake. However, the monoclonal protein assessment, tracer identity, and TTR genotype were not available. These details remain necessary to substantiate a non-biopsy ATTR diagnosis and classify the disease. The reduced ejection fraction also deserves emphasis: ATTR-CM is often recognized with preserved ejection fraction, but systolic impairment can occur in advanced disease.

Several limitations temper interpretation. We lack the radiotracer identity, detailed monoclonal screening results and TTR genotype, comprehensive pericardial fluid chemistry and cultures, detailed echocardiographic indices of tamponade, the pericardial staining protocol, and a defined duration of follow-up. We also cannot determine whether drainage produced a sustained symptomatic benefit. These limitations restrict causal attribution and assessment of longer-term outcome.

CONCLUSION

This case illustrates why a large pericardial effusion in ATTR-CM should be approached as a separate diagnostic and hemodynamic problem. Clinical stability does not make the finding trivial, but neither does the coexistence of amyloidosis establish its cause. Echocardiographic assessment, targeted investigation for alternative etiologies, and individualized decisions about drainage remain central to management.

Learning Points

- A large (>20 mm) pericardial effusion does not automatically imply cardiac tamponade.
- Pericardial effusion in ATTR-CM should not be attributed to amyloid deposition without supporting evidence.

- Drainage decisions depend on symptoms, hemodynamics, anatomy, and the need for etiologic sampling.
- Document the ATTR diagnostic pathway, amyloid typing, and meaningful longitudinal follow-up.

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