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A 5.5-Liter Paradox: Giant Pericardial Effusion Presenting as Cardiac Tamponade with Reactive CA-125 Elevation

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ABSTRACT

Background: *Cardiac tamponade is a life-threatening oncologic and cardiovascular emergency requiring prompt recognition and immediate drainage. While the pericardium can accommodate slowly accumulating fluid, extreme effusions exceeding 50 mm are exceptionally rare.*

Case Presentation: *We report the case of a 77-year-old female with an intellectual disability and a history of two prior pericardiocenteses who presented with severe dyspnea and hemodynamic collapse (BP: 76/42 mmHg, HR: 125 bpm). Transthoracic echocardiography (TTE) in end-diastole demonstrated a massive, giant pericardial effusion measuring up to 82 mm from the apical long- and short-axis views. Pulsed-wave Doppler revealed profound respiratory variation (>25% across the mitral valve and >40% across the tricuspid valve), confirming overt cardiac tamponade. The inferior vena cava was severely dilated (32 mm) without inspiratory collapse. Emergent pericardiocentesis initially evacuated 1,500 mL of hemorrhagic fluid, culminating in a total of 2 liters drained within the first 24 hours. This provided immediate hemodynamic relief without any subsequent deterioration in cardiac function. Over the course of the hospital stay, continuous catheter drainage yielded a remarkable cumulative total of 5.5 liters. Thoracic CT identified a pulmonary mass and mediastinal lymphadenopathy. Serum CA-125 was significantly elevated; surprisingly, repeated pericardial fluid cytological examinations showed no malignant cells. Tissue biopsy could not be obtained due to patient refusal. A surgical pericardial window was created, and the patient was discharged in a stable condition.*

Conclusion: *This case highlights that giant effusions (82 mm) can induce massive pericardial distension and that elevated serum CA-125 levels in such settings may stem from mesothelial mechanical stretch rather than direct tumor secretion. It also reinforces the critical utility of quantitative PW-Doppler and comprehensive imaging in validating tamponade hemodynamics and navigating diagnostic dilemmas in recurrent effusions despite negative cytology.*

Keywords

Cardiac tamponade, Giant pericardial effusion, CA-125, Negative cytology, Pericardiocentesis, Echocardiography.

Introduction

Pericardial effusion involves the abnormal accumulation of fluid within the pericardial space secondary to various systemic or local etiologies. The clinical presentation is primarily dictated by the volume and the rate of fluid accumulation. While slowly developing effusions may remain asymptomatic even upon reaching 2 to 3 liters, the rapid accumulation of merely 200-300 mL can precipitate cardiac tamponade [1]. Malignancies—particularly lung cancer, breast cancer, and lymphomas—are among the most prominent causes of large pericardial effusions [2,3]. Other frequent etiologies include tuberculosis, uremia, autoimmune diseases, and idiopathic origins. Echocardiography serves as the gold standard diagnostic modality; it precisely determines the effusion size, its hemodynamic impact, and the

necessity for urgent pericardiocentesis [4,5]. Herein, we discuss the diagnostic and therapeutic management of a patient presenting with an exceptionally rare, giant pericardial effusion measuring 82 mm in depth. The case is further characterized by a complex clinical picture involving an occult pulmonary mass, elevated CA-125, and paradoxically negative fluid cytology.

Case Presentation

A 77-year-old female patient with an intellectual disability and a significant past medical history of two prior pericardiocenteses for recurrent large pericardial effusions was brought to the emergency department via emergency medical services. Her primary complaints were general clinical deterioration and severe progressive dyspnea. Upon admission, her vital signs revealed profound cardiogenic shock with severe hypotension (blood pressure: 76/42 mmHg) and sinus tachycardia (heart rate: 125 beats/min).

Initial Investigations

A 12-lead electrocardiogram (ECG) demonstrated sinus tachycardia (HR ~110 bpm) and widespread low QRS voltage, most prominently in the limb leads (QRS amplitude < 5 mm). Crucially, classical electrical alternans—characterized by beat-to-beat variation in the QRS amplitude—was clearly visible across the precordial leads (e.g., V2–V5) (Figure 1). Physical examination was notable for elevated jugular venous pressure (JVP), distant and muffled heart sounds, right upper extremity lymphedema, and a congenital anomaly in the right lower extremity.

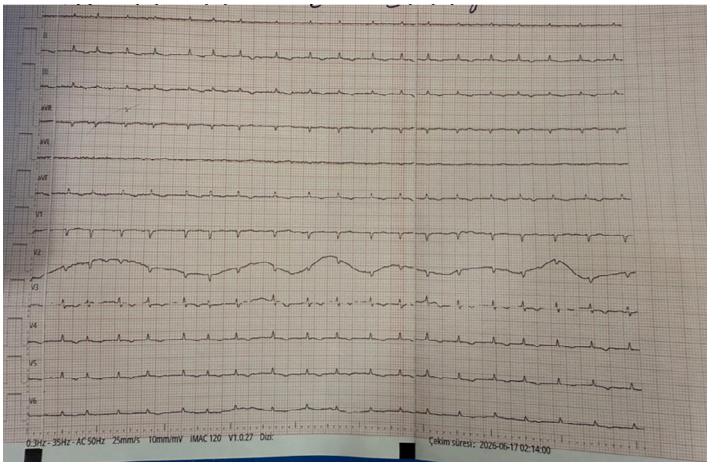
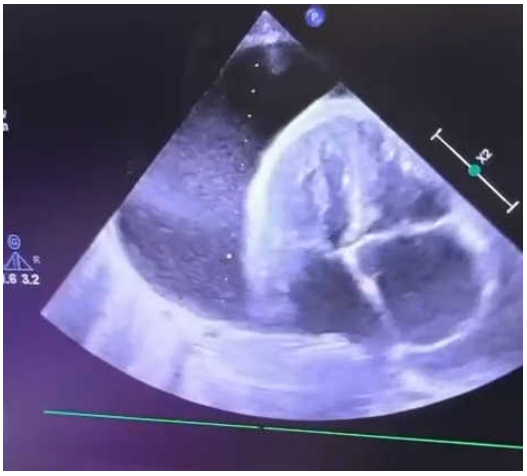


Figure 1: 12-lead electrocardiogram at initial clinical presentation. The tracing demonstrates sinus tachycardia and widespread low QRS voltage, most prominently in the limb leads (QRS amplitude < 5 mm), which is consistent with the massive insulating fluid volume. Notably, classical electrical alternans—characterized by beat-to-beat variation in the QRS amplitude—is clearly visible across the precordial leads (e.g., V2–V5). This electrical phenomenon perfectly correlates with the mechanical "swinging heart" observed on echocardiography.

Echocardiographic and Hemodynamic Assessment

Emergent bedside two-dimensional transthoracic echocardiography (TTE) was performed. Apical long- and short-axis views in end-diastole revealed a massive, circumferential, anechoic pericardial effusion measuring an extraordinary 82 mm at its maximum depth. The heart exhibited the classic "swinging heart" phenomenon (Supplementary Video 1). Echocardiographic signs of right ventricular early diastolic collapse and right atrial late systolic inversion were prominent.

To definitively confirm the hemodynamic compromise, pulsed-wave (PW) Doppler analysis was performed, revealing classic pulsus paradoxus hemodynamics: a >25% respiratory variation in mitral inflow velocity and a >40% variation in tricuspid inflow velocity. Furthermore, the inferior vena cava (IVC) was severely dilated, measuring 32 mm in diameter, with a complete absence of inspiratory collapse (plethoric IVC). This finding indicated profoundly elevated right atrial pressure, cementing the diagnosis of severe cardiac tamponade. Notably, underlying biventricular systolic function appeared preserved.



Supplementary Video 1: Bedside two-dimensional transthoracic echocardiography (apical 4-chamber view) demonstrating the massive, circumferential anechoic pericardial effusion. The prominent "swinging heart" phenomenon is clearly visualized, vividly illustrating the mechanical basis for the electrical alternans observed on the patient's electrocardiogram, alongside impending right-sided chamber collapse indicative of severe cardiac tamponade.

Intervention and Clinical Course

The patient was immediately transferred to the coronary intensive care unit, where an emergent, echocardiography-guided pericardiocentesis was successfully performed. An initial volume of 1,500 mL of hemorrhagic fluid was actively drained, culminating in a total of 2 liters evacuated within the first 24 hours. This rapid yet controlled decompression resulted in immediate hemodynamic stabilization. Remarkably, despite the massive fluid shifts, the patient exhibited no deterioration in underlying cardiac function. A pericardial pigtail catheter was left in situ for continuous management. Over the subsequent days, daily fluid drainage was meticulously monitored, ultimately yielding an extraordinary cumulative volume of 5.5 liters before the effusion completely resolved.

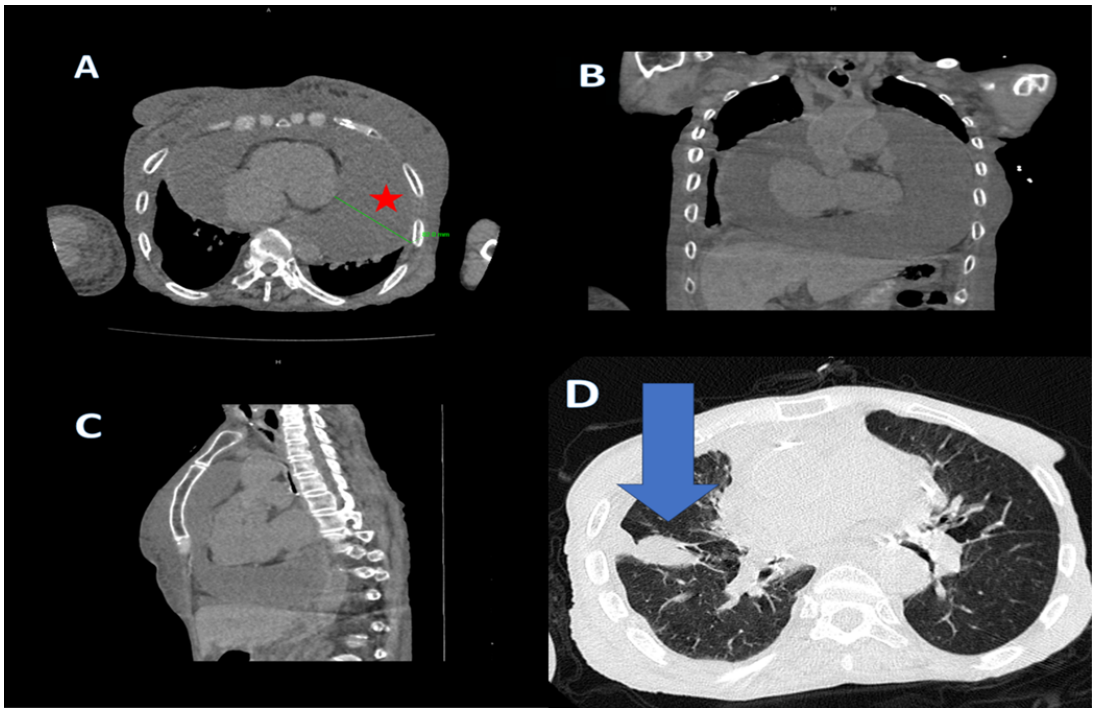


Figure 2: Radiologic evaluation of the giant pericardial effusion and the suspected primary pulmonary malignancy. **(A)** Axial non-contrast thoracic computed tomography (CT) soft-tissue window demonstrating a massive, circumferential pericardial effusion with a maximum depth of 82.0 mm (green line) compressing the cardiac chambers. **(B)** Coronal and **(C)** sagittal reformatted CT images highlighting the extreme distension of the pericardial sac and the resultant mass effect within the mediastinum. **(D)** Axial high-resolution CT (HRCT) lung window revealing a highly suspicious, irregular solid mass in the right lung (arrow), highlighting the diagnostic dilemma of an occult malignancy despite repeated negative pericardial fluid cytology.

A comprehensive etiological work-up was initiated. Pan-CT imaging revealed a massive pericardial effusion compressing the cardiac chambers and a highly suspicious pulmonary mass with associated mediastinal lymphadenopathy (Figure 2).

Laboratory and Pathological Findings

Interestingly, laboratory investigations revealed significantly elevated serum CA-125 levels. However, comprehensive cytopathological examination of the drained hemorrhagic fluid was entirely negative for malignant or atypical cells, demonstrating only a reactive/inflammatory pattern. Although the clinical and radiological picture was highly suggestive of an underlying primary lung malignancy (such as lung adenocarcinoma), definitive histopathological diagnosis was precluded by the patient's refusal to undergo a tissue biopsy of the pulmonary mass.

Following the complete resolution of the effusion by the tenth day of hospitalization, a surgical pericardial window was performed to prevent future recurrence. The patient was discharged in a hemodynamically stable condition without evidence of decompression-associated pulmonary edema, and referred for outpatient oncology and cardiology follow-up.

Discussion

This case vividly demonstrates the extreme adaptive reserve capacity of the pericardial sac in an elderly patient before culminating in life-threatening cardiac tamponade. In the current cardiovascular literature, pericardial effusions reaching an absolute measurement of 82 mm in diastole—and ultimately accommodating an unprecedented 5.5 liters of fluid—are exceedingly rare. Most major clinical registries define "large" effusions simply as >20 mm. The development of overt tamponade despite this immense volumetric accommodation highlights the nonlinear relationship between pericardial compliance and intrapericardial pressure [1].

Furthermore, this case presents a compelling diagnostic dilemma commonly encountered in cardio-oncology. The presence of a massive, recurrent hemorrhagic effusion alongside a pulmonary mass strongly points toward a malignant etiology, which accounts for 10-20% of all large effusions [2,7]. However, the pericardial fluid cytology in our patient was repeatedly negative. It is well-documented that the sensitivity of pericardial fluid cytology for detecting malignancy is roughly 70-75%, meaning false-negative results are not uncommon, especially if the pericardial involvement is paramalignant or if tumor cells are localized in epicardial nodules rather than floating freely in the exudate [8].

Adding to the complexity was the prominent elevation of serum CA-125. While CA-125 is traditionally viewed as an ovarian tumor marker, it is essentially a glycoprotein expressed by mesothelial cells (including the pericardium, pleura, and peritoneum). In the context of an 82-mm giant effusion, the extreme mechanical

stretch, immense wall stress, and severe inflammation of the pericardial layers can trigger massive reactive secretion of CA-125, irrespective of actual malignant cell invasion. Therefore, elevated CA-125 in the presence of extreme pericardial distension should be interpreted with caution, as it may reflect mechanical mesothelial stress rather than definitive tumor progression.

Echocardiography remains the unequivocal gold standard for both the diagnosis and therapeutic management of cardiac tamponade [6]. In this case, quantitative TTE (diastolic measurements, >25% mitral and >40% tricuspid inflow respiratory variations, and a non-collapsible 32-mm IVC) provided an immediate, irrefutable indication for life-saving pericardiocentesis. The successful step-wise evacuation of a massive 5.5 liters of fluid in total, remarkably without the subsequent development of decompression-associated pulmonary edema or any deterioration in systolic function, underscores the critical importance of prompt, targeted, and carefully monitored intervention.

Conclusion

This case emphasizes the diagnostic and therapeutic complexities of managing a giant (82 mm), recurrent pericardial effusion. A negative fluid cytology does not entirely rule out an occult malignancy, especially when radiological evidence (pulmonary mass) is present. Moreover, extreme mesothelial stretch caused by massive fluid accumulation can induce reactive elevations in tumor markers such as CA-125, posing a significant diagnostic challenge. Ultimately, comprehensive evaluation—encompassing clinical signs, ECG (electrical alternans), quantitative Doppler echocardiography, and cross-sectional imaging—is paramount in guiding the life-saving decompression of the pericardial space and navigating complex etiological dilemmas.

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