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HEART FAILURE

CASE REPORT: CLINICAL CASE

Fatal Left Ventricular Free Wall Rupture Complicating Acute Myopericarditis

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ABSTRACT

This is a case of a 59-year-old man presenting with myopericarditis. Over a 2-week period, he developed progressive symptoms and worsening pericardial effusion, leading to cardiac tamponade. Pericardiocentesis revealed hemopericardium, and multidetector computed tomography angiography showed left ventricular free wall rupture. The patient collapsed abruptly, and autopsy confirmed the findings. (J Am Coll Cardiol Case Rep 2024;29:102178) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 59-year-old man was referred to our hospital for evaluation and management of cardiac tamponade after a recent diagnosis of acute myopericarditis.

The patient had been admitted to a district hospital 14 days earlier with fever and nonanginal chest pain, and worsening while lying down, while being hemodynamically stable. Electrocardiography (ECG) revealed concave ST-segment elevation and PR-segment depression on inferior leads (**Figure 1A**), suggestive of acute pericarditis. Results of blood testing revealed elevated levels of inflammatory markers and high-sensitivity cardiac troponin I (Supplemental Table 1). Transthoracic echocardiography (TTE) showed preserved left ventricular (LV) systolic function and mild to moderate pericardial effusion (PE) (10.8 mm at end-diastole) (**Figures 2A and 2B**). Multidetector computed tomography angiography (MDCTA) of aorta depicted a small PE with low fluid density, representing transudate, and a limited left pleural effusion (**Figure 2C**). The patient received guideline-based treatment[1] and 10 days later was discharged on colchicine (1 g once daily) and

LEARNING OBJECTIVES

- To formulate a thorough differential diagnostic algorithm of cardiac tamponade causes in the emergency setting.
- To acknowledge that cardiac tamponade may present with relative stability and insidious character up to the point that disruption of equipoise occurs abruptly.
- To recognize ventricular rupture as a rare and serious complication of acute myopericarditis that requires immediate management.
- To identify the importance of different imaging modalities in the emergency setting for accurate decision-making.

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ABBREVIATIONS AND ACRONYMS

ACS = acute coronary syndrome

ECG = electrocardiography

LV = left ventricular

LVFWR = left ventricular free wall rupture

MDCTA = multidetector computed tomography angiography

PE = pericardial effusion

TTE = transthoracic echocardiography

ibuprofen (500 mg twice daily) after clinical and laboratory improvement.

Four days later, the patient was readmitted with progressive dyspnea and hemodynamic compromise, with blood pressure of 95/65 mm Hg and heart rate of 100 beats/min. TTE revealed mildly reduced LV systolic function and increased PE (20.5 mm) with respiratory variation of mitral inflow Doppler velocities (Figure 3), indicatory of cardiac tamponade. Intravenous crystalloid fluids and inotropic support with dobutamine infusion (5 μg/kg per minute) were provided, and the patient was emergently transferred to our hospital for further management.

Upon arrival, the patient was oligosymptomatic in good general condition, while receiving minimal inotropic support. His blood pressure was 120/80 mm Hg, heart rate was 85 beats/min, oxygen saturation was 98% on room air, and respiratory rate was 25 breaths/min. Clinical examination revealed muffled heart sounds, decreased basal breath sounds bilaterally, and jugular vein distention. ECG depicted negative T waves on lateral leads, without PR-segment depression or ST-segment elevation (Figure 1B).

PAST MEDICAL HISTORY

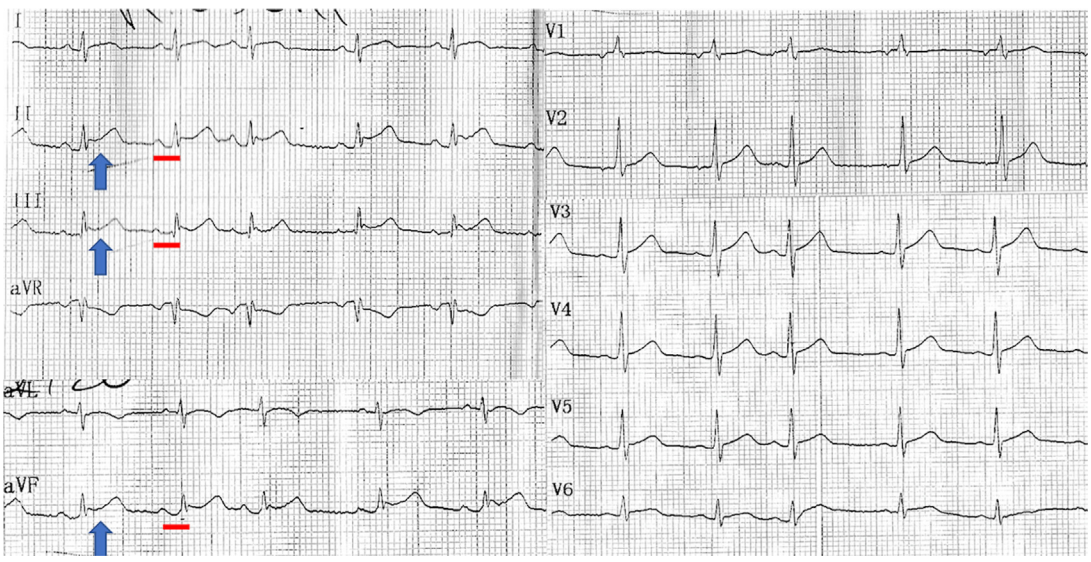
The patient reported no significant past medical history, except heavy smoking (80 pack-years).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of cardiac tamponade causes includes 2 disease categories that direct distinct treatment pathways: 1) mechanical complications, requiring surgical management; and 2) conditions manifesting with pericarditis, requiring interventional management. The first category consists of trauma, iatrogenic trauma, aortic dissection, and heart chamber wall rupture, with the latter commonly complicating acute coronary syndromes (ACS). The second category includes PE associated with infectious, autoimmune, neoplastic, or metabolic conditions. Rarely, drug-induced, idiopathic, post-radiation, or post-ACS pericarditis is the underlying cause. A key distinctive feature between the 2 categories is fluid composition, as the first group presents with a higher hemoglobin level on pericardial fluid analysis.

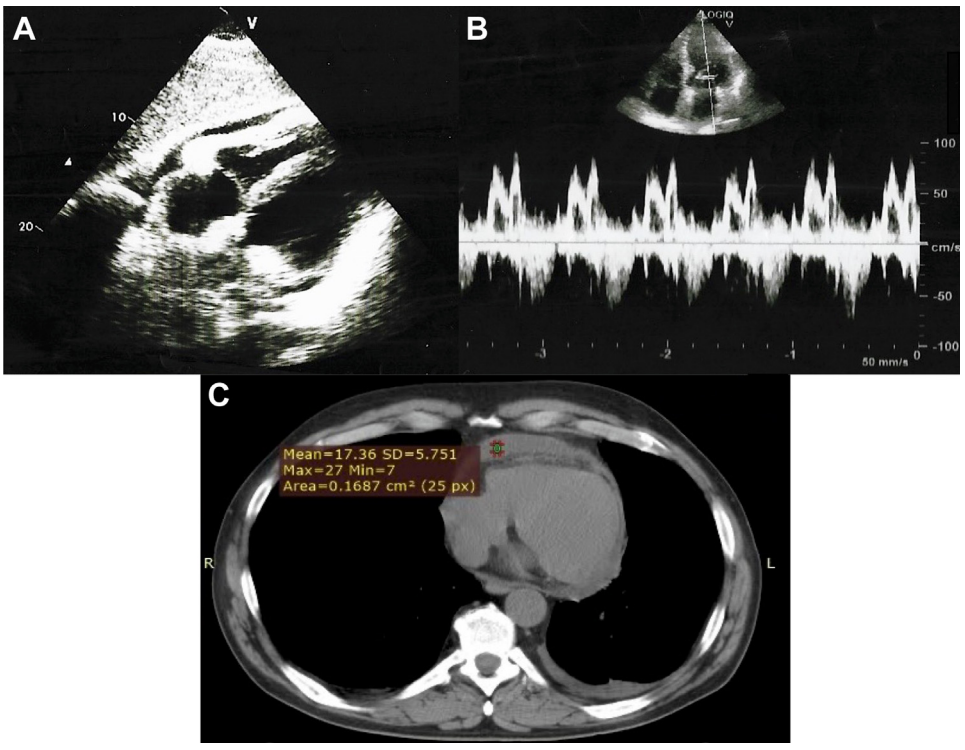
Our patient had not been subjected to iatrogenic interventions or trauma, and neither cancer,

FIGURE 1 Electrocardiograms



Concave ST-segment elevation (arrows). Negative T waves on lateral leads (arrows).

FIGURE 2 **Imaging at First Presentation**



(A) Pericardial effusion without hemodynamic compromise on echocardiography. (B) Normal mitral inflow velocities. (C) Low pericardial fluid density representing transudate, and small left pleural effusion on multidetector computed tomography angiography.

autoimmune disease, drug intake, nor radiation was reported from the history. During his hospitalization, inflammation markers and cardiac troponin were elevated, and ECG revealed typical pericarditis criteria, while common tumor and immunologic markers were negative on blood testing. He had no angina-like symptoms, and the ECG was non-indicative of ACS. However, he was discharged on nonsteroidal anti-inflammatory medicine, which has been linked to increased inflammation and mortality in experimental models of myocarditis.[1] Consequently, our differential diagnosis concludes to acute aortic dissection, myocardial wall rupture associated with silent ACS, and acutely accumulated PE secondary to infective, metabolic, or idiopathic causes.

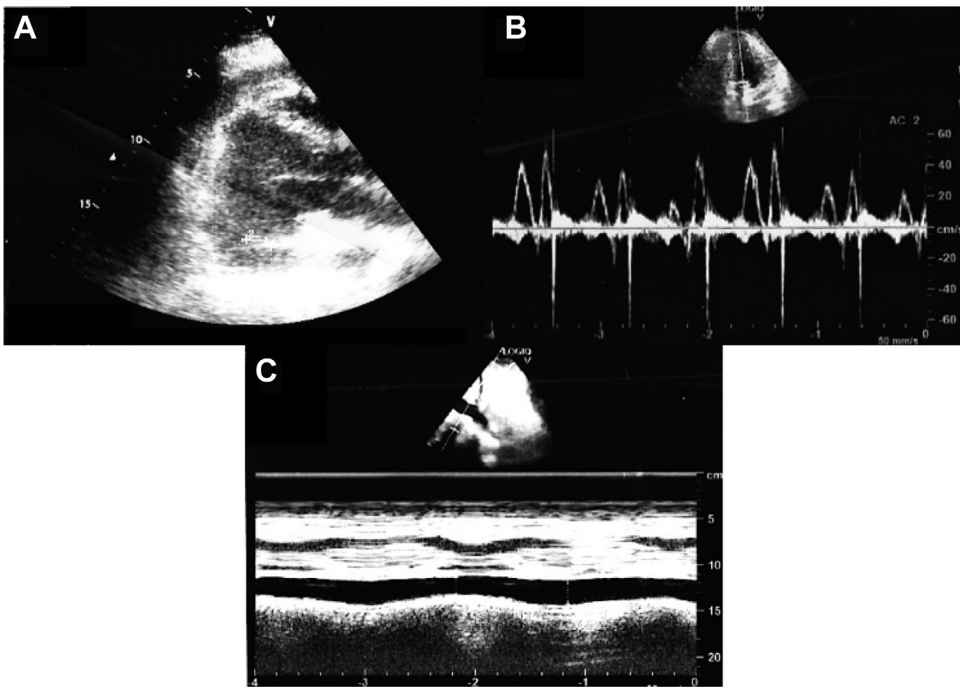
INVESTIGATIONS

Bedside TTE confirmed a large PE, with evidence of organized material inside the pericardial sac (**Figure 4**, Videos 1 to 4).

MANAGEMENT

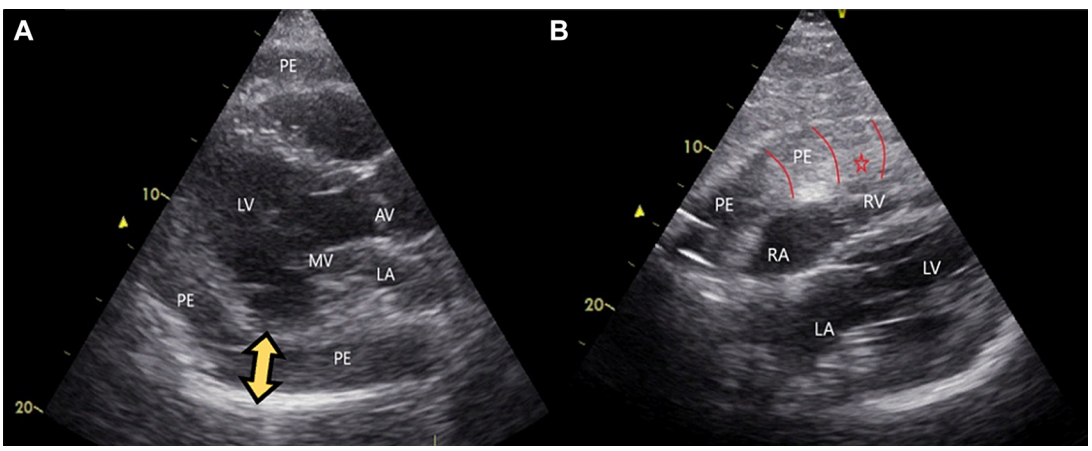
Pericardiocentesis was performed, with withdrawal of 650 mL of bloody fluid. Pericardial fluid gas analysis showed elevated hemoglobin (15.9 g/dL), indicative of hemopericardium. Emergent MDCTA of aorta depicted a large PE with increased fluid density (**Figure 5A**) and contrast pooling in the posterolateral LV wall, suggestive of LV free wall rupture (LVFWR) (**Figures 5B to 5D**). The patient was referred for immediate surgical management. However, during transfer, he developed electromechanical dissociation. Cardiopulmonary resuscitation was initiated, and repeat emergency pericardiocentesis was performed. After 90 minutes of resuscitation, 3 L of pericardial drainage, and simultaneous dual transfusion with uncrossmatched red blood cell products, no evidence of return of spontaneous circulation was observed, and the patient was declared dead. Unfortunately, blood cultures were not obtained due to the patient's acute deterioration.

FIGURE 3 Echocardiography at Second Presentation



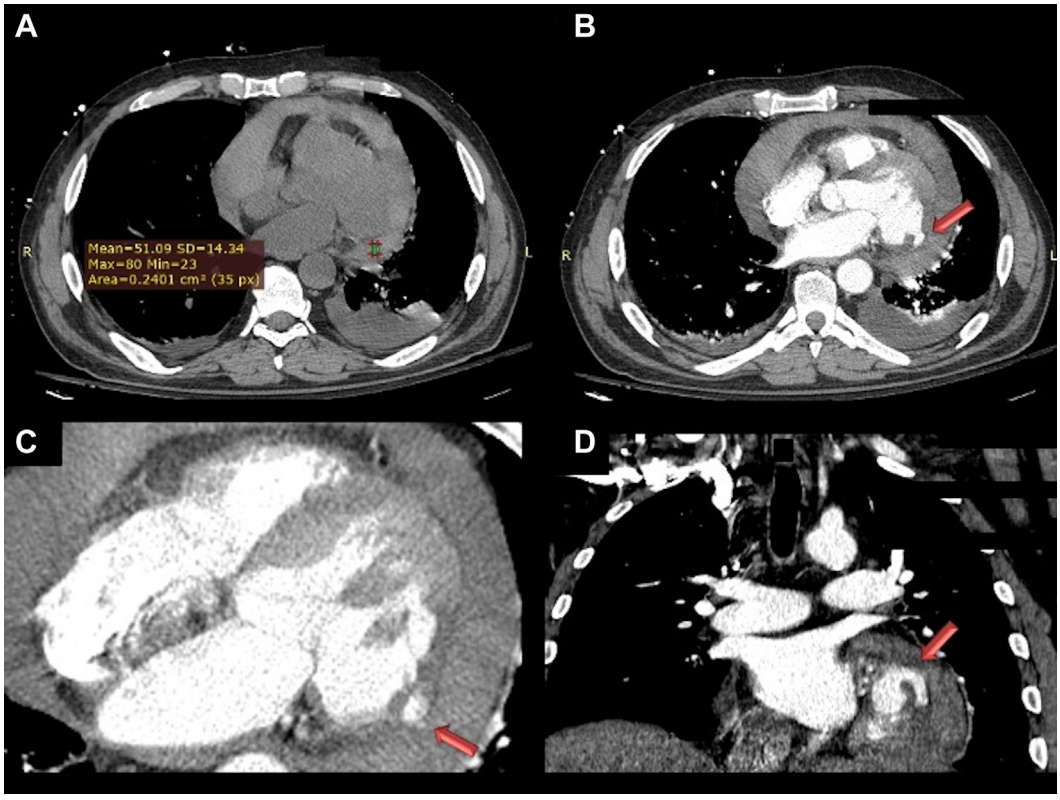
(A) Large (20.5 mm) pericardial effusion. (B) Increased respiratory variation of mitral inflow velocity. (C) Plethora and reduced collapsibility of inferior vena cava.

FIGURE 4 Patient Echocardiography Upon Arrival to Our Hospital



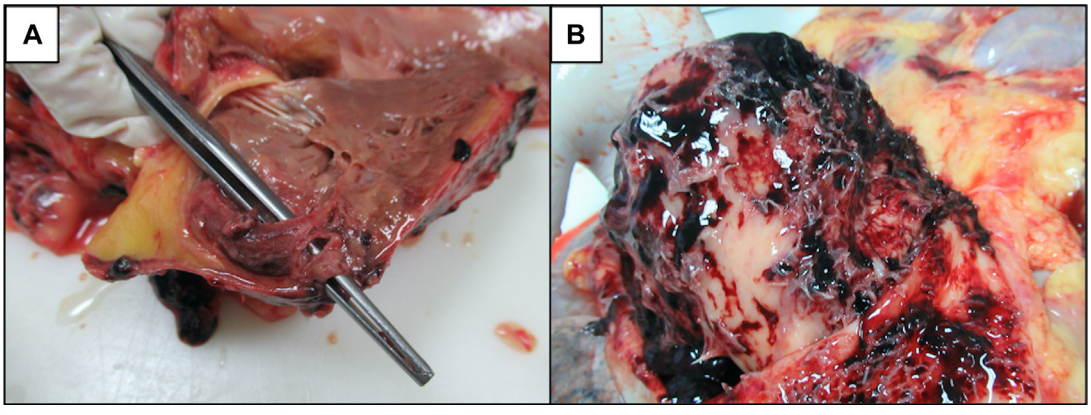
(A) Large pericardial effusion (yellow arrow). (B) Echogenic material inside the pericardial sac (red lines and asterisk). AV = aortic valve; LA = left atrium; LV = left ventricle; MV = mitral valve; PE = pericardial effusion; RA = right atrium; RV = right ventricle.

FIGURE 5 **Multidetector Computed Tomography Angiography**



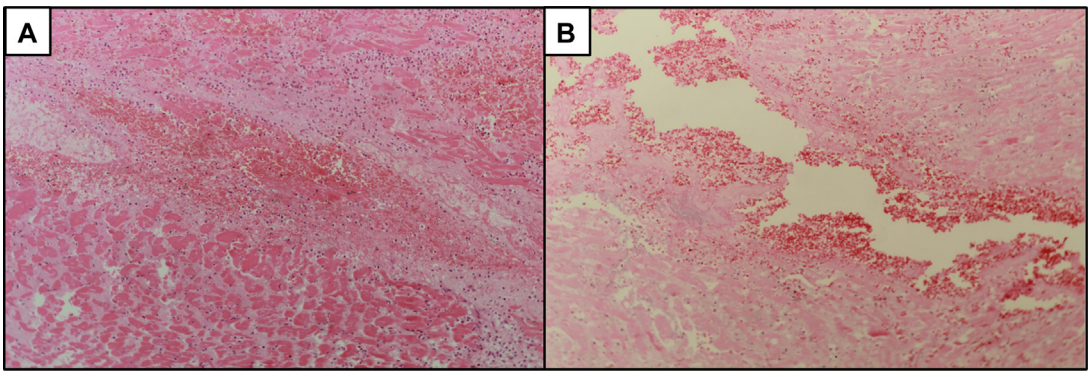
(A) Precontrast. Large hyperdense pericardial effusion with increased fluid density (>50 Hounsfield units), suggestive of hemopericardium. (B) Postcontrast during arterial phase. Contrast pooling in the posterolateral left ventricular (LV) wall corroborating free wall rupture (red arrow). (C) Active bleeding from LV wall to pericardial cavity (red arrow). (D) Coronal reformation postcontrast during arterial phase. Contrast outflow through the posterolateral LV wall (red arrow).

FIGURE 6 **Postmortem Examination**



(A) Left ventricular free wall rupture. (B) A thick fibrinopurulent exudate layer covers the epicardium. Areas of hemorrhagic infiltration.

FIGURE 7 Histology



Histologic examination of ventricular myocardial tissue (A) and rupture site (B).

On autopsy, a rupture (1.5 cm) was identified on the anterior segment of the LV free wall (Figure 6A). Blood infiltrated the myocardium. The cardiac surface was covered by a thick layer of fibrinopurulent exudate with areas of hemorrhagic infiltration (Figure 6B). The coronary arteries displayed mild nonobstructive atherosclerotic deposits, and there was no evidence of endocardial fibrosis. Results of microscopic histology revealed extensive fibrin exudate in the visceral pericardium and polymorphonuclear infiltration in superficial areas with edema and active granulation tissue. Histologic examination of the ventricular myocardial tissue (Figure 7) showed interstitial edema, myocyte necrosis with marked inflammatory infiltrate extending to the pericardium, and hemorrhagic infiltration. The infiltrate was composed mainly of polymorphonuclear cells, and few mast cells and lymphocytes. There were no giant cells, granulomata, or signs of vasculitis. The pathologic diagnosis was acute bacterial myopericarditis with LVFWR.

DISCUSSION

Ventricular free wall rupture post-myocarditis appears in the literature as an extremely rare complication,[2] without concomitant ACS. Although an extraordinary case, it illustrates the "last-drop" phenomenon in cardiac tamponade; due to fixed pericardium extensibility, even the slightest pericardial fluid increment over a specific threshold can disrupt the delicate equipoise, evoke steep pressure rise, and lead the patient from a falsely perceived stability to complete collapse within a single minute. Upon fluid drainage, distinction between "bloody" pericardial fluid and clear blood is of outmost importance, as the latter immediately designates a mechanical complication. In the absence of known trauma (iatrogenic or not), as was the case with our patient, the differential diagnosis of tamponade with clear blood aspirated from the pericardial sac narrows to either aortic dissection or ventricular wall rupture. MDCTA can reveal the specific site of mechanical complication, directing further surgical management.

Free wall rupture represents a rare and dramatic complication. It typically presents in the setting of ACS or iatrogenic manipulations. Prevalence after an ischemic event may be up to 2%, and treatment is surgical, including infarctectomy with patch or surgical glue application on the defect.[3] Abrupt sizable tears lead to cardiac tamponade, while smaller defects may be temporarily limited by localized concealing thrombus formation, easily destabilized by medical manipulations or spontaneously.[4] Suspicion of free wall rupture should be triggered by high hemoglobin levels on pericardial fluid gas analysis and by echocardiographic findings of intrapericardial organized material (thrombus) in any patient presenting with gross PE. Diagnosis may be confirmed by MDCTA imaging, and immediate surgical management should be promptly organized.

CONCLUSIONS

This case raises awareness of a rare cause of LVFWR in a patient with acute myopericarditis. Cardiac tamponade might be silent, and clinical stability in

such patients should not reassure the treating physician. A thorough differential diagnosis of cardiac tamponade causes should always be established to exclude mechanical complications that require urgent surgical management.

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KEY WORDS hemopericardium, left ventricular wall rupture, multidetector computed tomography, myopericarditis, pericardial effusion, tamponade

APPENDIX For a supplemental table and videos, please see the online version of this paper.