

MINI-FOCUS ISSUE: IMAGING

INTERMEDIATE

CASE REPORT: CLINICAL CASE

Loculated Pericardial Effusion

An Uncommon Cause of Left Ventricular Outflow Tract Obstruction



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ABSTRACT

A patient with a previous lung transplant and aortic valve replacement had progressive dyspnea. He presented with subacute tamponade secondary to a loculated pericardial effusion that caused impaired left ventricular filling and outflow tract obstruction secondary to distortion of the mitral valve apparatus. We demonstrate the imaging features of this presentation. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:128-32) Crown Copyright © 2021 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/>).

A 75-year-old man was admitted for progressive dyspnea and 2 syncopal episodes. He had experienced weakness, dyspnea, and a 6-kg weight gain over the last year. Investigations in the 3-months before admission included an echocardiogram demonstrating preserved biventricular systolic function, normal aortic bioprosthesis hemodynamics (mean gradient, 7 mm Hg), and no pericardial effusion or constrictive physiology. Cardiac catheterization 2 weeks before admission (**Figure 1**) showed elevated right atrial (22 mm Hg) and pulmo-

nary capillary wedge (26 mm Hg) pressures. There was an early-diastolic dip and plateau pattern in the right ventricular (RV) tracing, and the RV end-diastolic pressure was similar to the wedge, right atrial (RA), and pulmonary arterial diastolic pressures, thus raising suspicion of pericardial constriction or restriction. Pulmonary arterial pressures were mildly elevated at 39/23/29 mm Hg. A coronary angiogram showed nonobstructive disease, and the cardiac index was 1.5 l/min/m² by thermodilution, which was lower than anticipated. In light of the patient's symptoms and abnormal hemodynamics, an outpatient cardiac magnetic resonance scan was planned to assess for pericardial constriction or infiltrative cardiomyopathy. Pulmonary investigations suggested stable lung transplant function.

The patient's diuretic agents were increased given the elevated central venous and left ventricular (LV) filling pressures. The change in medication was associated with worsening dyspnea and 2 syncopal

LEARNING OBJECTIVES

- To recognize echocardiographic findings suggestive of tamponade or hemodynamic compromise in the setting of loculated pericardial effusion.
- To become familiar with an uncommon cause of left ventricular outflow tract obstruction.

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episodes prompting presentation to the hospital. His blood pressure was 103/73 mm Hg without a pulsus paradoxus, heart rate was 95 beats/min, and oxygen saturation was 98% on 3 l/min oxygen by nasal prongs. Jugular venous pulsation was not visible. There were normal S₁ and S₂ heart sounds with a 3/6 systolic murmur with bibasilar crackles. He was admitted and empirically treated for pneumonia and acute kidney injury with a creatinine value of 3.74 mg/dl.

PAST MEDICAL HISTORY

The patient had undergone replacement of the aortic root with a bioprosthetic valved conduit 1 year earlier, he had a remote lung transplant, and he has chronic kidney disease with a baseline creatinine value of 1.63 mg/dl.

INVESTIGATIONS

An inpatient echocardiogram demonstrated a large, well-circumscribed structure suggestive of a loculated pericardial effusion with mechanical compression of the posterior and lateral regions of the LV

base. The structure caused distortion of the mitral annulus, bringing the mitral leaflets toward the LV outflow tract and resulting in systolic anterior motion of the mitral valve and an 18 mm Hg peak outflow tract gradient at rest. There was at least mild to moderate mitral regurgitation with preserved biventricular systolic function (Figures 2A to 2F, Videos 1, 2, and 3). The effusion was further characterized on non-contrast computed tomography with a density in keeping with a hemopericardium.

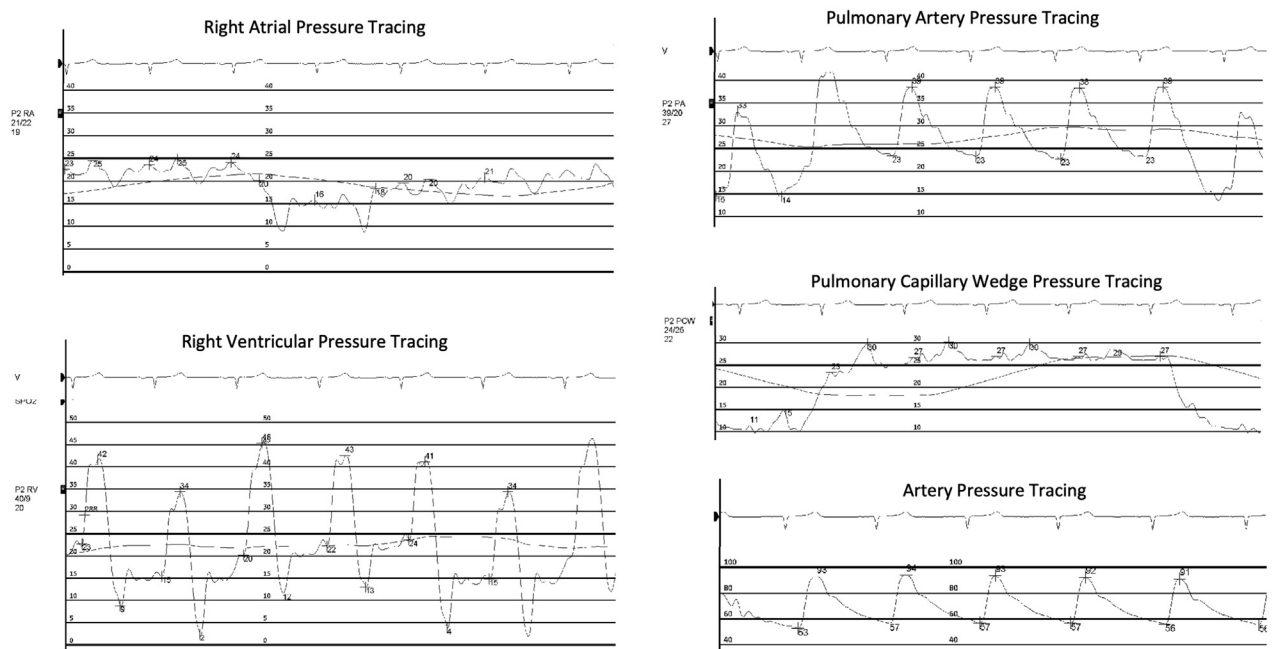
MANAGEMENT

The loculated pericardial effusion was not percutaneously accessible, and it caused hemodynamic compromise with localized tamponade and LV outflow tract obstruction. Given the clinical picture, the patient was taken urgently for evacuation of the pericardial collection by cardiovascular surgery with transesophageal echocardiogram guidance (Figures 3A to 3D, Video 4). The intraoperative echocardiogram confirmed a large hemopericardium with coagulum. There was mitral regurgitation secondary to systolic anterior motion of the anterior leaflet and

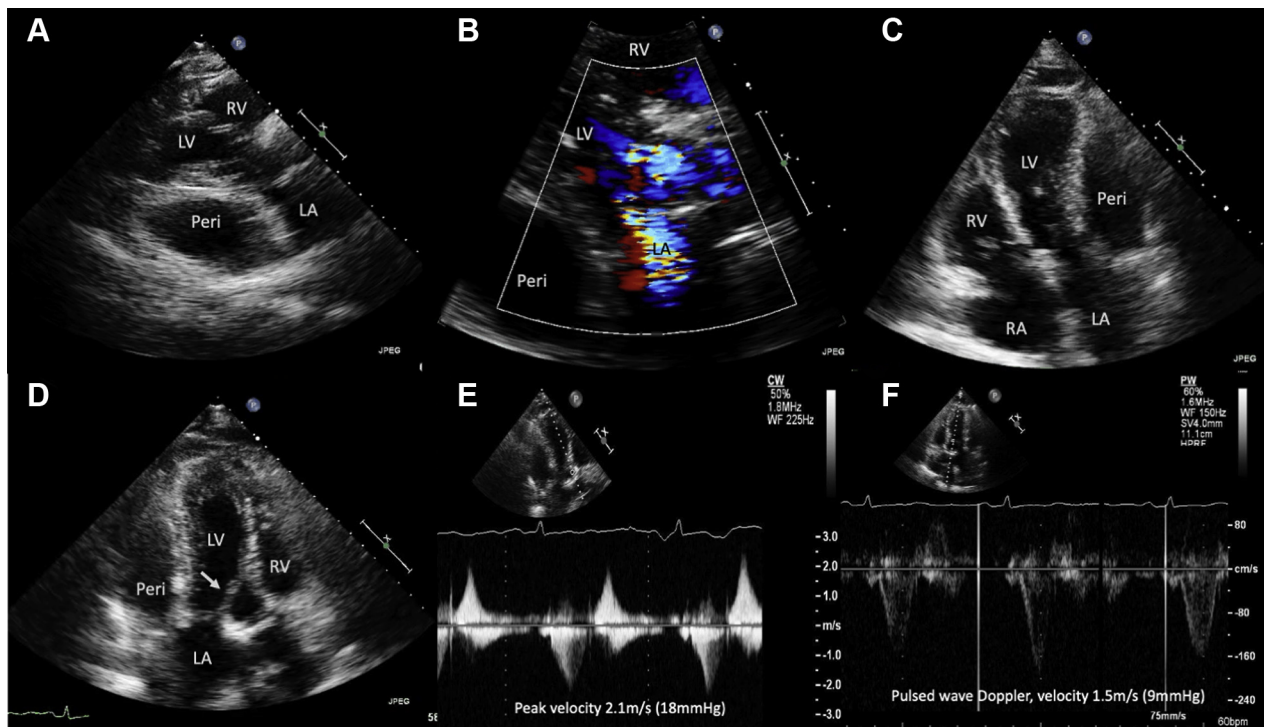
ABBREVIATIONS AND ACRONYMS

- LV = left ventricular
- RA = right atrial
- RV = right ventricular

FIGURE 1 Right-Sided Heart Catheterization Tracings



Elevated right atrial and pulmonary capillary wedge pressure with an early diastolic dip and plateau pattern in the right ventricular tracing. There is near equalization of the right ventricular end-diastolic, pulmonary capillary wedge, right atrial, and pulmonary artery diastolic pressures, thus raising suspicion of pericardial constriction or possibly restriction.

FIGURE 2 Transthoracic Echocardiogram

A large loculated pericardial effusion (Peri) causing compression of the left atrium (LA) and left ventricle (LV) as seen in the (A) parasternal long-axis view at end-diastole, (B) parasternal long-axis view at midsystole with mitral regurgitation, and (C) apical 4-chamber view at end-diastole. (D) The apical 3-chamber view demonstrates anterior displacement of the mitral apparatus with systolic anterior motion of the mitral valve (arrow). (E) There is a later peaking gradient of 18 mm Hg across the left ventricular outflow tract at rest and (F) a 9 mm Hg peak gradient by pulse-waved Doppler proximal to the left ventricular outflow tract. RA = right atrium; RV = right ventricle.

pseudo-prolapse of the posterior leaflet. Through a subxiphoid pericardial window, 350 ml of blood and coagulum was evacuated. There was no source of active bleeding identified, and at the conclusion of the procedure there was a complete resolution of the systolic anterior motion of the mitral valve and LV outflow tract obstruction.

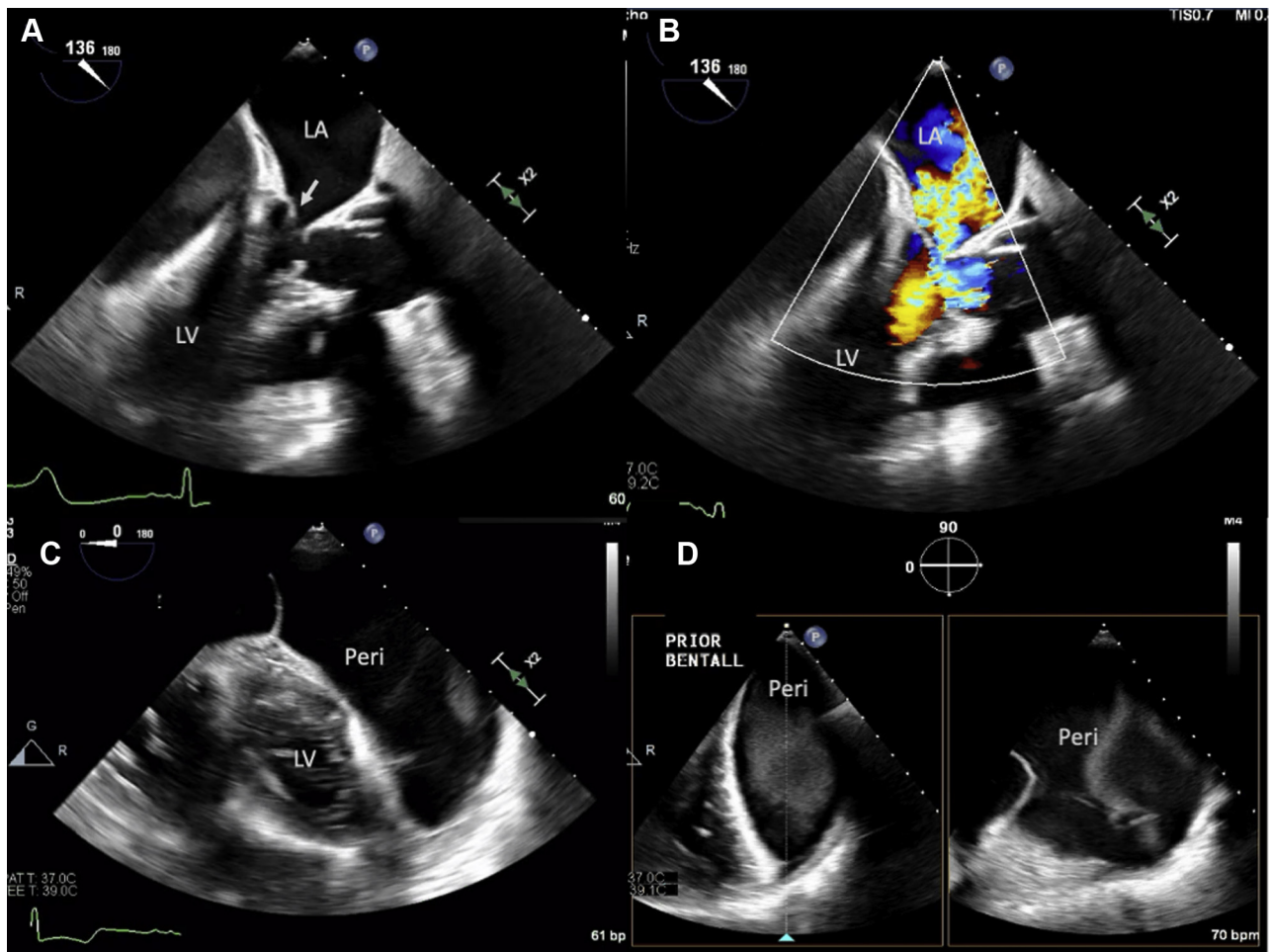
DISCUSSION

Loculated pericardial collections occur more frequently in patients who have had previous instrumentation or trauma because of the presence of adhesions that bridge the visceral and parietal pericardia. Postoperative adherence of the right side of the heart and anterior pericardium to the anterior chest can limit the amount of fluid or blood that can accumulate anteriorly (1). Although the published incidence of pericardial effusion and pericardiocentesis after combined left-sided and right-sided heart catheterization is very rare (0.03% each) (2), one cannot rule out

perforation given the deterioration that occurred post-procedure. The catheterization showed abnormal hemodynamics suggesting an impairment in LV filling. Given the equalization of end-diastolic pressures and the early diastolic dip and plateau pattern in the RV tracing, constriction was a diagnostic consideration; however, equalization of end-diastolic pressures can also be seen in tamponade. The presence of rapid early diastolic filling and the lack of pulsus paradoxus seen on the femoral artery tracing are not supportive of a diagnosis of pre-existing tamponade, although localized effusions can manifest with atypical hemodynamics. The effusion may well have been slowly progressing since the time of cardiac surgery, thus resulting in the symptoms and hemodynamic findings.

As seen in this case, loculated pericardial collections can cause isolated, differential compression of chambers, thereby compromising cardiac output. The echocardiographic appearance of a circumferential pericardial effusion would result in compression of all

FIGURE 3 Intraoperative Transesophageal Echocardiogram



(A) Systolic anterior motion of the mitral valve with **(B)** significant mitral regurgitation secondary to systolic anterior motion of the valve and pseudo-prolapse of the posterior leaflet (arrow in **A**). **(C)** The loculated pericardial effusion (Peri) is associated with left ventricular diastolic collapse and compression of the mitral valve apparatus as seen in the transgastric short-axis view. **(D)** The coagulum within the loculated pericardial effusion is displayed in cross plane. LA = left atrium; LV = left ventricle.

the chambers that would cause RA and RV diastolic collapse, as well as respirophasic changes in mitral and tricuspid inflow velocities. With localized pericardial effusion, the previously outlined Doppler and 2-dimensional findings are often absent. Studies that examine the hemodynamic impact of localized LV pericardial effusion are limited, but case reports and hemodynamic studies in animals suggest that LV diastolic collapse is a reliable finding in LV tamponade (3). The diastolic collapse is often associated with a reduction in cardiac output and an increase in LV end-diastolic pressure without a change in RA pressure or pulsus paradoxus (4).

In addition to tamponade, our patient manifested LV outflow tract obstruction with systolic anterior motion of the mitral valve resulting from distortion of the mitral apparatus. The hemopericardium caused the LV cavity to become small, with anterior displacement of the mitral apparatus predisposing this patient to prolonged contact of the interventricular septum and mitral valve. Although dynamic LV outflow tract obstruction has been described as a physiological consequence of aortic dissection with pericardial effusion (5), our case demonstrates a mechanical mechanism of obstruction.

FOLLOW-UP

Follow-up echocardiograms on post-operative days 2 and 4 showed a stable-sized residual collection without features of increased intrapericardial pressures or compression. The gradient across the LV outflow tract had decreased (peak gradient, 9 mm Hg). Following surgery, there was a marked improvement of symptoms, with near resolution of dyspnea and improvement in renal function.

CONCLUSIONS

We report a case of a large, loculated hemopericardium causing tamponade physiology and LV outflow tract obstruction secondary to systolic anterior mo-

tion of the mitral valve. To our knowledge, this is the first time that a loculated pericardial effusion has been reported as a cause of dynamic LV outflow tract obstruction.

AUTHOR DISCLOSURES


The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS left ventricular outflow tract obstruction, pericardial effusion, tamponade

 **APPENDIX** For supplemental videos, please see the online version of this paper.