

# A Case of Persistent Right Ventricular Failure after Rapid Decompression of a Large Chronic Pericardial Effusion

Harry Klimis, MBBS, BMedSci, Mikhail Altman, MD, FRACP, DDU, PhD,  
Timothy Tan, MBBS, BSc, PhD, FRACP, FCSANZ, FACC, FESC, FAHA,  
Jojie Natividad, BSc, MD, Robert Abraham, BSc, MBBS, FRACP, FCSANZ,  
and Liza Thomas, MBBS, FRACP, PhD, *Camperdown, Westmead, and Sydney, Australia*

## INTRODUCTION

We report the case of a patient with Hodgkin's lymphoma (diagnosis unknown at initial presentation) who presented with severe dyspnea secondary to a very large global pericardial effusion. Following pericardial drainage, the patient developed overt right ventricular (RV) failure. A detailed case report and brief overview of the potential mechanisms of persistent RV failure following pericardiocentesis are reported.

## CASE PRESENTATION

A 33-year-old man presented to the emergency department with right-sided pleuritic chest pain, exertional dyspnea, and lower limb edema over the preceding 2 months. There was no significant medical history, and he was not on any regular medications. He was tachycardic (heart rate 130 beats/min) but normotensive (blood pressure 123/83 mm Hg) and tachypneic (30 breaths/min) with normal oxygen saturation on room air. His heart sounds were muffled, and neck veins were distended, with an elevated jugular venous pressure. Pulsus paradoxus was not assessed. Electrocardiography showed sinus tachycardia with low-voltage QRS and electrical alternans. Chest radiography showed marked cardiomegaly and a possible mass along the right mid to upper lateral cardiac border (Figure 1). Urgent transthoracic echocardiography (TTE) demonstrated a very large global pericardial effusion measuring at most 50 mm (during diastole), with early diastolic RV collapse indicating early echocardiographic evidence of tamponade (Figure 2, Video 1). RV and left ventricular (LV) systolic function was normal. The inferior vena cava was dilated, measuring 4.5 cm, with <50% collapse on inspiration. During urgent pericardiocentesis, 2 L of straw-colored fluid was removed via the subxiphoid approach. A pigtail catheter was left in situ on free drainage.

Immediately after the procedure, heart rate was 118 beats/min and blood pressure was 133/83 mm Hg. Limited TTE performed immediately after drainage showed complete resolution of the pericardial effusion and a moderately dilated right ventricle with severely impaired systolic function (Figure 3). However, despite drainage of the pericardial fluid, the patient remained symptomatic with persistent dyspnea. Respiratory rate was 27 breaths/min, and oxygen saturation was 93% on room air, necessitating oxygen supplementation. Over the next few hours, the patient continued to be tachycardic, and blood pressure decreased to 90/60 mm Hg. Inotropic support was required to maintain normal blood pressure.

Progress TTE on day 1 after pericardiocentesis showed reaccumulation of a moderate (22 mm) residual pericardial effusion. There was no evidence of RV diastolic collapse as noted previously. However, the right ventricle was moderately dilated (basal diameter 54 mm) with severely impaired segmental systolic dysfunction (Figure 4, Videos 2 and 3). The RV basal segment was akinetic with hyperkinetic mid-apical RV free wall segments, appearance typical for McConnell's sign. The interventricular septum was flattened and showed paradoxical motion. LV size was normal with mildly impaired systolic function. Additionally, there was no evidence of intracardiac shunt, and tricuspid regurgitant jet was insufficient to estimate pulmonary pressures. The inferior vena cava was not dilated and collapsed >50% during inspiration, and there was trivial tricuspid regurgitation.

Computed tomographic pulmonary angiography (Figure 5) showed a large anterior mediastinal mass (10 × 18 × 14 cm) displacing the heart to the left and vessels posteriorly with associated large pericardial and a small right pleural effusion. The superior vena cava, right-sided pulmonary veins, and left atrium were partially compressed. However, the diameters of the main, right, and left pulmonary arteries were normal. Lymphoproliferative disease was suspected and remained high on the differential list. There was no evidence of a pulmonary embolus or features of pulmonary arterial hypertension. It was decided to proceed with surgical creation of a pericardial window because of evidence of reaccumulation of the pericardial effusion. Because of hypotension from severe RV systolic dysfunction coupled with anesthetic requirement, adrenaline and milrinone infusions were required during the procedure. The pericardial fluid had a yellow, turbid macroscopic appearance, and the analysis revealed a mixed inflammatory exudate comprising lymphocytes, neutrophils, and occasional eosinophils; no malignant cells were detected. Macroscopic appearance of the pericardium was normal, and microscopic appearance showed minor reactive changes only.

The patient proceeded to undergo computed tomography (CT)-guided biopsy of the mediastinal mass 2 days later through a right-sided anterior thoracotomy, which revealed Hodgkin's lymphoma

From the University of Sydney (H.K., M.A., L.T.); The George Institute for Global Health (H.K.), Camperdown; the Department of Cardiology Westmead Hospital, Westmead (H.K., M.A., T.T., J.N., R.A.); and the University of New South Wales (L.T.), Sydney, Australia.

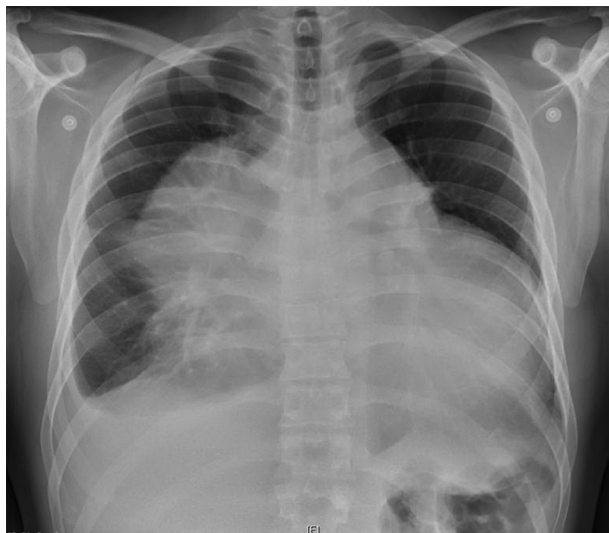
Keywords: Cardiac tamponade, Pericardial decompression syndrome, Pericardiocentesis, Right ventricular failure, Pericardial effusion

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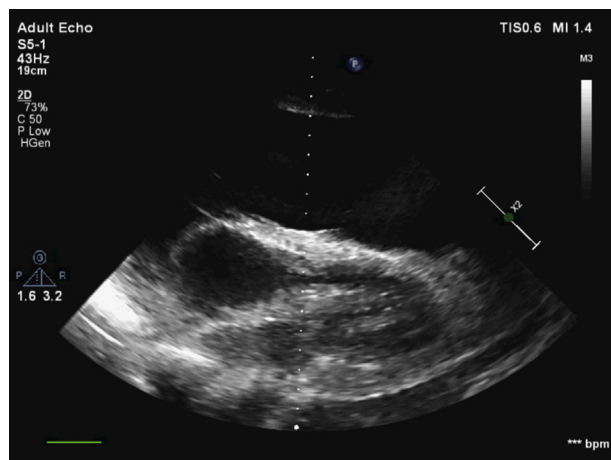
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**Figure 1** Plain posterior-anterior chest x-ray obtained in the emergency department on the day of presentation. There is marked cardiomegaly with bulging of the lateral right mid to upper cardiac margin. A small pleural effusion is present at the right lung base. No features of pulmonary edema or focal consolidations are seen.

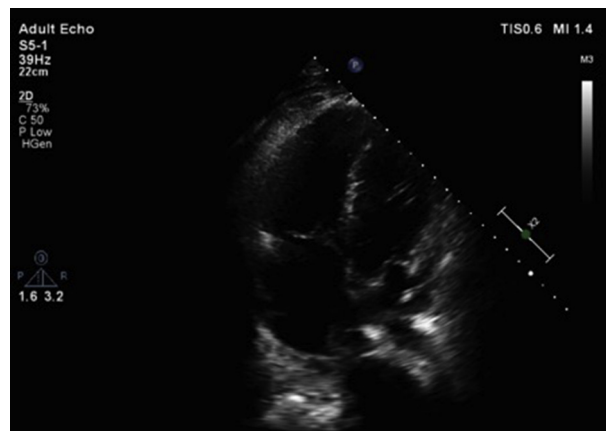


**Figure 2** Urgent TTE performed on presentation (*subcostal view*). A very large global pericardial effusion measuring at most 50 mm (during diastole) is seen with early diastolic right ventricular collapse and echocardiographic evidence of tamponade.

(classical nodular sclerosing type). Staging positron emission tomography and CT of the abdomen and pelvis showed stage IIa disease (a single lymph node involved in addition to the mediastinal mass and no involvement below the diaphragm), and treatment with standard chemotherapy and radiotherapy was initiated. The patient was discharged 18 days following his initial presentation. Follow-up TTE 12 months after discharge showed normal biventricular size and function.

## DISCUSSION

This case highlights the occurrence of pericardial decompression syndrome (PDS) with rapid drainage of a large pericardial effusion.



**Figure 3** TTE performed immediately after pericardiocentesis (*apical four-chamber view*). Complete resolution of pericardial effusion was noted immediately after pericardiocentesis; the RV was moderately dilated with severe systolic impairment.

Although in the vast majority of cases, drainage of pericardial effusion occurs without any significant compromise of ventricular function, PDS can occur in some instances and therefore necessitates recognition of its occurrence. In our case, the cause of the effusion was likely nonmalignant, and given the presence of an inflammatory milieu on pericardial fluid analysis and absence of opportunistic infection, the cause of the pericardial effusion is likely to be inflammatory or reactive. Partial superior vena cava obstruction, as was seen in this case, could also potentially explain the pericardial and pleural effusions. However, in this instance a transudate would have been expected on fluid analysis. A paraneoplastic process is also a possible differential diagnosis.

Pericardiocentesis is a lifesaving procedure in the situation of cardiac tamponade, aimed at rapidly reducing the pericardial pressures. However, complication rates as high as 10% have been reported and include cardiac puncture, coronary artery injuries, arrhythmias, pneumothorax, hemothorax, pneumopericardium, and hepatic injury.<sup>1</sup> Interestingly, there have been several, albeit infrequent, reports of paradoxical hemodynamic compromise manifesting as RV,<sup>2-4</sup> LV,<sup>5,6</sup> or biventricular<sup>7-10</sup> failure following pericardial drainage. Cases of normal biventricular function and acute pulmonary edema have also been reported.<sup>11-13</sup> PDS is defined as hemodynamic compromise and/or pulmonary edema paradoxically occurring soon after successful pericardial fluid drainage, which is often associated with ventricular impairment.<sup>13,14</sup> Treatment is supportive, and ventricular function is expected to recover in survivors.

A recent review of case reports found a total of 35 cases of PDS<sup>14</sup> with onset varying from immediately after drainage to 48 hours later. The causes of the effusion were unknown in the majority of cases ( $n = 15$  [42%]), with the second most common being malignant effusion ( $n = 14$  [40%]). The volume of pericardial fluid drained ranged from 450 to 2,100 mL (mean, 888 mL). Presentations of PDS were predominantly LV failure ( $n = 14$  [40%]) and cardiogenic pulmonary edema without shock ( $n = 10$  [29%]). RV and biventricular failures were less frequent presentations. There was a high mortality rate in this case series ( $n = 10$  [29%]), which occurred between 6 hours and 14 days after drainage. Interestingly, mortality was associated only with surgical

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