

Acute Left Atrial Compression after Ventricular Assist Device Placement



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INTRODUCTION

We present a 58-year-old woman with end-stage ischemic cardiomyopathy who developed hemolysis on postoperative day 15 after left ventricular assist device (LVAD) and subsequent right ventricular (RV) assist device (RVAD) placement. Echocardiogram revealed localized compression of the left atrium with near-complete collapse causing turbulent blood flow, which resolved with surgical mediastinal hematoma evacuation. The next day she developed a thrombus at the tip of her RVAD cannula, requiring RVAD replacement. This case highlights the use of device parameters and echocardiography in the diagnosis of these two ventricular assist device complications.

CASE PRESENTATION

A 58-year-old woman with a history of end-stage ischemic cardiomyopathy (ejection fraction of 20%) on home milrinone, coronary artery disease (left anterior descending artery stent 15 years prior), and biventricular implantable cardioverter-defibrillator placed 10 years prior was admitted for placement of a Heartmate (Abbott) III LVAD (admission timeline in [Figure 1](#)). On postoperative day 2, she became hypotensive with low LVAD flows (1.7-2.1 L/minute). Hemodynamic parameters at the time of the episode revealed a central venous pressure of 20 mm Hg without evidence of tamponade on transthoracic echocardiography (TTE). An RVAD was placed percutaneously for severe RV failure with end-organ dysfunction.

On postoperative day 15, she developed tea-colored urine and sustained ventricular tachycardia, requiring cardioversion. Laboratory workup was indicative of hemolysis including a haptoglobin < 10 mg/dL, rising total bilirubin peaking at 11.7 mg/dL (direct bilirubin, 7.5 mg/dL), plasma-free hemoglobin of 11.7 mg/dL, and lactate dehydrogenase of 1,144 IU/L. However, her LVAD (power, 3.2 watts; flow, 3.0 L/minute) and RVAD (flow, 2.7 L/minute) parameters re-

mained unchanged from prior days. No clinical signs of RV failure were present, and her central venous pressure was 14 mm Hg.

TTE demonstrated a greater than 3 cm, loculated pericardial effusion causing posterior left atrial compression ([Figure 2](#)). The left ventricle was small and underfilled with mild to moderately reduced left ventricular ejection fraction based on visual assessment from the parasternal long-axis view, parasternal short-axis view, and apical four-chamber view zoomed left ventricle (though unable to be quantitated due to the technically difficult study). The left ventricular internal dimension at diastole was 3.0 cm. On visual estimation in the apical four-chamber, right ventricle-focused view, RV dilation and decreased function were present. The inferior vena cava was >21 mm, with <50% decrease during inspiration, indicative of an elevated right atrial pressure.

Bedside transesophageal echocardiography (TEE) confirmed the finding of localized compression of the left atrium with near-complete collapse ([Figure 3A](#), [Figure 4A](#)), resulting in a turbulent flow pattern ([Video 1](#)). Left ventricular ejection fraction was estimated to be 35%-40% with hypokinesis of the inferior, inferoseptal, and inferolateral walls. Mild RV dilation and dysfunction were also present on visual estimation from the midesophageal four-chamber and the RV inflow views. Mild mitral regurgitation was demonstrated when color flow Doppler was applied to the midesophageal four-chamber view.

Given the lack of device parameter fluctuations and the significant findings on TTE and TEE, device thrombosis was thought to be a less likely etiology, so a contrast-enhanced computed tomography (CT) scan was not obtained.

She was emergently taken to the operating room where diffuse mediastinal oozing was noted, and 30-40 mL of loculated mediastinal hematoma that was compressing the left atrium was removed, resulting in significantly improved left atrial filling and resolution of the previously turbulent flow ([Figure 3B](#), [Figure 4B](#), [Video 1](#)). The left ventricular internal dimension at diastole was 5.8 cm postoperatively. Intraoperative TEE directly visualized the inflow cannula and outflow grafts of both the RVAD and LVAD, confirming placement and lack of thrombus at the visualized portions of the devices. Postoperatively, her lactate dehydrogenase and total bilirubin were downtrending to 904 IU/L and 8.7 mg/dL, respectively, and her urine became significantly less dark.

The next day, she had acute worsening of her RVAD flows, and she was found to have a hemoglobin of 4.7 g/dL, for which she received 5 units of packed red blood cells. Bedside TEE demonstrated recurrence of the pericardial effusion that was more circumferential than the prior and resulted in near-complete collapse of the left atrium and left ventricle. Additionally, a 1.4 cm thrombus was seen on the tip of the RVAD cannula in the main pulmonary artery ([Figure 5](#), [Video 2](#)). She was emergently taken back to the operating room for a second evacuation of the cardiac tamponade, and she underwent replacement of percutaneous RVAD with stabilization of her hemoglobin.

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TEE, midesophageal, apical four-chamber view with color flow Doppler demonstrating turbulent flow through the nearly collapsed left atrium (A) on postoperative day 15. After surgical mediastinal hematoma evacuation (B), left atrial volume significantly improved. Color flow Doppler at this time revealed laminar flow through the left atrium.

Video 2: Two-dimensional TEE, upper esophageal, rightward rotated, 51° view without (left panel) and with (right panel) color flow Doppler on postoperative day 16 demonstrates the RVAD cannula (arrow) with associated thrombus (arrow) within the main pulmonary artery (PA). A typical turbulent blood flow pattern is demonstrated exiting the cannula.

View the video content online at www.cvcasejournal.com.

She remained critically ill and required vasopressor and inotropic support. Due to her complicated postoperative course, it became clear the patient would not have the independence she sought, which was the driving factor in her decision to pursue mechanical circulatory support. As the hope of this independence dwindled, the family decided that comfort care was more in alignment with the patient's wishes, and she ultimately passed away 43 days after RVAD placement.

DISCUSSION

This case highlights the various etiologies of hemolysis after recent ventricular assist device implantation. On postoperative day 15, TTE and TEE demonstrated left atrial compression with turbulent flow and subsequent resolution of hemolysis with surgical hematoma evacuation, leading to our hypothesis that the left atrial compression was the etiology of the hemolysis. However, it is difficult to attribute the hemolysis solely to the turbulent flow from left atrial compression when other explanations remain plausible. For example, the severe underfilling of the left ventricle could have resulted in modification of the inflow cannula alignment, which could have induced hemolysis. Other hemodynamic changes within the underfilled left ventricle could also be responsible, particularly given the presence of prosthetic material in this patient. Furthermore, although the absence of device fluctuations makes pump thrombosis less likely, this should remain on the differential as contrast-enhanced CT was not obtained.

Though multiple causes were possible, we suspected that turbulent flow within the compressed left atrium was most responsible for the hemolysis. The thin-walled left atrium is susceptible to compression by gastrointestinal, pulmonary, or various other mediastinal structures.^{1,2} Left atrial compression is relatively rare and typically presents with signs of congestive heart failure or even cardiogenic shock. Turbulent flow in the left atrium can also result, which carries a theoretical risk of hemolysis.¹

The diagnostic test of choice is TTE or TEE, which is typically able to identify the extracardiac compression.¹ In addition to direct visualization, a variety of other findings will also be present on echocardiography. The extrinsic compression will cause decreased left atrial volume

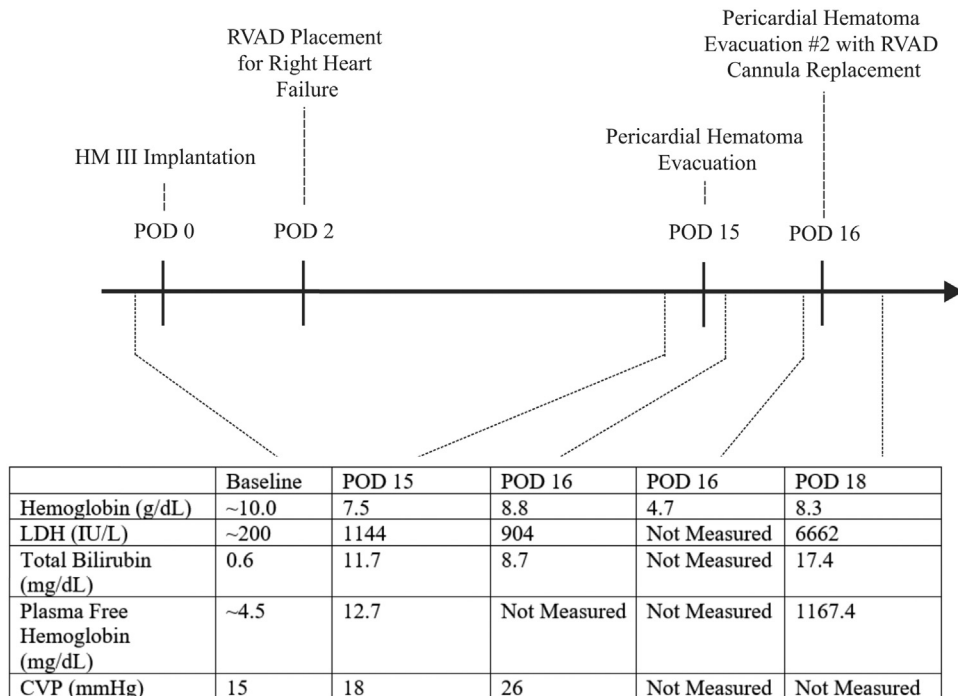


Figure 1 Timeline of key case events with relevant laboratory values. Laboratory measures of hemolysis were found to be rising on postoperative day 15 at the time the focal pericardial compression was discovered. After hematoma evacuation, these labs were initially downtrending. Then the patient developed RVAD flow changes and acute anemia, so she was emergently taken back to the operating room where she was found to have recurrence of the hematoma in addition to an RVAD thrombus. After this operation, these hemolysis labs were then measured and found to be markedly higher than earlier. CVP, Central venous pressure; HM, Heart-Mate; LDH, lactate dehydrogenase; POD, postoperative day; RVAD, right ventricular assist device.

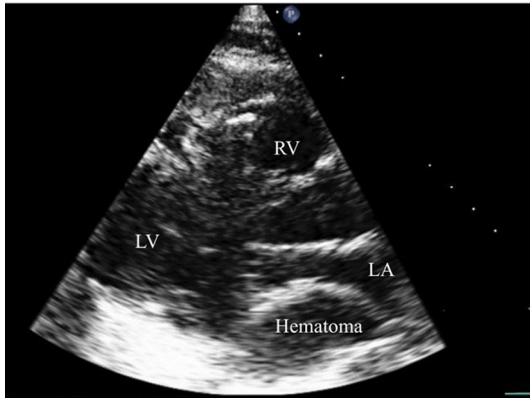


Figure 2 Two-dimensional TTE parasternal long-axis view obtained on postoperative day 15 when the patient developed acute hemolysis and tea-colored urine demonstrating 3 cm pericardial effusion causing compression of the left atrium. The left ventricle was also underfilled, with a left ventricular internal diameter at diastole of 3.0 cm. Left ventricular ejection fraction was visually estimated at 35%-40%. LA, Left atrium; LV, left ventricle; RV, right ventricle.

and subsequent underfilling of the left ventricle. Severe underfilling can result in near left ventricular collapse with the papillary muscles almost touching during systole (the so-called kissing papillary muscle sign).³ Subsequently, stroke volume and flow across the left ventricular outflow tract would be very low. Color flow Doppler of the residual left atrium can also be used to differentiate laminar versus turbulent blood flow to identify those at high risk for hemolysis. Right heart failure can also be seen in acute left atrial compression, which could include findings of RV dilation, decreased RV systolic function, tricuspid regurgitation, or a dilated inferior vena cava with poor compressibility, which is indicative of an elevated right atrial pressure.

This case presents a unique diagnostic dilemma as the presentations of device thrombosis and left atrial compression have significant overlap, and ultimately our working diagnosis was determined through a combination of ventricular assist device parameters and echocardiographic findings.⁴ During the first episode of hemolysis, LVAD dysfunction was thought to be less likely because the pump powers and device interrogation did not reveal evidence of thrombosis. RVAD thrombosis was less likely due to the absence of fluctuations in device flow. Therefore, hemolysis secondary to turbulent blood flow through the compressed left atrium was our working diagnosis,

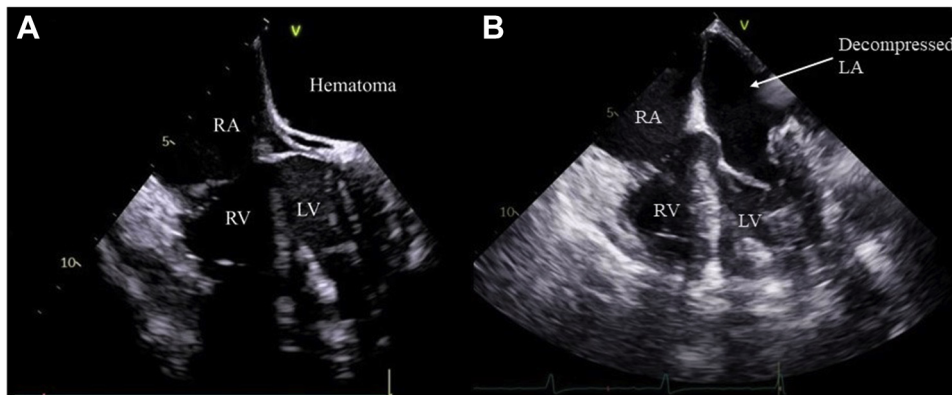


Figure 3 Pre- and postoperative two-dimensional TEE, midesophageal four-chamber view before (A) and after (B) evacuation of focal pericardial effusion on postoperative day 15. Preevacuation, the pericardial hematoma was causing near-complete compression of the left atrium, resulting in the turbulent flow seen with color flow Doppler. After evacuation of the pericardial hematoma, left atrial volume immediately improved (arrow), and color flow Doppler demonstrated laminar flow. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

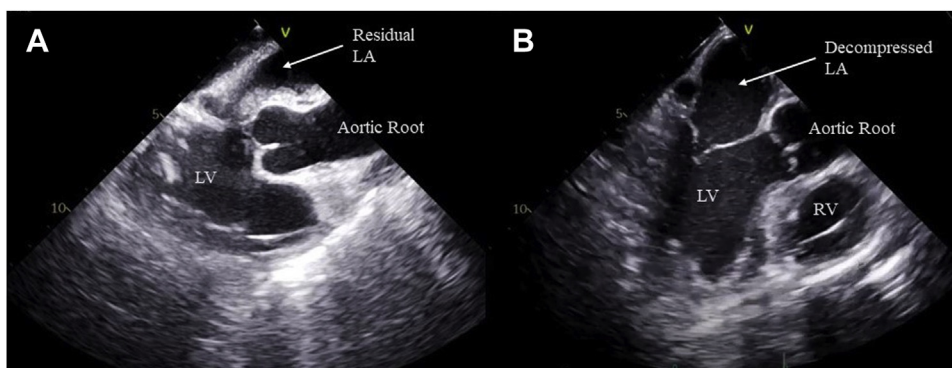


Figure 4 Two-dimensional TEE, midesophageal aortic valve long-axis view before (A) and after (B) evacuation of focal pericardial effusion on postoperative day 15. Preevacuation, the pericardial hematoma caused severely decreased size of the left atrium (arrow in A) and left ventricle, which immediately improved (arrow in B) after evacuation of focal pericardial effusion. LA, Left atrium; LV, left ventricle; RV, right ventricle.

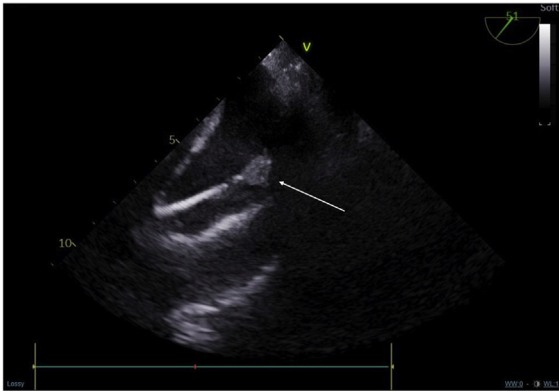


Figure 5 Two-dimensional TTE upper esophageal aortic arch short-axis view at 51° on postoperative day 16 demonstrating a 1.4 cm thrombus (*arrow*) at the tip of the RVAD cannula in the main pulmonary artery. Color flow Doppler (*Video 2*) demonstrates the turbulent flow surrounding the thrombus.

a hypothesis that was supported by the initial improvement in lactate dehydrogenase and total bilirubin immediately following the first mediastinal evacuation. During the second episode, the acute decrease in RVAD flow increased suspicion for cannula thrombosis, which was ultimately confirmed with direct visualization of the thrombus at the tip of the outflow cannula in the main pulmonary artery.

In the evaluation of suspected pump thrombosis, echocardiography is the recommended initial imaging modality though it does not visualize the entire outflow graft and therefore cannot rule out a diagnosis of device thrombosis. In our case, the thrombus was directly identified due to its location on the tip of the cannula, which confirmed the diagnosis. Although not performed in this patient, outflow graft velocity > 2 m/sec measured over three to five cardiac cycles is concerning for a possible obstruction, but the sensitivity and specificity of this abnormal finding is not clear.⁵ In cases where echocardiography is not diagnostic, the reference standard test is a contrast-enhanced CT scan, which allows for visualization of the ventricular assist device and has a sensitivity of 85% and a specificity of 100% in detecting either mispositioned or occluded devices.⁶

In addition to its key role in diagnosing complications after ventricular assist device placement, echocardiography is a crucial component of routine, postimplantation surveillance for all ventricular assist device patients. The 2015 American Society of Echocardiography guidelines recommended echocardiographic screening exams after LVAD implantation in all patients at the following intervals: 2 weeks; 1, 3, 6, and 12 months; and then every 6-12 months.⁵ Complications

that should be screened for with echocardiography include right or left ventricular failure, LVAD-related aortic or mitral regurgitation, inflow cannula or outflow graft abnormalities, and pump malfunction.⁵

CONCLUSION

Left atrial compression is a rare complication of ventricular device placement that has significant overlap in presentation with the more common pump thrombosis. This case uniquely presents both complications in the same patient, allowing for comparison. Close evaluation of ventricular assist device parameters along with utilization of echocardiography allowed for proper diagnosis and treatment.

ACKNOWLEDGMENT

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SUPPLEMENTARY DATA

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.case.2022.01.008>.

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