Echocardiography-Guided Percutaneous Ventricular Assist Device for Postinfarct Ventricular Septal Rupture

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INTRODUCTION

Ventricular septal rupture (VSR) is an increasingly rare yet devastating complication of acute myocardial infarction (MI). Mortality after early operative repair remains elevated, with rates as high as 40%.[1,2] As VSRs commonly result in the formation of a large shunt and frequently cardiogenic shock, mechanical circulatory support (MCS) devices have been increasingly used as a bridge to surgery, allowing for adequate perfusion and hemodynamic support while the infarcted myocardium recovers.[1,3,4] Intraoperative transesophageal echocardiography (TEE) serves a critical role in placement of such devices. This report describes a case in which the Impella 5.5 (Abiomed), a percutaneous ventricular assist device (pVAD), was used as part of a bridging strategy in a patient who developed VSR after acute MI.

CASE REPORT

A 75-year-old patient with a history of coronary artery disease, chronic obstructive pulmonary disease, hypertension, and breast cancer presented to the emergency department with an anterolateral ST-segment elevation MI. No murmur was identified on the physical exam, which was thought to be due to the large size of the defect and decreased flow from cardiogenic shock. Coronary angiography was performed, and a drug-eluting stent was placed for a 100% occluding left anterior descending artery lesion. Ticagrelor and aspirin were given for dual antiplatelet therapy. However, the patient remained unstable in the recovery area with continued ST changes. After 6 hours, a repeat coronary angiogram and pulmonary artery catheterization revealed a patent left anterior descending stent but noted a significant oxygen saturation step up from the right atrium to the right ventricle (RV); invasive left ventriculography and transthoracic echocardiography (TTE) confirmed a large VSR with left-to-right shunt (Figure 1).

The patient, who was on a norepinephrine infusion with severe lactic acidosis (pH 6.8, lactate of 18 mmol/L) in stage D cardiogenic shock, was brought to the operating room for MCS device insertion.[5] A multidisciplinary team of cardiologists, surgeons, and structural interventionalists concluded that a pVAD would be used for MCS, instead of venoarterial extracorporeal membrane oxygenation (VA ECMO), because of the depressed left ventricular function (ejection fraction of 40%-45%), normal RV function, and adequate oxygenation (measured by oxygen saturation and PaO_2 on arterial blood gas). The option to escalate to VA ECMO, if needed, remained.

After induction with general anesthesia, TEE was used to guide the pVAD into the appropriate position via the midesophageal long-axis view of the aortic valve (AV). The pVAD should be about 4.0 to 5.5 cm into the left ventricle (LV; when measured from the AV to the tip of the pVAD), directed at the apex, away from the mitral valve and the inferolateral wall (Figure 2, Video 1) with the outflow tract positioned appropriately in the ascending aorta. A transgastric short-axis view of the LV showed the presence of the VSR with left-to-right shunting (Figures 3 and 4, Videos 2 and 3). The midesophageal long-axis view of the AV confirmed the VSR with left-to-right flow as visualized by color-flow Doppler (Figure 5, Video 4). Transesophageal echocardiography also showed that the RV was dilated from volume overload with an RV:LV ratio greater than 1 (Figure 6, Video 5). The goal of the pVAD, therefore, was to provide LV support and to reduce the left-to-right shunt.

There was the possibility that the pVAD might reverse the left-to-right flow through the VSR, resulting in hypoxia. As such, pVAD flow was increased in a slow, stepwise fashion under intraoperative TEE guidance in the midesophageal long-axis view until a flow of 3.0 L/min was achieved, at which point norepinephrine and epinephrine requirement decreased. The transgastric short-axis view of the LV showed a decrease in LV diameter and shunt (Figure 7, Video 6). Subsequently, the midesophageal LV long-axis view with color-flow Doppler also helped confirm a continued but significantly decreased left-to-right shunt with decreased overall RV volume overload (Figure 8, Video 7). The patient was then transferred to the cardiothoracic intensive care unit until it was deemed safe to operate on the VSR.

In the intensive care unit, the patient had the pVAD position monitored with serial TTE and mixed venous oxygen saturation. Once-daily TTE was performed to assess pVAD position and monitor left and right heart function or was performed more frequently as necessary for significant hemodynamic changes. Subsequent poor perfusion and RV failure necessitated placement of VA ECMO on postoperative day (POD) 2. Systemic anticoagulation for VA ECMO was accomplished with heparin infusion and monitored with a goal partial thromboplastin time of 45 to 55 seconds. The patient continued on aspirin and ticagrelor antiplatelet therapy for the recent drug-eluting stent as well.

The patient initially improved hemodynamically after ECMO placement, and vasopressors were weaned off. The patient demonstrated some evidence of improved organ perfusion, such as improvement in liver function and creatinine on laboratory studies, as well as decreasing lactate levels. However, oral bleeding and melena developed with worsening anemia on POD6, at which point antiplatelet therapy was stopped. The bleeding resulted in worsening hemodynamics, requiring the resumption of vasopressors and transfusion of

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TEE, midesophageal long-axis (137°) view, demonstrates the pVAD cannula in an appropriate position. Video playback speed was slowed for enhanced clarity.
Video 2: Two-dimensional TEE, transgastric short-axis (0°) view, demonstrates the large VSR pre-pVAD placement. Video playback speed was slowed for enhanced clarity.
Video 3: Two-dimensional TEE, transgastric short-axis (0°) view with color-flow Doppler, demonstrates the large shunt through the VSR pre-pVAD placement.
Video 4: Two-dimensional TEE, midesophageal long-axis (145°) view without *(left)* and with *(right)* color-flow Doppler, demonstrates the shunt through the VSR pre-pVAD placement.
Video 5: Two-dimensional TEE, midesophageal 4-chamber (0°) view, demonstrates normal global LV systolic function, distal septal and apical akinesis, a small LV with a dilated RV, reduced RV systolic function, and leftward septal displacement. Video playback speed was slowed for enhanced clarity.
Video 6: Two-dimensional TEE, transgastric short-axis (0°) view with color-flow Doppler, demonstrates the large left-to-right shunt through the VSR post-pVAD placement. Video playback speed was slowed for enhanced clarity.
Video 7: Two-dimensional TEE, midesophageal long-axis (143°) view with color-flow Doppler, demonstrates the shunt through the VSR and the pVAD cannula position. Video playback speed was slowed for enhanced clarity.

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blood products. Ultimately on POD7, when confronted with uncontrolled bleeding and multiorgan failure, the patient's family requested no further interventions be performed and all care was withdrawn.

DISCUSSION

Ventricular septal repair, a rare complication after acute MI, is difficult to manage due to the hemodynamic instability that frequently develops. Once the rupture occurs, an LV-to-RV shunt develops. This shunt results in volume overload of the RV and pulmonary circulation, followed by possible secondary overload of the left atrium and LV. As forward flow from the LV decreases, systemic vascular resistance increases to compensate for lack of perfusion. This vicious cycle continues until the RV and/or LV fail, resulting in catastrophic hemodynamic compromise and multiorgan failure.

The most recent American College of Cardiology guidelines from 2013 recommend emergency surgical repair for VSR. However, growing evidence suggests that delayed repair may improve mortality rates.[4,6-8] The more recent 2017 European guidelines note the high mortality of early operative repair and suggest that delayed repair may be considered in patients responsive to heart failure therapy, while giving the use of MCS devices in VSR patients with cardiogenic shock a grade IIb recommendation.[2] Some studies on delayed repair in unstable patients using MCS to improve hemodynamics have been promising, with long-term hospital mortality rates around 12.5%.[7] These devices have the benefit of decompressing the ventricle, decreasing oxygen demand and myocardial wall stress while improving overall organ perfusion.[9] In this MCS-assisted delayed approach, the optimal time to perform definitive surgical repair requires a balance between recovery of the tissue for surgical repair and the progression of low-output heart failure refractory to treatment, as well as the risk of complications from the various MCS devices.

We describe the use of a pVAD similar to other case reports that highlight the successful use of such a device to stabilize hemodynamics in patients with VSR.[3] The Impella 5.5 device is Food and Drug Administration approved for short-term LV support. It decreases LV volume, LV wall tension, and myocardial oxygen demand, while providing up to 6.0 L/min of continuous flow. A pVAD may also have a more beneficial hemodynamic profile for VSR patients, as described in a recent computer simulation–based study suggesting that it may improve pulmonary capillary wedge pressure and left-right

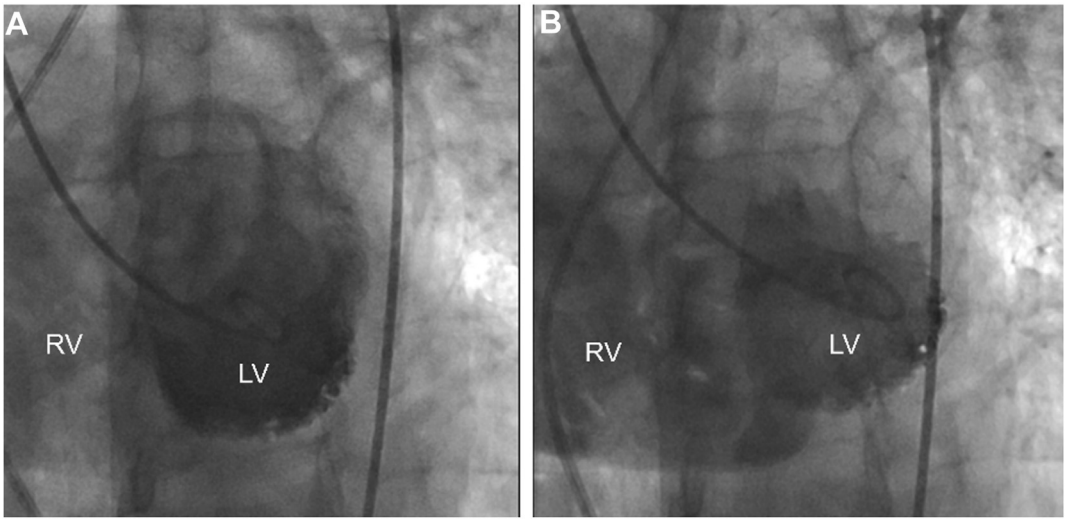

Figure 1 Invasive ventriculogram, fluoroscopic display during **(A)** diastole and **(B)** systole, demonstrates an LV-to-RV shunt.

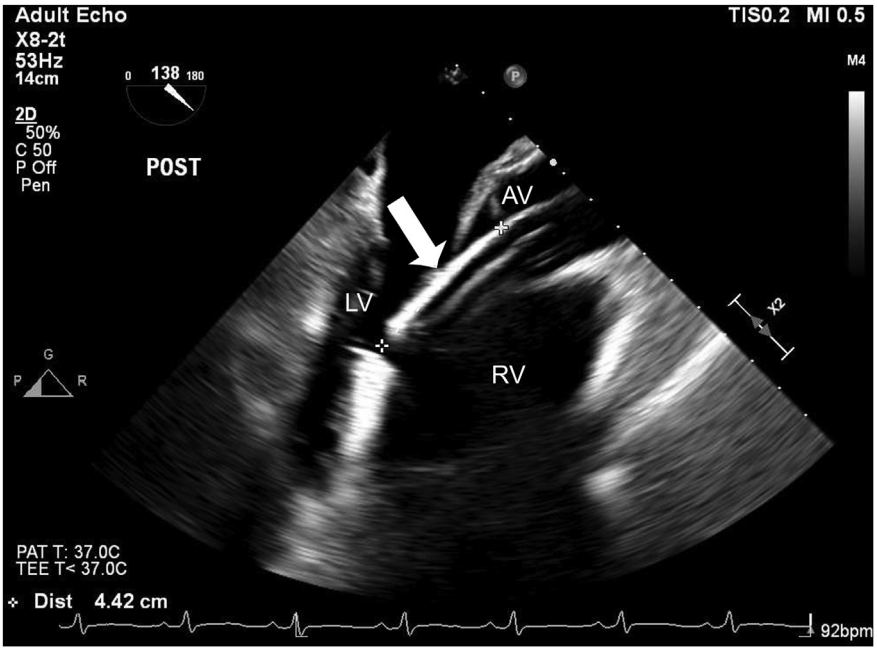

Figure 2 Two-dimensional TEE, midesophageal long-axis (138°) view, demonstrates the pVAD cannula (*arrow*) in an appropriate position, as shown by caliper markers 4.4 cm into the LV.

shunting when compared to other MCS modalities like VA ECMO, which is known to increase afterload, myocardial oxygen demand, LV distention, pulmonary edema, and ventricular wall ischemia.[10] Venoarterial extracorporeal membrane oxygenation also requires systemic heparinization. In this case, RV dilation was noted on initial TEE with normal LV function. After the pVAD was placed, volume overload in the RV decreased as the left-to-right shunt decreased. Overall perfusion improved until POD2 as evidenced by the lower lactate levels. Despite this initial improvement with pVAD, this patient progressed to requiring VA ECMO due to poor end-organ perfusion and RV failure, likely in the setting of worsening cardiogenic shock. The combination of pVAD and VA ECMO resulted in improved hemodynamics and end-organ function. However, this improvement was followed by development of uncontrolled bleeding in the setting of systemic anticoagulation, leading to hemorrhagic shock.

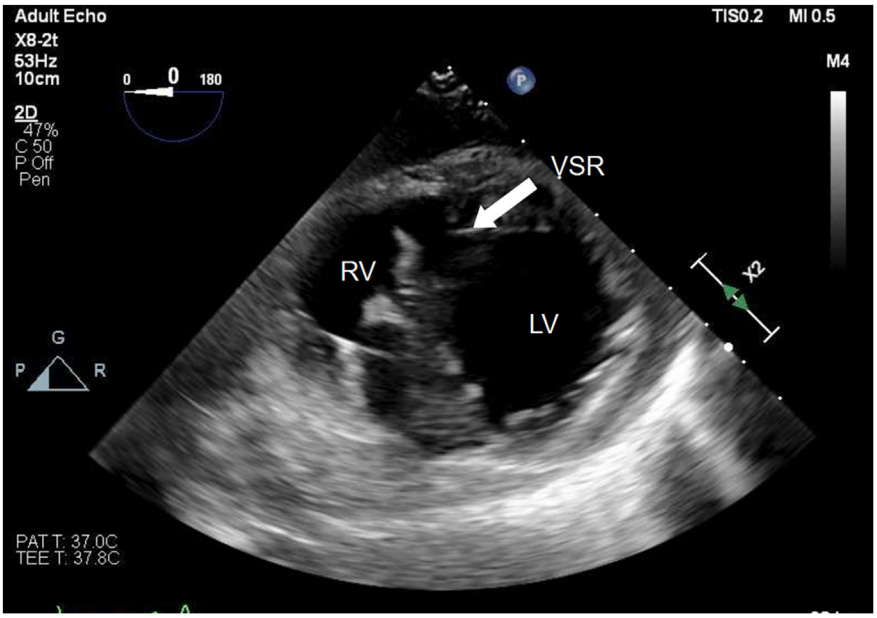

Figure 3 Two-dimensional TEE, transgastric short-axis (0°) systolic view, demonstrates the large VSR pre-pVAD placement.

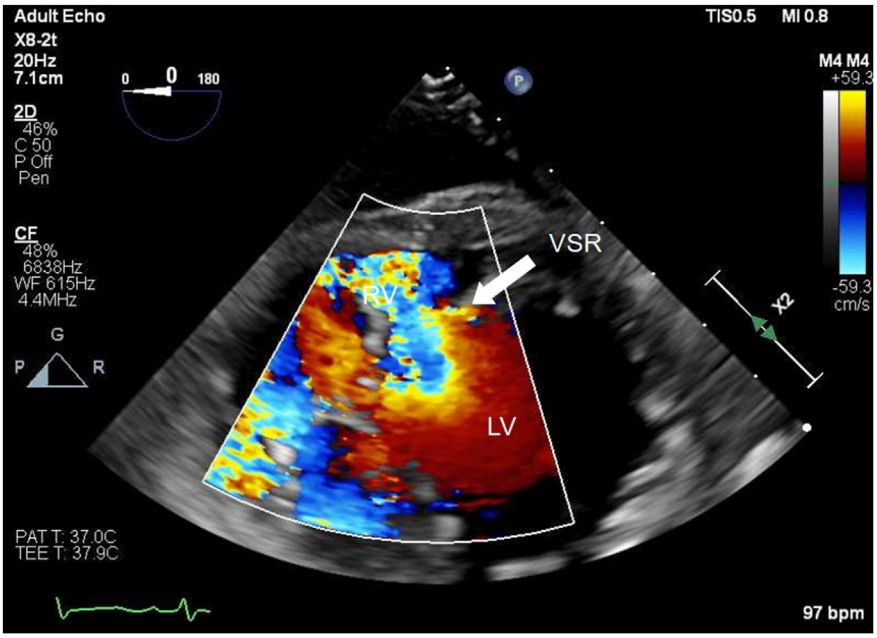

Figure 4 Two-dimensional TEE, transgastric short-axis (0°) systolic view with color-flow Doppler, demonstrates the large shunt through the VSR pre-pVAD placement.

It is possible that earlier institution of VA ECMO along with a pVAD for LV decompression would have resulted in a better outcome, at the risk of earlier systemic heparinization in a patient already loaded with antiplatelet medications. Biventricular VAD support with an independent right-sided device could have also been considered when RV dysfunction became apparent; however, the hemodynamics of such arrangements in the presence of a VSR would be difficult to manage.

While there are other publications reporting on the use of pVAD with or without ECMO as a bridge to surgery in patients with acute VSR complicated by cardiogenic shock, this report focuses on the important role of TEE in guiding both placement and optimization

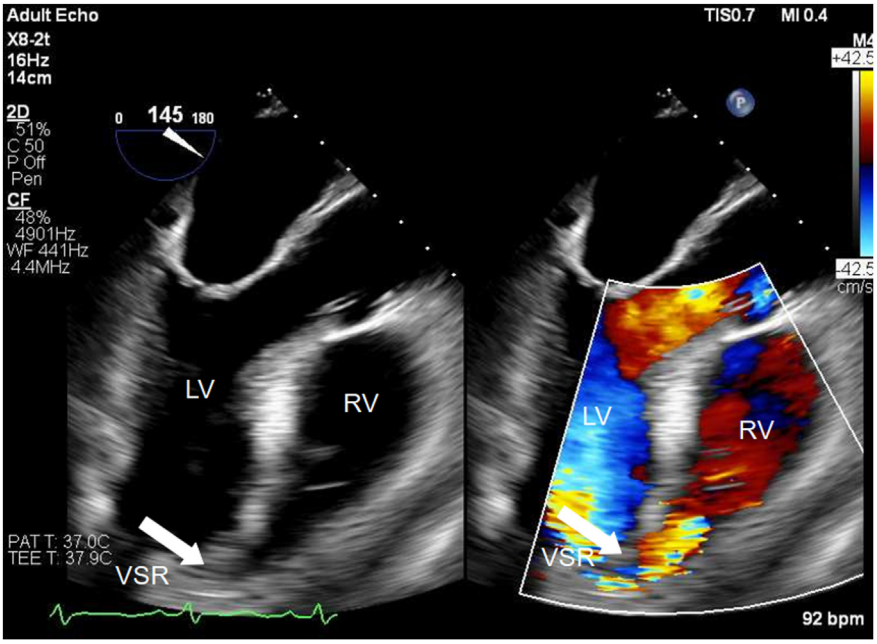

Figure 5 Two-dimensional TEE, midesophageal long-axis (145°) systolic view without (*left*) and with (*right*) color-flow Doppler, demonstrates the shunt through the VSR (*arrow*) pre-pVAD placement.

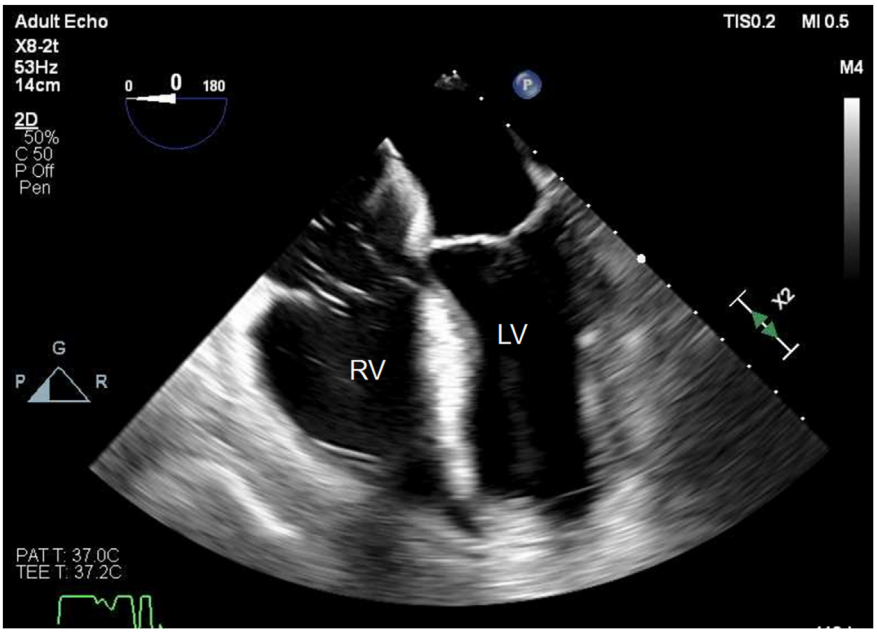

Figure 6 Two-dimensional TEE, midesophageal 4-chamber (0°) end-diastolic view, demonstrates a small LV with a dilated RV and leftward septal displacement.

of the device in the setting of VSR. Transesophageal echocardiography serves a valuable role in assessing the position of the pVAD while evaluating shunt direction and severity. Improper placement can lead to increased LV end-diastolic pressure, myocardial oxygen demand, and physical damage to structures such as the AV, mitral valve, and aorta. Left-to-right shunt reversal because of lower LV volume and pressure is also a possibility. This could worsen hemodynamics and also cause hypoxemia. To avoid this complication, the flow rate of the pVAD needs to be carefully titrated under direct TEE guidance to monitor LV diameter and the shunt direction. We found the midesophageal long-axis view of the AV and transgastric short-axis view of the LV, with color-flow Doppler, to be the most useful for assessing LV volume and shunt flow.

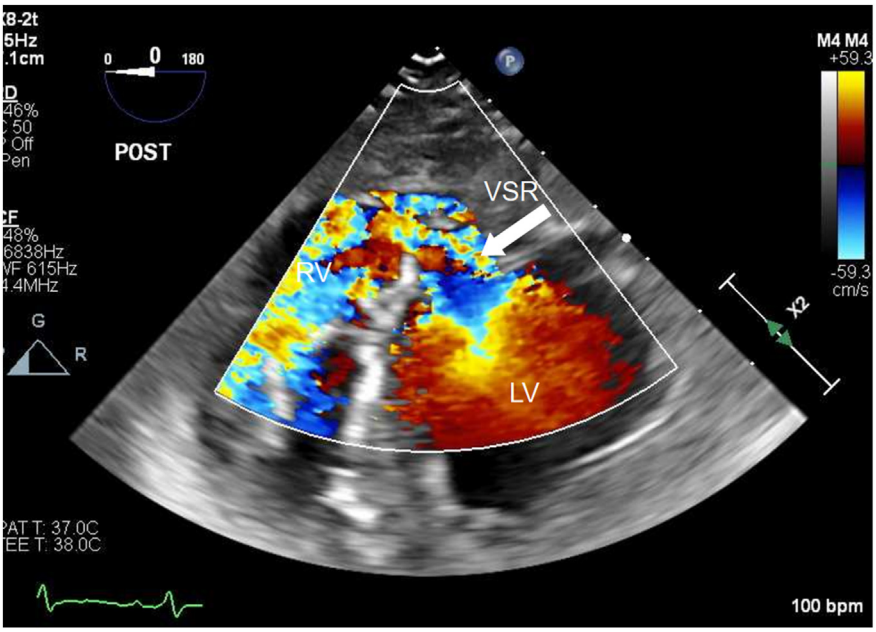

Figure 7 Two-dimensional TEE, transgastric short-axis (0°) late systolic view with color-flow Doppler, demonstrates the shunt through the VSR post-pVAD placement.

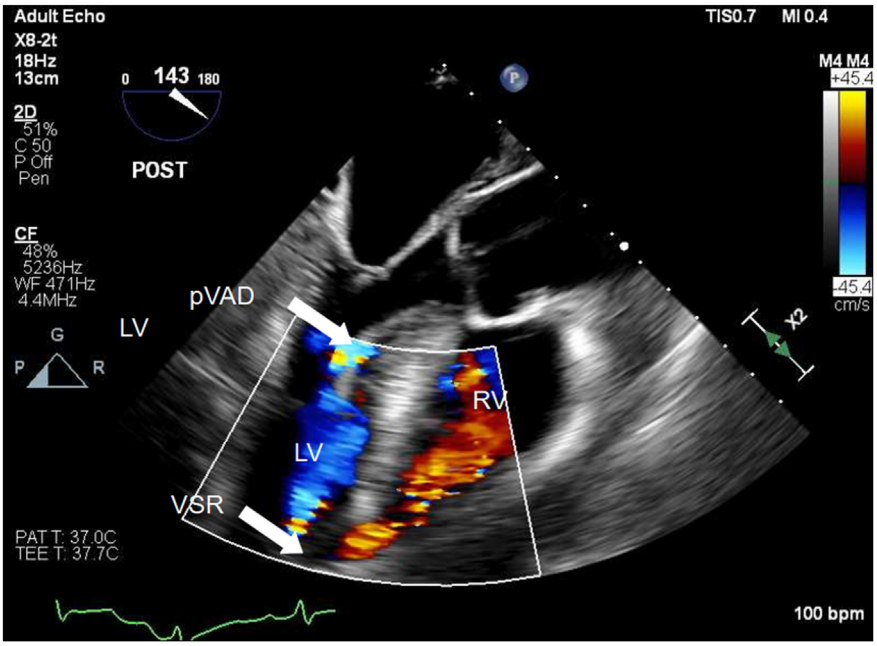

Figure 8 Two-dimensional TEE, midesophageal long-axis (143°) systolic view with color-flow Doppler, demonstrates the shunt through the VSR (*arrow*) and the distal end of the pVAD cannula placement (*arrow*).

CONCLUSION

In this case report, we describe the role of TEE in the placement of a pVAD for hemodynamic support as a bridge to planned surgical repair of a VSR. Transesophageal echocardiography is important for placement of a pVAD; however, in the context of a VSR it was critically important for titrating the flow of the device. When taking this approach, the benefits of delayed operative repair must be weighed against the risks of MCS, such as bleeding. This case serves as an example of both of these benefits and risks, as the patient's cardiogenic shock was initially improved by the use of MCS and they showed some organ recovery; however, systemic anticoagulation led to subsequent uncontrolled bleeding.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

The authors declare that since this was a non-interventional, retrospective, observational study utilizing de-identified data, informed consent was not required from the patient under an IRB exemption status.

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DISCLOSURE STATEMENT

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2024.09.004.

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