CHEST

"Right" or Wrong Diagnosis? The Importance of Transesophageal Echocardiography to Assess the Etiology of Hypotension After Left Ventricular Assist Device Implantation

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A 59-year-old man was admitted to the hospital for cardiogenic shock caused by non-ischemic cardiomyopathy. He experienced acute kidney injury that required temporary hemodialysis. After his condition did not respond to medical treatment, mechanical circulatory support was instituted with the use of the Impella 5.0 (Abiomed, Danvers, MA) to optimize end-organ perfusion. Once his medical condition improved, he was eligible for destination therapy with a durable left ventricular assist device (LVAD).

The patient underwent general anesthesia for LVAD implantation. He was sedated with propofol 40 mg, fentanyl 75 μg, and rocuronium 80 mg. After induction, he became hypotensive with a systemic BP of 60/40 mm Hg. He was treated with epinephrine boluses and continuous infusion of epinephrine 0.04 μg/kg/min. Transesophageal echocardiography (TEE) was performed (Video 1) to assess the etiology of hypotension.

After hemodynamic optimization, a HeartMate 3[1] (Abbott, IL) was implanted without complication. TEE was used to assess cardiac function and LVAD performance (Video 2). Postoperatively, the patient was admitted to the cardiovascular ICU receiving epinephrine 0.07 μg/kg/min, dobutamine 5 μg/kg/min, norepinephrine 0.09 μg/kg/min, and vasopressin 0.01 units/min. His LVAD and hemodynamic parameters are given in Table 1.

Five hours after surgery, the mixed venous oxygen saturation decreased to 51% from the initial 77%. Mean arterial pressure declined from 84 mm Hg to 52 mm Hg (Table 1). Because right ventricular (RV) failure is often the culprit for hemodynamic instability after LVAD implantation, vasoactive infusions were increased to epinephrine 0.12 μg/kg/min, vasopressin 0.06 units/min, and norepinephrine 0.08 μg/kg/min until more data could be obtained. The patient also experienced a fever of 40.4°C. Because of the ongoing hemodynamic instability, a TEE was performed (Videos 3a, 3b, and 3c). The LVAD and hemodynamic parameters at the time of Videos 3a, 3b, and 3c are in Table 1.

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TABLE 1] Patient's Left Ventricular Assist Device and Hemodynamic Parameters

Parameter	Admission to ICU	Videos 3a, 3b, 3c	Increased Left Ventricular Assist Device Speed
Left ventricular assist device flow, L/min	3	3	3.9
Central venous pressure, mm Hg	7	11	10
Pulmonary artery pressure, mm Hg/mm Hg	28/13	29/18	26/16
Mean arterial pressure, mm Hg	84	52	80
Cardiac index, L/min/m^2	2.8	3.1	2.9
Mixed venous oxygen saturation, %	77	51	63
Hear rate, beats/min	86	100	96
Heart rhythm	Sinus	Sinus	Sinus

Question: Based on the interpretation of Videos 3a, 3b, and 3c and the patient's clinical presentation, what is the most likely cause of the hypotension and decreased mixed venous oxygen saturation and what is the recommended treatment?

Answer: Although RV failure is the most common cause of clinical deterioration after LVAD insertion, our TEE findings suggested this diagnosis was unlikely. Videos 3a, 3b, and 3c show a nondilated, hyperdynamic RV, an adequately filled left ventricle (LV), and LVAD inflow cannula in the LV apex. RV failure is less likely the cause of hemodynamic decline, based on these images. Because the patient had fever and tachycardia, systemic inflammatory response syndrome (SIRS) was suspected as the cause of a hyperdynamic RV. To match the patient's metabolic demand, LVAD speed was increased gradually under TEE guidance to optimize oxygen delivery. In addition, the fever was treated aggressively with a cooling blanket and acetaminophen. With increased LVAD speed, the patient's hemodynamic parameters improved (Table 1). As the fever resolved, we used frequent TEE assessments to determine the optimal LVAD speed by observing LV size and septal motion. Once the inflammatory state had subsided, we were able to return the LVAD speed back to its baseline setting

Discussion

LVAD implantations are becoming increasingly common to bridge patients with heart failure to heart transplantation and for destination therapy in those who are not transplantation candidates. A centrifugal LVAD is a continuous, nonpulsatile flow device that is implanted directly in the LV apex with an outflow graft sutured into the ascending aorta.

RV failure is the most common complication post-LVAD implantation occurring in up to 70% of cases.[2] Several physiologic changes occur after LVAD placement that account for the increased risk of right heart failure. One pertains to the role of ventricular interdependence on ventricular septal motion change. Because of the large RV surface area-to-volume ratio, a small reduction in septal-to-wall distance can contribute to large volume displacement. Therefore, when the interventricular septum is flattened or inverted towards the LV during systole owing to the effect of LVAD, it can reduce RV output significantly.[3,4] Also, improved cardiac output after LVAD increases venous return to the right ventricle, which may overwhelm a functionally impaired RV and cause dilation. Leftward shift of the interventricular septum and tricuspid regurgitation lead to suboptimal RV geometry and decreased RV stroke volume, respectively.[5]

Echocardiography is a useful tool to diagnose RV failure quickly after LVAD implantation. Published guidelines on RV size and function are validated with transthoracic echocardiography, although there is insufficient data to validate these measurements for TEE.[6] However, when transthoracic echocardiography views are difficult to obtain because of the postsurgical state, body habitus, or lung disease, TEE is a useful alternative. There are several easily obtained echocardiography parameters that can be assessed at the bedside. It is important to note that, because of the complex crescent shape of the RV, multiple views and measurements are needed to assess function accurately. RV size and function can be evaluated visually with the use of the TEE mid-esophageal four-chamber view or deep transgastric view.[6] Simple visual signals of RV dysfunction can be reflected by RV enlargement and abnormal interventricular septal behavior. Normally, RV size should be less than two-thirds LV size.[6] A dilated RV is concerning for pressure overload, volume overload, or both. Flattening of the interventricular septum during end-diastole signals RV volume overload, whereas septal flattening in end-systole may indicate pressure overload. The presence of significant tricuspid regurgitation can also implicate RV dilation and/or dysfunction. If a tricuspid regurgitation jet is present, a pressure gradient can be calculated across the tricuspid valve that allows for the estimation of pulmonary artery systolic pressure, thereby shedding light on the severity of RV pressure overload. Another quantifiable, yet simple, measurement that can be used to assess RV function is the tricuspid annular plane systolic excursion, which requires M-mode evaluation of the lateral tricuspid valve annulus in the apical four-chamber view to assess longitudinal RV function. RV fractional area of change is another easily performed quantification that measures ventricular area percent change throughout the cardiac cycle. Other more advanced measures, which include tissue Doppler imaging and RV index of myocardial performance, can be used for RV assessment but may be difficult to obtain or interpret.

Once RV failure is diagnosed, therapy includes maintenance of sinus rhythm, augmentation of perfusion with the use of inotropes, vasopressors, inhaled pulmonary vasodilators, and diuresis.[7] Volume management is especially critical because the failing RV is sensitive to changes in volume. Volume optimization

can be achieved by using the aforementioned echocardiographic parameters. Through diuresis and afterload reduction, one can monitor the improvement and treatment responsiveness of RV dysfunction. When RV failure is refractory to conventional treatment, mechanical RV support may be considered.[7]

Video 1 is a midesophageal four-chamber view that shows severely depressed LV and RV function and dilation. This was seen in the operating room after induction of anesthesia when the patient was hypotensive. A pulmonary artery catheter and Impella 5.0 are in place. After hemodynamics were optimized, the RV function and size improved significantly.

Video 2 is a deep transgastric view that shows a nondilated RV with improved function immediately after LVAD implantation in the operating room.

Videos 3a, 3b, and 3c were done in the ICU during the period of hemodynamic instability. Video 3a is a midesophageal four-chamber view with LVAD cannula observed in apex. Video 3b is a transgastric short axis view. Video 3c is a deep transgastric view. These videos show a hyperdynamic, nondilated RV (normal basilar diameter range, 35 to 41 mm) and an adequately filled LV. From this observation, RV failure was unlikely the cause of hypotension and decreasing mixed venous oxygen saturation in the ICU.

In this case, SIRS was suspected to be the primary cause of this patient's hemodynamic deterioration. Though SIRS has been reported rarely in patients who undergo LVAD implantation, it is reported to occur in at least 20% of patients in the acute postoperative period after cardiac surgery.[8] SIRS is defined by two or more symptoms that include fever (>38.0°C) or hypothermia (<36.0°C), tachycardia (heart rate >90 beats/min), tachypnea (>20 breaths/min), and leukopenia ($<4 \times 10^9$/L) or leukocytosis ($>12 \times 10^9$/L). The cause of SIRS after cardiac surgery is multifactorial and includes surgical trauma, hypothermia, blood loss, blood transfusion, and inflammatory response from cardiopulmonary bypass surgery. Exposure of the blood to foreign surfaces, ischemia-reperfusion because of aortic cross-clamping, and endotoxemia because of splanchnic hypoperfusion during cardiopulmonary bypass surgery induce the release of inflammatory mediators.[8] Systemic inflammation results in peripheral vasodilatation, capillary leak, myocardial dysfunction, and major organ dysfunction.[8] Patients who experience SIRS after LVAD are at increased risk of RV failure, infection, and bleeding.[9]

Treatment in this case consisted of temperature management and augmentation of perfusion by increasing LVAD speed to compensate for the increased metabolic demand and to increase cardiac output and end-organ perfusion.

Previous reports have described TEE leading to change in management in up to 50% of cases when used to assess patients with hemodynamic instability in the ICU.[10] Though transthoracic echocardiography is often used and well-validated to evaluate the cardiac condition in patients with heart failure, the existence of surgical wounds and mechanical ventilation can pose a challenge in patients after LVAD.

We report a rare case of acute SIRS after LVAD implantation. To rule out other causes of systemic malperfusion, TEE assessment was instrumental in determination of the diagnosis and guiding therapy. (Discussion Video).

Reverberations

1. *Critical care TEE can be performed in the ICU to diagnosis hemodynamic and physiologic derangements accurately.*
2. *RV failure is common after LVAD implantation. TEE is invaluable in the assessment of RV function.*
3. *SIRS is common after cardiopulmonary bypass surgery and should be treated aggressively to reduce oxygen demand.*

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Additional information: To analyze this case with the videos, see the online version of this article.

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