Heart & Lung 48 (2019) 111–113

Contents lists available at ScienceDirect

Heart & Lung

journal homepage: www.heartandlung.com

Case Report

Right ventricular failure following placement of a percutaneous left ventricular assist device☆

Hassan Alkhawam, MD *, Rahil Rafeedheen, MD, Elsayed Abo-Salem, MD

Center for Comprehensive Cardiovascular care, St Louis University, 3635 Vista Avenue, FDT 13th Floor, Cardiology, St Louis, MO, United States

ARTICLE INFO

Article history:
Received 12 September 2018
Received in revised form 2 December 2018
Accepted 3 December 2018
Available online 11 December 2018

Keywords:
Right ventricular failure
Ventricular assist device
Impella

ABSTRACT

Right ventricular (RV) dysfunction following surgical implantation of a left ventricular assist device (LVAD) is a well-documented phenomenon, and it is associated with poor outcomes. We are reporting a 25-year-old male patient who presented to the hospital with flu-like symptoms, hypotension and acute hypoxic respiratory failure. The patient's Laboratory data was significant for elevated troponin, and his Chest X-ray showed acute pulmonary edema. Echocardiogram revealed reduced left ventricular (LV) ejection fraction and normal RV function. Coronary angiography was normal, and the cardiac index was 1.3 L/min/m^2. Impella 5.0 (Abiomed, MA) was placed through the left axillary artery graft and 4.5 L/min flow was achieved with an improvement in blood pressure. Thirty minutes later, he developed hypotension, the device flow dropped to 3.0 L/min, and right atrial pressure increased. The Pulmonary artery pulsatility index was consistent with RV failure. Possible causes of RV failure include unmasking of RV dysfunction with high LVAD flow and altered RV geometry due to ventricular septum shift. Impella RP (Abiomed, MA) was placed for RV support achieving a flow of 3.8 L/min with a significant improvement in impella LV flow, cardiac output and blood pressure (mean 90 mmHg). Ventricular support devices were weaned off on day 9. The patient was discharged on day 15. Conclusion: our case highlights the risk of RV failure following percutaneous LVAD placement. Early identification and appropriate mechanical support is imperative.

Introduction

Cardiogenic shock is one of the most lethal cardiovascular disorders, associated with a high mortality rate and significant morbidity including longer hospital stay, multi-organ dysfunction and high cost of care. Medical therapy alone is associated with a high mortality rate that is proportional to the number of inotropic and vasopressor agents needed for hemodynamic support and associated organ dysfunction.[1]

The use of percutaneous mechanical circulatory support (PMCS) devices has been increasing recently as a bridge to recovery, decision or destination therapy; either a heart transplant or surgical ventricular assist device. The use of temporary PMCS devices results in significant acute improvement in hemodynamics. However, the mortality and morbidity with cardiogenic shock remain elevated.[2] This can be explained by the delayed institution of circulatory support or inadequate device support.

Acute right ventricular failure (RV) after left ventricular assist devices (LVAD) is encountered in 24% of cases, requiring a right ventricular device or extended periods of inotropic support, and associated with poor clinical outcomes.[3] The incidence of acute right ventricular failure following percutaneous left ventricular support has not been well-described in literature. In this report, we aimed to raise the awareness of the risk of RV failure following percutaneous LVAD placement. It's very crucial to identify the RV failure very early and use the appropriate mechanical support.

Case report

A 25-year-old man with no significant past medical history presented to the hospital with cough, diffuse myalgia, epigastric pain, and progressive dyspnea. Family history was negative for the cardiovascular disease. The examination was remarkable for tachypnea (34/min), tachycardia (140 bpm) and rales on lung auscultation. Blood pressure was 90/60 mmHg, and arterial oxygen saturation was 94% on 15 L O2 through a non-rebreather mask. Chest X-ray showed diffuse airspace opacities consistent with acute pulmonary edema without evidence of cardiomegaly. Initial laboratory data were remarkable for elevated cardiac troponin I (10 ng/dL) and

Abbreviations: LV, left ventricle; RV, right ventricle; PMCS, percutaneous mechanical circulatory support; LVAD, left ventricular assist devices.

☆ **Disclosure:** None

* Corresponding author.
E-mail address: hassan.alkhawam@health.slu.edu (H. Alkhawam).

https://doi.org/10.1016/j.hrtlng.2018.12.002
0147-9563/$ - see front matter © 2018 Elsevier Inc. All rights reserved.

compensated lactic acidosis. The urine drug screen was positive for amphetamines.

The patient was clinically diagnosed with acutely decompensated left-sided heart failure and diuresis was attempted using intravenous furosemide, but there were little urine output and no improvement in his respiratory status. The patient's respiratory status continued to worsen and required emergent endotracheal intubation. A transthoracic echocardiogram was performed which revealed severely reduced left ventricular ejection fraction (25%) without regional wall motion abnormalities. RV size and systolic function were normal. Initial suspected diagnosis was myocarditis versus amphetamine-induced cardiomyopathy. Inotropic support was provided with milrinone at 0.25 mcg/kg/min. The patient was urgently brought to the cardiac catheterization laboratory with plans for coronary angiography along with right and left heart catheterization. Coronary arteries were angiographically normal. Hemodynamic data from cardiac catheterization are listed in Table 1.

Pelvic/femoral angiography revealed diffuse iliac and femoral artery spasm precluding percutaneous insertion of an impella device via the femoral approach. The patient was started on norepinephrine, and a 40 ml intra-aortic balloon pump was inserted for hemodynamic support through the femoral artery. The cardiac output was relatively unchanged, but the augmented diastolic pressure was 120 mmHg and mean arterial pressure was 90 mmHg. Six hours later, he had clinical deterioration with increase in heart rate to 180 bpm (sinus tachycardia), decrease in intra-aortic balloon pump augmentation to 90 mmHg and drop in mean arterial pressure to 48 mmHg requiring vasopressor support in addition to inotropes. Serum lactic acid increased from 3.1 to 5.5 mmol/L.

Hemodynamic support was escalated by inserting an Impella 5.0 after gaining access to the left axillary artery. A flow of 4.5 L/min was achieved with the impella with improvement in mean arterial pressure to 80 mmHg and cardiac output increased from 2.3 L to 4.7 L/min (cardiac index 2.65 L/min/m^2). The rest of the hemodynamic data are listed in Table 1.

Thirty minutes later, right atrial pressure acutely increased from 5 mmHg to 18 mmHg, and device flow dropped to 3.0 L/min, and blood pressure decreased (Table 1). Pulmonary artery pulsatility index (0.6) was consistent with RV failure. Right ventricular mechanical support was promptly initiated by inserting an Impella RP through the right femoral vein achieving a flow rate of 3.8 L/min following which the left ventricular device flow increased to 4.0 L/min. Fig. 1

His clinical condition improved immediately. All vasopressors were weaned off within 6 hours. Lactic acid trended down, and urine output increased within 24 hours. Mechanical support was weaned down for both ventricles. Impella RP device was removed on day 5. A repeat echocardiogram on day 6 revealed an improvement in left ventricular systolic function. Impella 5.0 was weaned off on day 9. The patient was discharged from the hospital on day 14.

Discussion

Although others have reported right ventricular dysfunction after LVAD implantation,[4] we are reporting the first case report of acute right ventricular failure after percutaneous mechanical circulatory support device insertion. Prior data suggested a low risk of RV dysfunction with percutaneous mechanical support devices; however, these conclusions were limited by the low flow with percutaneous compared to surgical LVAD.[5] Current technology permitted the development of cardiac mechanical devices with higher flow and lower profile. These high flow pumps are likely to gain more use in the future, and subsequently higher risk of RV dysfunction like surgical LVAD. Early recognition of acute right ventricular failure and biventricular support in these cases can provide a superior outcome among cases with percutaneous left ventricular devices.[6]

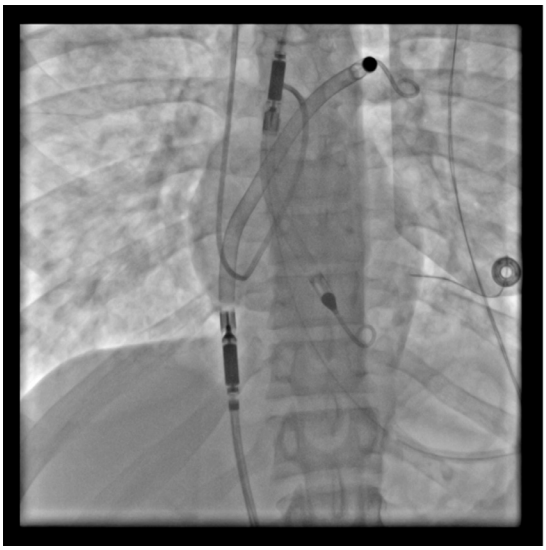

Fig. 1. Chest X-ray shows left ventricular impella 5.0 through left axially axis, impella RP through right femoral artery access and Swan Ganz catheter through right internal jugular access.

The mechanism of RV failure after left-sided mechanical support is not clear but is probably related to a concealed right ventricular dysfunction that was unmasked with increased left ventricular cardiac output following placement of a percutaneous LVAD. LVADs also create a negative pressure in the left ventricle during systole leading to deviation of the interventricular septum to the left. This contributes to RV dysfunction due to distortion of the RV geometry and loss of septal contribution to right heart contractility. Submaximal flow can potentially decrease septal shift and RV volume overload and improve hemodynamics. Several parameters have been identified as predictors of acute RV failure requiring RV mechanical support following implantation of an LVAD[7–9] (Table 2).

After initiation of an LVAD, acute RV failure may present with increased right ventricular filling pressures, decreased cardiac output, increased requirement for vasopressor/inotropic agents, acute kidney and liver dysfunction and suction events in the LV device with the decreased flow. Echocardiography can show dilated RV and right atrium, new or worsening tricuspid regurgitation and decreased RV function. Findings can be confirmed with a heart catheterization.

Table 1
Hemodynamic data from right heart catheterization

	Milrinone 0.25 mcg/kg/min	Immediately after Impella 5.0	30 min later after Impella 5.0
MAP (mmHg)	71	80	60
RA pressure (mmHg)	5	5	18
RV pressure (mmHg)	25/5	25/5	34/18
PA pressure (mmHg)	25/22/23	24/18 (20)	34/22 (26)
Heart rate (bpm)	132	120	150
Fick Cardiac output (L/min)	2.3	4.7	3.5

MAP: Mean arterial pressure; RA: Right atrium; RV: Right ventricle; PA: Pulmonary Artery.

Table 2
Predictors of acute right ventricular failure following surgical LVAD

Hemodynamic data	Other parameters
CVP > 15 mmHg	Severe TR
CVP/PCWP ratio > 0.63	RV dilation and dysfunction
PAPi ratio < 1	Previous cardiac surgery
PVR > 2 woods units	Creatinine ≥ 1.9 mg/dL
RV SWI < 0.3 mmHg. L/m2	Pulmonary embolism

CVP: Central venous pressure; PCWP: Pulmonary capillary wedge pressure; PAPi: Pulmonary artery pulsatility index; PVR: Pulmonary vascular resistance; RV SWI: Right ventricular stroke work index.

Studies in patients with surgical LVAD have shown that the RV function may improve after a few days leading to improved intermediate-term outcomes.[10] However, there is a need for temporary RV hemodynamic support in the immediate post-operative period. Two percutaneous mechanical circulatory support devices are currently available for RV hemodynamics support; Impella RP (Abiomed, MA) and Protek Duo (CardiacAssist Technologies, Pittsburgh, PA). Most clinical trials for percutaneous right ventricular support devices are limited to small single-center trials in the setting of surgical LVADs.[11,12] These studies have revealed significantly improved right ventricular function with right-sided PMCS. Planned or early use of mechanical RV support is also associated with better outcomes compared to a delayed rescue intervention after failed medical therapy.[2]

Our case report highlights the risk of acute RV failure following placement of an impella 5.0 (Abiomed, MA) and benefit of a percutaneous right ventricular assist device for RV failure. As the use of PMCS device increases, further trials are needed to identify the incidence and outcome of right ventricular dysfunction after LV percutaneous device support.

Conclusion

Right ventricular dysfunction can lead to failure of percutaneous left ventricular mechanical circulatory support device leading to poor clinical outcomes. Early identification of this phenomenon and proper management including use of right ventricular mechanical support is mandatory. It is imperative to assess the right ventricular function before and immediately after initiation of left ventricular mechanical circulatory support.

References

1. Tarvasmäki T, et al. Current real-life use of vasopressors and inotropes in cardiogenic shock - adrenaline use is associated with excess organ injury and mortality. *Crit Care*. 2016;20:208.
2. Mozaffarian D. Executive summary: heart disease and stroke statistics–2016 update: a report from the American heart association. *Circulation*. 2016;133 (Jan 26 (4)):447–454.
3. Slaughter MS, et al. Advanced heart failure treated with continuous-flow left ventricular assist device. *N Engl J Med*. 2009;361(23):2241–2251.
4. Dang NC, et al. Right heart failure after left ventricular assist device implantation in patients with chronic congestive heart failure. *J Heart Lung Transplant*. 2006;25 (Jan (1)):1–6.
5. Yourshaw JP, et al. Effects of Percutaneous LVAD Support on Right Ventricular Load and Adaptation. *J Cardiovasc Trans Res* 2018. https://doi.org/10.1007/s12265-018-9806-0.
6. Fitzpatrick JR, et al. Early planned institution of biventricular mechanical circulatory support results in improved outcomes compared with delayed conversion of a left ventricular assist device to a biventricular assist device. *J Thorac Cardiovasc Surg*. 2009;137(4):971–977.
7. Neragi-Miandoab S, et al. Right ventricular dysfunction following continuous flow left ventricular assist device placement in 51 patients: predicators and outcomes. *J Cardiothorac Surg*. 2012;7:60.
8. Kormos RL, et al. Right ventricular failure in patients with the HeartMate II continuous-flow left ventricular assist device: incidence, risk factors, and effect on outcomes. *J Thorac Cardiovasc Surg*. 2010;139(5):1316–1324.
9. Fitzpatrick JR, et al. Risk score derived from pre-operative data analysis predicts the need for biventricular mechanical circulatory support. *J Heart Lung Transplant*. 2008;27(12):1286–1292.
10. Maeder MT, et al. Changes in right ventricular function during continuous-flow left ventricular assist device support [corrected]. *J Heart Lung Transplant*. 2009;28 (4):360–366.
11. Anderson MB, et al. Benefits of a novel percutaneous ventricular assist device for right heart failure: the prospective recover right study of the Impella RP device. *J Heart Lung Transplant*. 2015;34(12):1549–1560.
12. Haneya A, et al. Temporary percutaneous right ventricular support using a centrifugal pump in patients with postoperative acute refractory right ventricular failure after left ventricular assist device implantation. *Eur J Cardiothorac Surg*. 2012;41 (1):219–223.