ARTICLE IN PRESS
CARREV-01972; No of Pages 3
Cardiovascular Revascularization Medicine xxx (xxxx) xxx
Contents lists available at ScienceDirect
Cardiovascular Revascularization Medicine

Abrupt development of a trans-aortic valve gradient in the setting of acute left-sided circulatory support identifies right heart failure in cardiogenic shock: The Kapur-Langston sign

Navin K. Kapur [a,*], Phillip Langston [b], Michele L. Esposito [a], Daniel Burkhoff [c]

[a] The Cardiovascular Center, Tufts Medical Center, Boston, MA, United States of America
[b] The Oklahoma Heart Institute, Tulsa, OK, United States of America
[c] Cardiovascular Research Foundation, New York, NY, United States of America

Over the past decade, the use of durable left ventricular assist devices (LVAD) and acute mechanical circulatory support (MCS) pumps to support the failing left ventricle (LV) has grown exponentially. However, worsening right ventricular (RV) failure after activation of a LVAD is a major cause of morbidity and mortality and often requires prolonged inotropic support or use of an acute MCS pump to support RV function. Non-surgical options for acute RV MCS include the Impella RP axial flow catheter, veno-arterial extracorporeal membrane oxygenation (VA-ECMO), or the TandemHeart right atrial-to-pulmonary artery centrifugal pump [1]. Recent studies have identified that early institution of RV MCS in the setting of RV failure may lead to improved clinical outcomes [2]. However, little is known about the impact of percutaneous LV MCS pumps (i.e. Impella CP and 5.0) in the setting of RV failure.

We now report 3 consecutive patients with cardiogenic shock and RV failure where the combination of elevated central venous pressures and low LV-Impella device flows were associated with worsening RV failure necessitating initiation of RV support.

1. Case one

A 40-year-old man was resuscitated from an out of hospital cardiac arrest and underwent primary stenting of the left anterior descending artery (LAD) for an acute anterior ST-segment elevation myocardial infarction (STEMI). Despite revascularization, the patient remained in cardiogenic shock refractory to inotropes and an intra-aortic balloon pump (IABP). An echocardiogram showed an LV ejection fraction (LVEF) of 15% with normal RV dimensions, however pulmonary artery (PA) catheter indices suggested RV dysfunction (Table 1). A double-lumen pigtail catheter and an Impella CP axial flow pump were inserted into the LV. Upon activation of the Impella CP, LV systolic pressure rapidly declined while aortic pressure became minimally pulsatile. The aortic to LV (AO-LV) gradient was 50 mmHg (Fig. 1). Despite maximal rotations per minute (RPMs) (P-level 8), flows of 1.8 liters per minute (LPM) were noted along with suction alarms and cardiac index remained unchanged. Echocardiography showed a dilated RV. VA-ECMO using bifemoral cannulation was performed to provide RV mechanical support. The Impella CP was left in place at P4 to decompress the LV while on VA-ECMO. Over the next 5 days, vasopressors were weaned, all devices were removed, and the patient was discharged home. Three months later, the LVEF was 55% with normal RV function.

2. Case two

A 40-year old woman presented with chest pain and underwent stenting of a mid-LAD stenosis. Despite revascularization, the patient developed cardiogenic shock. An echocardiogram showed biventricular failure with an LVEF of 10%. PA catheter indices suggested RV dysfunction (Table 1). A double-lumen pigtail catheter was inserted into the LV followed by an Impella CP axial flow pump. Upon activation of the LV AMCS pump, LV systolic pressure declined while aortic pressure became minimally pulsatile. The AO-LV gradient was 70 mmHg (Fig. 1). Despite maximum RPMs, device flows were only 2.4 LPM and several suction alarms occurred. RPMs were reduced to relieve LV suction and an Impella RP axial flow pump inserted into the RV. Impella CP flows improved to 3.1 LPM. Over the next 7 days, vasopressors were weaned, the Impella CP, then Impella RP were removed and the patient was discharged home. Three months later, LVEF was 40% with normal RV function.

3. Case three

A 70-year-old man with chronic non-ischemic cardiomyopathy presented with biventricular failure due to profound volume overload. Echocardiography showed an LVEF of 10% with severe RV systolic dysfunction. PA catheter indices suggested RV dysfunction (Table 1). A double-lumen pigtail catheter and an Impella CP axial flow pump were inserted into the LV. Upon activation of the Impella CP, LV systolic pressure declined while Ao pressure became minimally pulsatile. The AO-LV gradient was 60 mmHg (Fig. 1). Despite maximal RPMs, Impella CP flows were 2.1 LPM. An Impella RP axial flow pump was inserted into the RV and Impella CP flows improved to 3.2 LPM. Over the next 5 days, vasopressors were weaned, the Impella RP, then Impella CP were

* Corresponding author at: The Cardiovascular Center, Tufts Medical Center, 800 Washington Street, Box # 80, Boston, MA 02111, United States of America.
E-mail address: Nkapur@tuftsmedicalcenter.org (N.K. Kapur).

https://doi.org/10.1016/j.carrev.2020.05.043

Table 1
Hemodynamic data.

	Patient 1			Patient 2			Patient 3		
	Baseline	Post-CP	ECMO + CP	Baseline	Post-5.0	5.0 + RP	Baseline	Post-CP	CP + RP
RA (mmHg)	18	22	16	20	44	10	24	28	16
PA (mmHg)	30/24	28/24	40/22	49/28	32/22	34/23	47/34	38/22	42/26
RA:PCWP	1.5	–	0.89	0.8		0.5	0.9		0.7
PCWP (mmHg)	12	–	18	26	–	20	26	–	22
Fick CI (L/min/m^2)	1.6	–	3.98	1.58	–	2.3	1.96	–	2.43
PA sat (%)	43	–	73	49	–	57	50	–	57
LV systolic (mmHg)	67	30	N/A	62	30		82	5	
Ao systolic (mmHg)	65	70	N/A	62	78		70	63	
MAP (mmHg)	42	45	69	60	–	65	88		65
PAPi	0.3	0.18	1.1	1.05	–	1.1	0.5		1.2
SVR (dynes-s-cm^5)	914	–	694	1146	–	–	2132		820
Lactate (mEq/L)	8.7	–	4.7	1.5	–	1.5	4.2		5.7
Creatinine (mg/dL)	1.71	–	1.15	2.17	–	0.66	0.81		3.63
BSA (m^2)	1.91	–	–	1.9	–	–	1.53		–

removed. Despite maximal medical therapy, the patient had worsening multi-organ failure and subsequently expired.

4. Discussion

We report three cases of cardiogenic shock where the combination of an elevated central venous pressure, low Impella flow, and suction alarms were associated with the abrupt development of an AO-LV gradient and subsequent RV failure requiring additional mechanical support. These findings suggest that operators may identify subclinical RV failure by either observing low Impella flows and a high RA pressure (without the need for a full PA catheter) or by monitoring AO-LV pressures alone at the time of left heart Impella support. Identification of subclinical RV failure should prompt further evaluation with quantification of RA:PCWP ratios, the PAPi, and other imaging modalities to identify concomitant RV failure in cardiogenic shock.

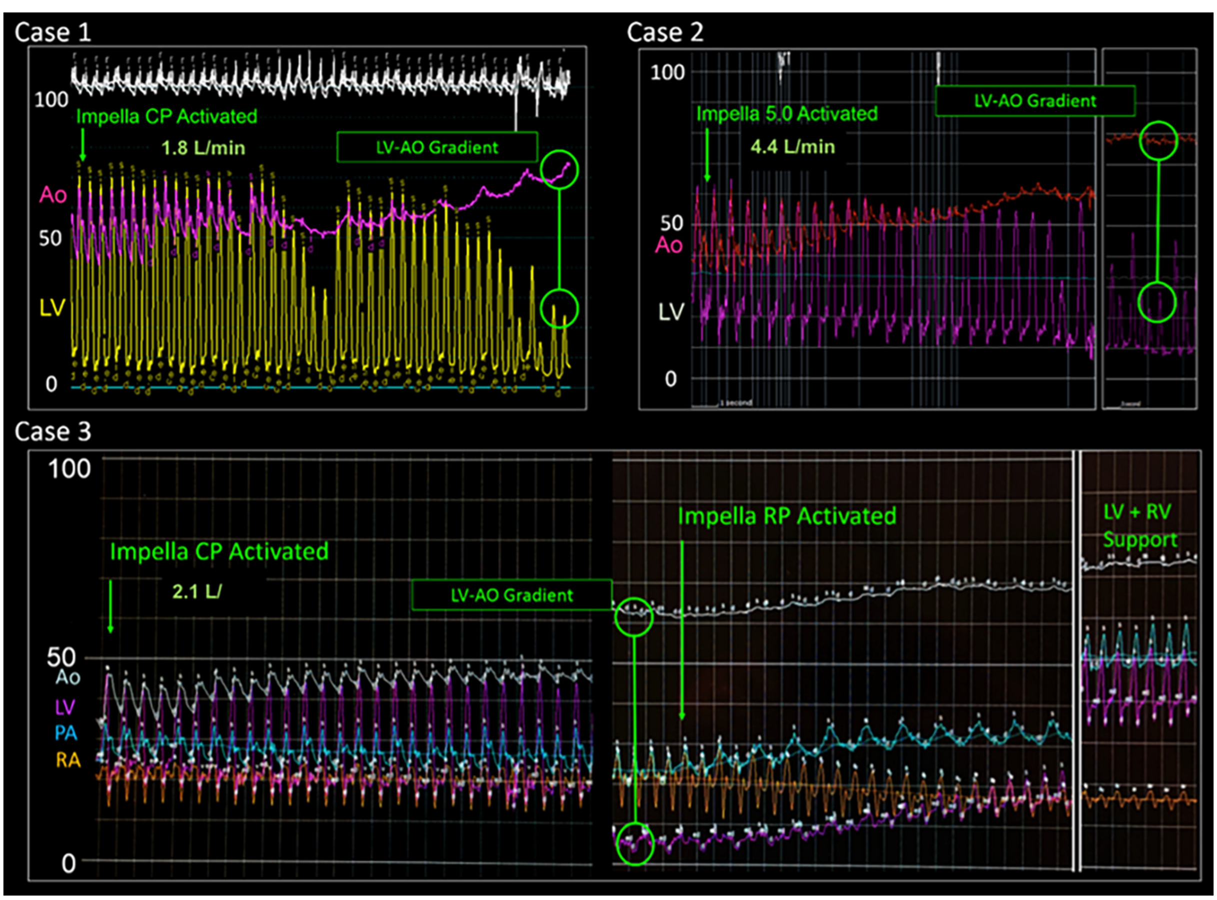

Fig. 1. Three cases illustrating the Kapur-Langston sign. Hemodynamic tracings from three patients with biventricular failure demonstrate the abrupt development of a gradient between the left ventricle (LV) and aorta (AO) as a marker of worsening RV failure after activation of a left sided axial flow catheter (Impella CP or 5.0). In case 3, activation of an Impella RP bypass the failing RV and restores LV preload. Impella RP activation decreases right atrial (RA) pressure and increases pulmonary artery (PA) pressure.

Please cite this article as: N.K. Kapur, P. Langston, M.L. Esposito, et al., Abrupt development of a trans-aortic valve gradient in the setting of acute left-sided circulatory s..., Cardiovascular Revascularization Medicine, https://doi.org/10.1016/j.carrev.2020.05.043

Full activation of a surgical LVAD with high flow may uncouple peak LV-Ao systolic pressures. However, we and others have previously reported that when an LVAD is dysfunctional, peak AO-LV systolic pressure is not uncoupled and the aortic valve opens with every beat [3,4]. We now describe a novel observation where despite activation of a LV-Impella device at maximal RPMs, low flows were associated with abrupt development of a AO-LV systolic pressure gradient. This finding is suggestive of inadequate LV preload, which may be due to RV failure, hypovolemia, cardiac tamponade, pulmonary embolus, or stenosis of the tricuspid, pulmonic, mitral valves or pulmonary veins. In each these cases, hemodynamic data suggested RV failure before LV-Impella activation (Table 1) and once RV support was provided in two patients with an Impella RP, then LV-Impella flows, LV systolic pressure, and MAP improved.

In cardiogenic shock elevated right heart filling pressures may be due to volume overload, not RV failure. In these cases, initiation of LV support with an Impella device may increase cardiac output and systemic perfusion. However in a subset of patients, activation of the left sided pump may worsen subclinical RV failure. Echocardiography is an excellent way to identify RV failure, however as illustrated in these cases, hemodynamic data, which is acquired in real-time during the catheterization procedure, can quickly distinguish venous congestion from RV failure in the presence of left sided support. Furthermore, if RV failure is not identified in a rapid manner, patients may receive partial support from a left sided pump, but continue experiencing suction events, which lead to hemolysis and have progressive worsening of RV failure due to increased venous return. To avoid this vicious cycle of deterioration, methods to rapidly diagnose RV failure may help operators rapidly optimize patients before leaving the catheterization laboratory.

5. Conclusion

Using a dual-lumen pigtail catheter to identify the abrupt development of AO-LV gradient in the setting of low LV-MCS flows may allow for rapid, intra-procedural detection and early treatment of worsening RV failure and improved patient outcomes. Further studies are required to test the sensitivity and specificity of this hemodynamic sign as a predictor of the need for a concomitant RV MCS device in the setting of biventricular failure.

Declaration of competing interest

Dr. Kapur receives research funding from Abiomed Inc., Cardiac Assist Inc., Maquet Inc., and speaker/consultant honoraria from Abiomed Inc., Maquet Inc., and Heartware Inc. None of the remaining authors have relevant disclosures. Dr. Burkhoff receives consulting/speaking honoraria from Abiomed.

References

[1] Kapur NK, Esposito M. Hemodynamic support with percutaneous devices in patients with heart failure. Heart Fail Clin. 2015;11:215–30.

[2] Kormos RL, Teuteberg JJ, Pagani FD, Russell SD, John R, Miller LW, 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;139:1316–24.

[3] Myers TJ, Bolmers M, Gregoric ID, Kar B, Frazier OH. Assessment of arterial blood pressure during support with an axial flow left ventricular assist device. J Heart Lung Transplant. 2009 May;28(5):423–7.

[4] Kapur NK, Jumean M, Halin N, Kiernan MS, DeNofrio D, Pham DT. Ventricular square-wave response: case illustrating the role of invasive hemodynamic in the management of continuous-flow left ventricular assist device dysfunction. Circ Heart Fail. 2015 May;8(3):652–4.