

Letter to the Editor

Catheter-Based Left Ventricular Assist Device for the Management of Cardiogenic Shock Complicating Acute Myocardial Infarction: A First-in-Singapore Experience

Dear Editor,

A 48-year-old Indian man was seen in the emergency department for acute chest pain and was diagnosed with acute anterior ST-segment elevation myocardial infarction (STEMI). Coronary angiogram revealed culprit 100% occlusion of the proximal left anterior descending (LAD) artery and incidental 90% stenosis in the proximal right coronary artery (RCA). He underwent primary percutaneous coronary intervention (PCI) to the LAD artery occlusion. However, the patient had persistent chest pain and tachycardia of around 125 beats per minute (bpm) after primary PCI. A transthoracic echocardiogram was performed, showing severely depressed left ventricular ejection fraction (LVEF) of 20%, with no mechanical complications of acute myocardial infarction (AMI). Despite normal mean arterial pressure (MAP) of >65mmHg, serum lactate level was elevated at 2.5mmol/dL (normal <2.1mmol/dL), indicative of end-organ hypoperfusion. Central venous oxygen saturation obtained from the inferior vena cava (IVC) measured 43% (normal >65%). He was diagnosed with early cardiogenic shock (CS). Decision was then made to revascularise the residual RCA lesion, with implantation of an intra-aortic balloon pump (IABP). Despite all these measures, the patient continued to be tachycardic with a heart rate of >120 bpm, and persistently elevated serum lactate level of 2.3mmol/dL 36 hours after initial presentation. Repeat central venous oxygen saturation from the IVC measured 31.4%. He also started to develop shortness of breath. His CS was progressing despite the treatment he received, and he was developing acute pulmonary oedema.

The in-house multi-disciplinary CS team was activated. Invasive haemodynamics were obtained using right heart catheterisation, showing elevated left and right heart filling pressures (Table 1). The cardiac index was also significantly reduced at 1.74L/min/m² (normal 2.5–4.0L/min/m²). These findings were consistent with CS from acute left ventricular (LV) failure. Right heart function was acceptable, given the pulmonary artery pulsatility index (PAPi) of 1.1. A

decision was made to implant a catheter-based left ventricular assist device (cVAD), the Impella CP® (Abiomed Inc., Danvers, USA), via the right femoral artery, for LV support. After cVAD implantation, he was put on maximum support (Performance level 8, 'P8'. Range: 0–8), which provided 3.5L/min of additional cardiac output. Marked improvement in the patient's LV filling pressures and cardiac index were seen (Table 1). The patient had complication of cVAD insertion site bleeding on day 1 of implantation, requiring 1 unit of packed cell transfusion and temporary withdrawal of heparin infusion. After this initial complication, the patient tolerated subsequent cVAD support well, avoiding the need for endotracheal intubation or initiation of inotropic medications. cVAD position in the LV was checked daily using chest x-ray (Figure 1) and echocardiogram (Figure 2). Adequate anticoagulation was achieved with unfractionated heparin infusion, maintaining activated partial thromboplastin time (aPTT) of 55–80 seconds. Weaning of support level was attempted twice daily, guided by invasive haemodynamic parameters. After 4.5 days of cVAD support, the patient had normal haemodynamics on minimal cVAD support at 'P2'. He was deemed to have recovered sufficient native heart function for cVAD explantation. Patient was discharged from the hospital 3 days after cVAD explantation. He spent a total of 10 days in the hospital.

Discussion

CS is a serious complication of AMI, occurring in 5–15% of cases.¹ It is defined as (1) systolic blood pressure of <90mmHg for >30 minutes, or the need of vasoactive medications to maintain a systolic blood pressure of >90mmHg and (2) evidence of end-organ hypoperfusion. Despite high rates of primary revascularisation therapy in the modern era, early mortality for patients with AMI complicated by cardiogenic shock (AMI-CS) remains high at 40–50%.^{2,3} No inotropic therapy has been shown to improve survival in this group of patients. Early mechanical circulatory support (MCS) and multi-disciplinary 'shock

Table 1. Haemodynamic parameters during catheter-based left ventricular assist device support. 'P' denotes performance level (range 0–8).

Parameter	Prior to support	P8 support	P2 support	Normal values
Mean arterial pressure (mmHg)	101	75	75	>65
Right atrial pressure (mmHg)	16	11	10	<8
Pulmonary artery pressure (mmHg)	42/24	30/20	25/15	<30/12
Pulmonary artery wedge pressure (mmHg)	24	18	13	<15
Mixed venous oxygen saturation (%)	62.1	57.5	66.9	>65
Cardiac output (L/min)	2.95	3.89	4.8	4.0 – 8.0
Cardiac index (L/min/m ²)	1.74	2.29	2.82	2.2 – 4.0
Pulmonary artery pulsatility index (PAPi)	1.12	0.91	1.0	>0.9
Systemic vascular resistance (dynes-sec-m ⁻⁵)	2305	1312	1083	800 - 1500
Pulmonary vascular resistance (Wood units)	2.03	1.79	1.04	<1.5

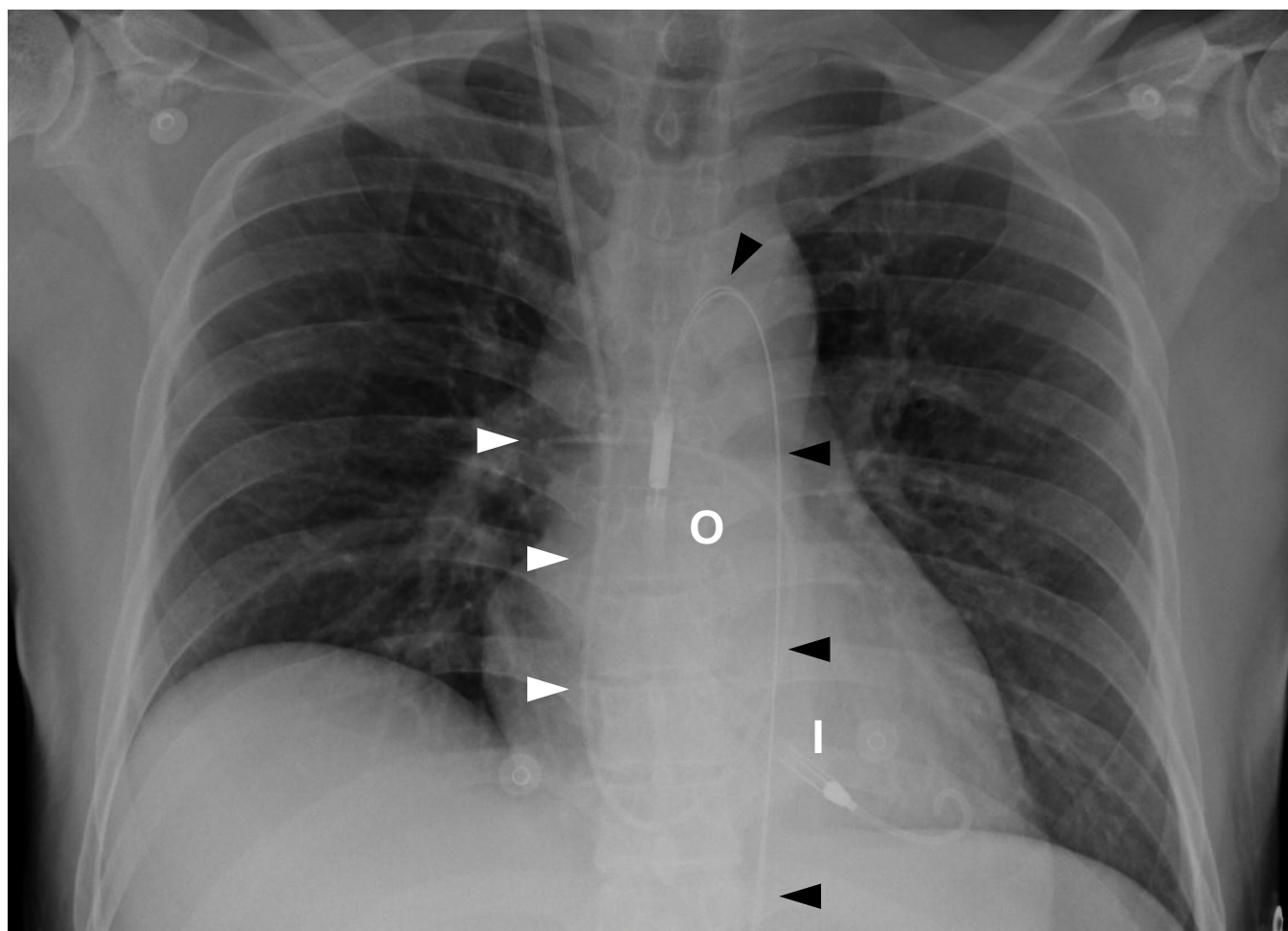


Fig. 1. Chest radiograph showing pulmonary artery catheter (white arrowheads) and the Impella CP® catheter-based left ventricular assist device (cVAD) (black arrowheads). The pulmonary artery catheter was inserted from the right internal jugular vein, with its tip in the proximal right pulmonary artery. The cVAD was inserted from the right femoral artery, with its tip sitting in the left ventricle. 'I' denotes the inlet of the cVAD in the left ventricle and 'O' denotes the outlet of the cVAD in the ascending aorta.

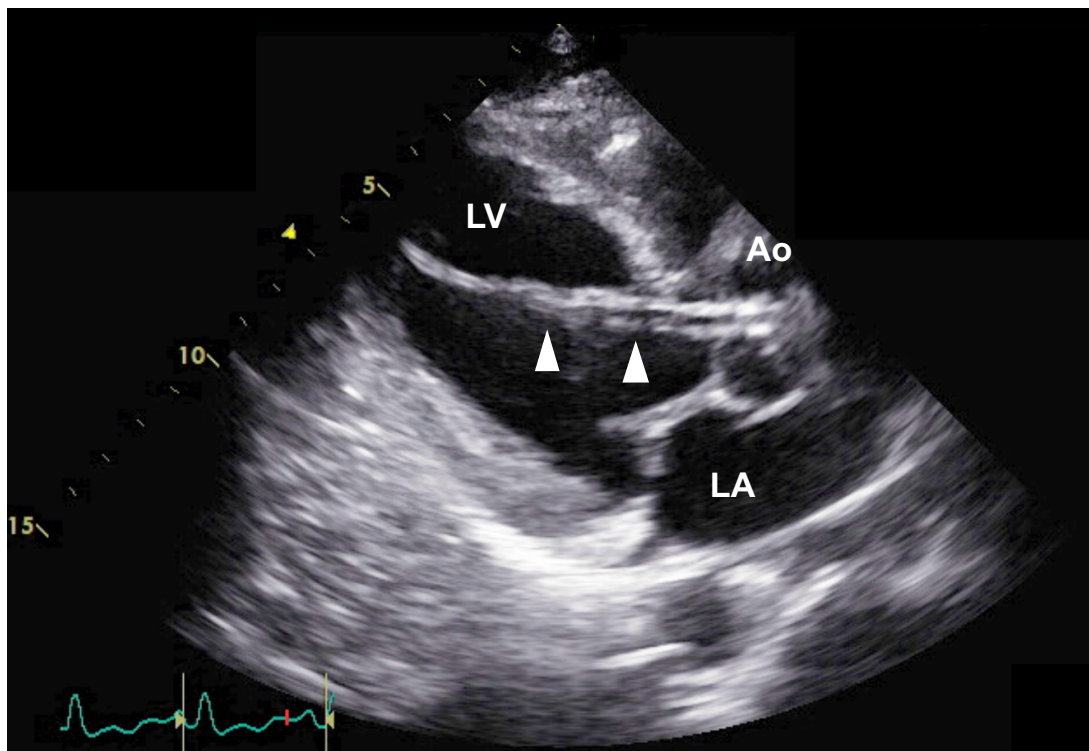


Fig. 2. Parasternal long axis view on echocardiogram showing position of the Impella CP® catheter-based left ventricular assist device (white arrowheads) in the heart. (LV denotes left ventricle, LA = left atrium, Ao = ascending aorta)

team' management strategy is now seen as the new frontier for the treatment of AMI-CS, potentially able to alter the disease trajectory.⁴

The ideal short term MCS device should be easy to implant, maintain, and remove. It must provide adequate haemodynamic support for the failing heart and, at the same time, provide LV decompression. It should also be durable and biocompatible, with low rates of haemolysis or thrombosis. An added bonus would be the ability for patients to undergo continuous rehabilitation or ambulation while on support.⁵

The most commonly employed MCS device has been the IABP. The IABP-SHOCK II trial randomised 600 patients with AMI-CS into IABP implantation and conventional medical therapy. However, there was no difference seen in 30-day mortality between the 2 groups.⁶ Another commonly employed MCS strategy is venoarterial extra-corporeal membrane oxygenation (VA-ECMO).⁷ It provides full haemodynamic support of up to 10L/min of cardiac output, and has the ability to oxygenate the blood in the event of respiratory failure. However, owing to retrograde filling of the aorta from the ECMO inflow cannula, the cardiac afterload

is increased.⁸ There is lack of LV decompression and a risk of pulmonary oedema from the increased LV pressure throughout the cardiac cycle.

The Impella® family of devices are catheter-based, axial-flow ventricular assist devices. The Impella CP® is inserted peripherally, most commonly through the femoral artery, travelling retrogradely up the aorta and across the aortic valve.⁹ The inlet of the cVAD resides in the LV, while the outlet is located in the ascending aorta. It draws blood from the LV, decompressing the chamber and delivering it across the aortic valve to the ascending aorta in a continuous-flow manner. It generates up to 3.5L/min of additional cardiac output, allowing the diseased myocardium to rest and recover.

Despite this cardiac output advantage, the initial study comparing the cVAD with IABP in a cohort of 48 AMI-CS patients did not show a survival benefit at 30-days and 6-months.¹⁰ A more recent study compared cVAD use in 4782 patients who underwent PCI against propensity-matched controls who received IABP.¹¹ These patients were part of a nationwide payment database, with 50% of the cohort having the diagnosis label of CS. In this cohort, cVAD use was, in

fact, associated with increased mortality, bleeding and stroke, compared to IABP.

However, there is still a role for cVAD in patients with AMI-CS, provided they are carefully selected and managed in a protocolised manner. The Detroit cardiogenic shock initiative studied a protocolised management strategy for CS, using the Impella CP® cVAD as the MCS device of choice in 95% of the cases.¹² This protocol mandated the use of invasive haemodynamic measurements to stratify patients' risks of RV failure prior to cVAD implantation. Invasive haemodynamic data were also used in subsequent decision-making regarding therapy escalation or MCS weaning. The result was 75% survival to discharge, with 100% of these patients having sufficient native heart recovery. None required a durable LV assist device or heart transplantation. This initiative has grown to become the National Cardiogenic Shock Initiative (NCSI), adopting the same protocol. In the first 171 patients recruited, survival was 72%, uncannily similar to the original Detroit initiative.¹³

These encouraging results seen in the Detroit Initiative and NCSI were, of course, contingent on the degree of reversible myocardial injury sustained during the AMI episode. On top of best supportive care, protocolised management and active cVAD unloading, the key factor in a good outcome would be the reversibility of myocardial damage sustained during the initial AMI-CS episode. Besides the above single-arm studies, we await the results of the DanGer trial which aims to study the use of cVAD against guideline-driven therapy in 360 patients with AMI-CS.¹⁴ We hope that results of this trial can help identify patients who can derive the most benefits from cVAD therapy, thus refining our selection process.

Protocol adherence aside, another key for successful cVAD use in AMI-CS is adequate RV function. RV output is important to ensure adequate LV preload, which, in turn, ensures adequate cVAD output. Following the initiation of cVAD support, LV decompression will cause the inter-ventricular septum to shift away from the RV, distorting RV anatomy. Simultaneously, there will be increased RV preload, from the extra cVAD flow.¹⁵ In a diseased RV, all these structural and volume shifts can potentiate RV failure, manifesting as persistent hypotension, low cardiac output, pump suction, arrhythmias and haemolysis. To avoid this complication of RV failure following LV support, risk stratification prior to initiation of support must be performed. The NCSI protocol recommends the use of a novel index, the PAPI, calculated using [(pulmonary artery systolic

pressure – pulmonary artery diastolic pressure)/right atrial pressure] to guide decision on the use of cVAD. In the AMI setting, a PAPI value of ≤ 0.9 was previously shown to be useful in predicting poor RV outcomes.¹⁶

In our patient, the ECMO team was put on standby when the patient was being evaluated for cVAD implantation. Should his RV function be deemed to be suboptimal on right heart catheterisation, he would have been put on VA-ECMO. However, as he was found to have uni-ventricular (LV) failure with no major issues with oxygen saturation, the decision was made to use the cVAD strategy, given the relative ease of cVAD implantation and subsequent care, compared to VA-ECMO. In this patient, cVAD support allowed for cardiac decompression and subsequent recovery, while ensuring adequate cardiac output for end-organ perfusion. Together with protocolised care by a multi-disciplinary team, this patient had a good outcome.

Conclusion

Managing AMI-CS using a CS protocol, which includes (1) multi-disciplinary shock team approach, (2) use of invasive haemodynamic assessment and (3) early initiation of MCS, can potentially improve patient survival in a select group of patients with adequate native myocardial recovery. We present the first-in-Singapore use of such a protocol in managing an AMI-CS patient, cumulating in cVAD support for 4.5 days, with good outcome.

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