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Alejandro Lemor
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William O’Neill
Mir B. Basir

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CASE REPORT

Unmasking right ventricular failure in cardiogenic shock: The importance of serial hemodynamics

Alejandro Lemor MD1,2 | Waleed Al-Darzi MBBCh1 | William O’Neill MD1 | Mir Babar Basir DO1

1Department of Cardiology, Henry Ford Hospital, Detroit, Michigan
2Universidad de San Martín de Porres, Facultad de Medicina, Centro de Investigación de Epidemiología Clínica y Medicina Basada en la Evidencia Lima, Peru

Correspondence
Alejandro Lemor, MD, MSc, Henry Ford Hospital, Cardiovascular Department, 2799 W Grand Blvd, Detroit, MI 48202.
Email: alejandrolemor@outlook.com

Abstract
A 65-year-old female was transferred with myocardial infarction, three-vessel coronary artery disease, cardiogenic shock and an intraaortic balloon pump. Given persistent shock, mechanical circulatory support (MCS) was upgraded using a left ventricular hemodynamic support device (Impella CP). The patient was monitored in the catheterization laboratory and serial hemodynamic measures were obtained. Initial hemodynamics showed relative improvement; however, serial assessments demonstrated worsening hemodynamics secondary to right ventricular failure, ultimately requiring a right ventricular hemodynamic support device. The case highlights the rapid changes that can occur with mechanical circulatory support devices and demonstrates the importance of obtaining serial hemodynamics in the cardiac catheterization laboratory.

KEYWORDS
acute myocardial infarction, biventricular failure, mechanical circulatory support

1 | INTRODUCTION

Acute myocardial infarction complicated by cardiogenic shock (AMI-CS) is associated with significant morbidity and mortality. When left ventricular (LV) dysfunction is associated with right ventricular (RV) dysfunction, morbidity and mortality are even higher. Routine assessment of hemodynamics with the use of a pulmonary artery catheter (PAC) remains infrequently performed. PACs allows for early hemodynamic assessment in patients treated with mechanical circulatory support (MCS). In the following case we highlight the importance of serial hemodynamic measures when using MCS and why serial assessments performed in the cardiac catheterization laboratory can be helpful. The case demonstrates the early detection of RV failure after placement placement of a LV MCS device.1

2 | CASE REPORT

A 65-year-old female with a history of hypertension presented with 2 days of worsening chest pain and fatigue to an outside hospital. Electrocardiogram revealed anterior q-waves and ST depression in the inferior leads. Her initial laboratory revealed a mildly elevated troponin, normal serum creatinine, and mild transaminitis. Echocardiogram demonstrated depressed LV ejection fraction approximately 15%, normal RV size, moderately reduced global RV function, and severe mitral regurgitation (Videos S4–S7). The patient was treated as a non-ST elevation myocardial infarction with intravenous heparin infusion. Shortly after transfer to the medical floor, the patient became hypoxic developing flash pulmonary edema and requiring emergent intubation. Emergent coronary angiography was performed due to suspected cardiogenic shock and revealed three-vessel coronary artery disease (Figure 1, Videos S1–S3), for which an intraaortic balloon pump (IABP) was placed. The patient was transferred to our hospital for escalation of care. On arrival, the patient was hypotensive (80/41 mmHg) and tachycardic (104 bpm) with SCAI stage D shock refractory to IABP and inotropes (norepinephrine and dobutamine). Her laboratory results revealed a high-sensitivity troponin of greater than 20,000 ng/L (Normal <19 ng/L), lactic acid of 3.6 mg/dL, oliguria with a rising serum creatinine, and elevated liver function tests concerning for multiorgan failure. The patient was urgently taken to the cardiac...
catheterization laboratory, where a PAC was placed revealing a cardiac index (CI) of 1.94 L/min/m², a cardiac power output (CPO) of 0.52 W and a pulmonary pulsatility index (PAPI) of 0.73. Given the patient’s deteriorating condition the decision was made to upgrade MCS to an Impella CP. Immediate hemodynamic assessment was promising (Table 1). However, as we continued to monitor the patient in the catheterization laboratory and performed serial hemodynamic assessments, hemodynamic worsened with a CPO of 0.4 W and a PAPI of 0.3. We treated the patient for RV failure with a RV MCS device (Impella RP) (Figure 2). The patient was transferred to the cardiac intensive care unit (CICU) where hemodynamics and end organ perfusion continued to improve. Immediate revascularization was delayed due to the patient’s late presentation and to allow for multidisciplinary discussion for consideration of coronary artery bypass grafting and mitral valve repair. Per recommendation of the Heart Team she underwent multivessel percutaneous coronary intervention (PCI) and subsequent weaning of MCS on day 3 of her hospitalization. MCS was removed on day 4 and the patient was slowly weaned from dobutamine 5 mcg/kg/hr over the next few days. Repeat echocardiogram on day 10 revealed an ejection fraction of 40%. Unfortunately, the patient’s hospital course was complicated by ventilator-associated pneumonia and sepsis. She completed her antibiotic course, her kidney function improved, her mental status remained intact and she was discharged on guideline-directed medical therapy to a rehabilitation facility. She was subsequently discharged home and has done well with 12 months of follow up.

3 | DISCUSSION

Cardiogenic shock (CS) affects 5–10% of patients who present with acute myocardial infarction (AMI) and mortality remains greater than 40%. Early identification of CS is crucial for optimizing treatment in an effort to improve mortality in this critically ill patient population. Use of PCI, PAPCs, and early utilization of MCS devices are often required. RV dysfunction occurs in approximately 40% of patients presenting with predominant LV shock and is associated with poorer outcomes when compared with isolated univentricular shock. Clinicians who do not routinely utilize PAC may fail to recognize RV dysfunction or failure; and this may significantly impact patient care. Our

**FIGURE 1** Coronary angiograms showing three-vessel coronary artery disease

**TABLE 1** Trend of hemodynamics

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Prior mechanical circulatory support (MCS)</th>
<th>Immediately post-left sided MCS</th>
<th>45 min post-left sided MCS</th>
<th>24 hr post-biventricular MCS</th>
<th>48 hr post-biventricular MCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>104</td>
<td>149</td>
<td>140</td>
<td>121</td>
<td>92</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>80/41 (61)</td>
<td>115/84 (94)</td>
<td>73/53 (60)</td>
<td>77/ 70 (72)</td>
<td>94/71 (78)</td>
</tr>
<tr>
<td>RA (mmHg)</td>
<td>15</td>
<td>12</td>
<td>15</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>PA (mmHg)</td>
<td>34/26 (30)</td>
<td>24/16 (20)</td>
<td>23/19 (20)</td>
<td>27/19 (22)</td>
<td>25/14 (18)</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>26</td>
<td>16</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>PA sat (%)</td>
<td>55</td>
<td>59</td>
<td>38</td>
<td>62</td>
<td>71</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>3.38</td>
<td>4.18</td>
<td>3.1</td>
<td>3.7</td>
<td>5.7</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>1.94</td>
<td>2.4</td>
<td>1.7</td>
<td>2.1</td>
<td>3.3</td>
</tr>
<tr>
<td>CPO (W)</td>
<td>0.52</td>
<td>0.9</td>
<td>0.4</td>
<td>0.59</td>
<td>0.99</td>
</tr>
<tr>
<td>PAPI</td>
<td>0.73</td>
<td>0.8</td>
<td>0.27</td>
<td>0.8</td>
<td>1.6</td>
</tr>
<tr>
<td>RVSWI</td>
<td>281</td>
<td>161</td>
<td>61</td>
<td>209</td>
<td>395</td>
</tr>
<tr>
<td>Inotropes</td>
<td>Dobutamine 5 mcg/kg/hr and norepinephrine 3–5 mcg/min</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
case highlights the need for PACs and understanding invasive hemodynamic measures.

Invasive hemodynamic are important in understanding the complex changes that occur when utilizing MCS devices. Early identification of RV failure is one such example.6,7 The use of invasive hemodynamic parameters, such as CPO, PAPI, CVP/PCWP, and RVSWI, can help in the early diagnosis and management of RV failure. In our case, knowing the patient had RV dysfunction was a warning to closely monitor RV hemodynamics after placement of LV MCS. It is important to note that the most common cause of RV dysfunction is LV failure. The majority of patients with RV dysfunction, however, will do well with LV unloading, diuresis, inotropes and supportive measures without the need for RV MCS. Hence in our case we elected to start with a strategy of LV support and subsequently assess the need for RV support. However, as we revealed above our patient had significant hemodynamic changes, unmasking RV failure, which ultimately necessitated the need for RV MCS.

As shock management slowly diffuses away from surgeons to advanced heart failure and interventional cardiologists, it is important for our community to understand the importance of PACs and invasive hemodynamics. The tools can guide important decision making and must become routine vocabulary. A CPO less than 0.6 W, for example, suggests significant ventricular dysfunction and poor forward flow, while a PAPI of less than 1.0 is suggestive of RVF in AMI-CS.8 Additional hemodynamic parameters such as CVP/PCWP greater than 0.8, a CVP greater than 12–15 mmHg and a right ventricular stroke work index (RVSWI) less than 300 g m/m² are all important measures interventional cardiologists should be familiar with when caring for shock patients.8 In our patient, all of the above measures confirmed the presence of RVF. If a PAC had not been placed and serial hemodynamic measures had not been obtained, immediate measures such as blood pressure may have reassured many clinicians post-LV-MCS leading them to transfer the patient back to the cardiac ICU where hemodynamic deterioration would have occurred.

Early identification of RV dysfunction plays an important role in AMI-CS management. This is especially true after LV MCS placement, when the strained RV will receive an increased flow that could culminate into RV failure. It is thus critical to have a high suspicion of RV failure and consider serial hemodynamic monitoring. This can understandably be challenging in a busy cardiology center or in the late hours of the night; however, it is important to note that these decisions and steps can greatly alter patient management. Invasive hemodynamic assessment of RV function is the fastest and most reliable way to diagnose RV failure, with echocardiography providing additional benefit.7 It is important to mention that echocardiography alone in the acute setting, may not reflect the acute changes to RV hemodynamics that can occur when using MCS.

Lastly, percutaneous right ventricular assist devices (RVAD) include the Impella RP, Tandem Heart/Protek Duo, and extracorporeal membrane oxygenation. There is no data to support the superiority of a specific RVAD and a multidisciplinary approach emphasizing operator and institutional expertise should be paramount.

4 | CONCLUSION

Patients presenting with cardiogenic shock treated with MCS undergo significant and rapid hemodynamic changes that require the use of pulmonary artery catheters to guide patient management. With greater utilization of MCS devices, it is imperative for clinicians to understand the importance of these measures and their clinical utility. We highlight an example of unmasking right ventricular failure after placement of left ventricular MCS through serial hemodynamic measures in the cardiac catheterization laboratory.

CONFLICT OF INTEREST

Dr. Basir has the following disclosures: Abbott Vascular, Abiomed, Chiesi, Cardiovascular Systems, and Zoll. Dr O’Neill has the following disclosures: Abiomed and Abbott. The rest of the authors have nothing to disclose.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

ORCID

Alejandro Lemor https://orcid.org/0000-0002-4649-8479
Mir Babar Basir https://orcid.org/0000-0003-3486-6753

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SUPPORTING INFORMATION
Additional supporting information may be found online in the Supporting Information section at the end of this article.