Management of Cardiogenic Shock

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Case Presentation

A 60 year-old man with known coronary artery disease complained to coworkers of intermittent chest pain for several days prior to admission. On the morning of admission, he developed crushing chest pain at work and then lost consciousness. Coworkers phoned 911 and performed CPR. Emergency Medical Technicians arrived 15 min later, and reported an initial cardiac rhythm of ventricular fibrillation. He was successfully resuscitated, and a post arrest ECG was then performed (Fig. 10.1). The hospital's ST-elevation myocardial infarction (STEMI) team was activated from the field, and the patient was transported to the Emergency Department. Upon arrival he was hypoxemic and hypotensive. He was intubated prior to emergent coronary angiography. Coronary angiogram revealed a complete occlusion at the site of a prior proximal left anterior descending (LAD) coronary artery stent (Fig. 10.2) as well as a 70 % stenosis of the mid right coronary artery. After much difficulty, a wire was passed through the proximal LAD blockage and the artery was re-stented with restoration of normal flow. The patient remained hypotensive, requiring vasopressor support with norepinephrine to maintain a blood pressure of 80/60 mmHg.

Question What is the next step in management to optimize this patient's hemodynamics and treat his shock?

Answer Initiation of mechanical circulatory support.

For patients in cardiogenic shock (CS) from an acute myocardial infarction (MI) who continue to have hypotension and inadequate cardiac output despite revascularization and pharmacotherapy, it is reasonable to consider the use of mechanical circulatory support. This patient underwent placement of an intra-aortic balloon pump (IABP) and placement of a flow-directed pulmonary artery catheter for invasive hemodynamic monitoring. His initial hemodynamics were notable for an elevated pulmonary capillary wedge pressure (PCWP) of 29 mmHg and a low cardiac index of 1.5 L/min, confirming the diagnosis of CS. With placement of the IABP there was mild improvement in his hemodynamics, and he was admitted to the Cardiac Intensive Care Unit (CICU) for ongoing management. A transthoracic echocardiogram was obtained, which demonstrated a severely reduced ejection fraction of 15 % with anterior and anteroseptal wall akinesis and global hypokinesis of the remaining wall segments. Over the next 12–18 h, despite the IABP and increasing

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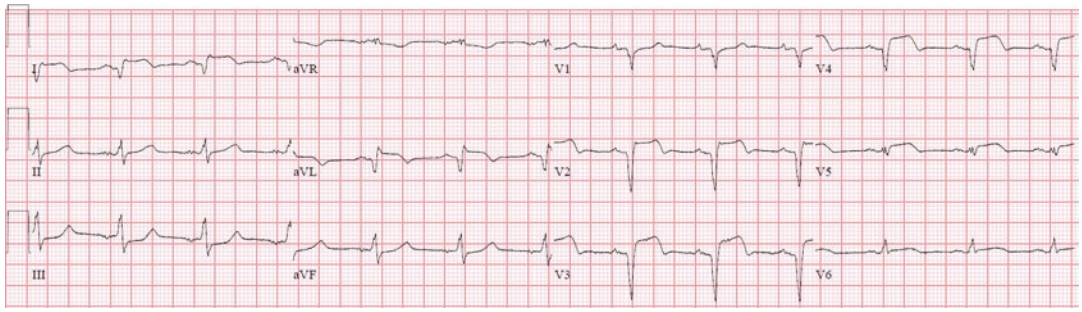

Fig. 10.1 Admission ECG

doses of pharmacologic support with vasopressors/inotropes, his hemodynamics remained marginal with worsening renal failure. In the setting of progressive shock, the patient was brought back to the catheterization lab to upgrade his mechanical circulatory support to a TandemHeart® percutaneous left ventricular assist device (LVAD). Although his filling pressures and cardiac output improved with the increased mechanical circulatory support, his overall clinical picture continued to deteriorate, and his family ultimately decided to transition his goals of care to comfort measures only. His mechanical and pharmacologic supports were withdrawn and he expired.

Principles of Management

Diagnosis

Cardiogenic shock (CS) occurs in roughly 8% of individuals who present with STEMI, while 80% of CS cases are due to an acute MI [1, 2]. The diagnosis of CS can be made based on the following established clinical criteria: (1) Hypotension – systolic blood pressure < 90 mmHg for more than 30 min or the need for vasopressor/mechanical support to achieve this blood pressure; (2) pulmonary edema or evidence of elevated left ventricular filling pressures; (3) evidence of end-organ hypoperfusion with at least one of the following: altered mental status, cold clammy skin or extremities, urine output less than 30 ml/h, or elevated serum lactate greater than 2 mmol/L [2–4]. The hemodynamic criteria for CS include a cardiac index (CI) of less than 2.2 L/min/m^2 as well as a PCWP greater than 18 mmHg [3]. Invasive hemodynamics with a pulmonary arterial catheter have been recommended and are often used to help confirm the diagnosis of CS and help guide management [3, 5, 6].

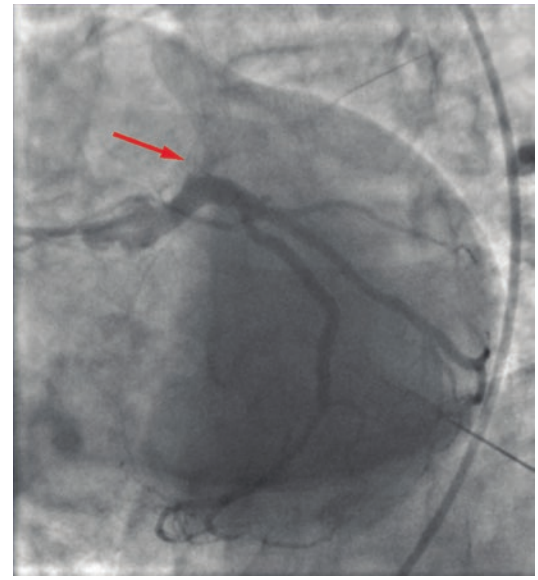

Fig. 10.2 Coronary angiogram, LAO Caudal view, demonstrating stent thrombosis of LAD (*arrow*)

Early Revascularization

The most significant advance in treatment of CS has been the implementation of early revascularization of the infarct-related artery, which has led to a significant decrease in mortality [4]. The Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK)

trial randomized 302 patients with CS from an acute MI to undergo either emergency revascularization (152 patients) or initial medical stabilization (150 patients) [7]. Patients underwent revascularization with either percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). Although the primary endpoint of 30 day all-cause mortality was not significantly different between the revascularization and the medical-therapy groups (46.7 % and 56.0 % respectively, p = 0.11), there was a significant difference between the respective groups at 6 months favoring the revascularization group (50.3 % versus 63.1 %, p = 0.027). The 13 % absolute risk reduction persisted at longer term follow up of 1 and 6 years [7, 8]. As a result of this trial, current guidelines give early revascularization with either PCI or CABG a class Ib recommendation [3, 5, 9].

Vasopressors/Inotropes

Vasopressors and inotropes are often required to treat patients with CS [2]. Dopamine and Norepinephrine are commonly used vasopressors that were compared in a randomized control trial including 1,679 patients with shock, of whom 280 were classified as having CS. The overall trial demonstrated an increased burden of arrhythmic events in the dopamine treated group compared with the norepinephrine treated group, although there was no difference in the primary endpoint of all-cause mortality. However, in the predefined subgroup of 280 patients with CS, norepinephrine was associated with a significantly lower death rate compared to dopamine [10]. As a result, the European Society of Cardiology (ESC) guidelines recommend norepinephrine over dopamine for medical management of hypotension from CS, and the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines caution that there may be, "excess hazard" associated with the use of dopamine in CS [3, 5].

Inotropic support with dobutamine increases heart rate, stroke volume, and cardiac output, while decreasing left ventricular filling pressures and systemic vascular resistance [11]. Since dobutamine can decrease blood pressure, it is often used in conjunction with vasopressors to improve cardiac output in the setting of CS from acute MI [2, 4]. Milrinone also increases heart rate, stroke volume, and cardiac output while decreasing left ventricular filling pressures and systemic vascular resistance. However, because milrinone can cause more significant vasodilation and hypotension, it is not a preferred inotropic agent in CS from acute MI [4]. Although inotropes and vasopressors can improve cardiac output and blood pressure, they also increase myocardial oxygen demand, increase risk for arrhythmias, and can impair microcirculation; therefore their use should be limited to the lowest dose for the shortest duration possible [2, 4, 11].

The goal of inotropic/vasopressor support is to maintain end-organ perfusion. In general, a target of a mean arterial pressure >=65 mmHg is reasonable. However, evidence of organ function (mental status, renal function, absence of biochemical evidence of organ ischemia) is more important.

Percutaneous Mechanical Circulatory Support

Given the limitations of pharmacologic support with vasopressors and inotropes to maintain adequate blood pressure and tissue perfusion, there has been much interest in the use of percutaneous mechanical circulatory support (MCS). There are now multiple device options, and over the past several years there has been a significant increase in the use of percutaneous MCS [12]. Currently available devices include the intra-aortic balloon pump (IABP), the Impella® micro-axial rotary pumps (2.5, CP, and 5.0), the TandemHeart® continuous flow centrifugal pump, and percutaneous venoarterial extracorporeal membrane oxygenation (v-a ECMO). These devices all require anticoagulation and have been associated with adverse events including limb ischemia, stroke, infection, and hemolysis. The ACC/AHA guidelines give a class IIa recommendation for

Table 10.1 Comparison of device characteristics and hemodynamics

	IABP	Impella® 2.5	Impella® CP	Impella® 5.0	TandemHeart™	ECMO
Hemodynamic support (L/min)	0.5–1.0	2.5	3.7–4.0	5.0	4.0	4.0–7.0
Pump mechanism	Pneumatic	Axial flow	Axial flow	Axial flow	Centrifugal	Centrifugal
Effect on LV pre-load	Reduced	Reduced	Reduced	Reduced	Reduced	Reduced
Effect on LV afterload	Reduced	Neutral	Neutral	Neutral	Increased	Increased

IABP intra-aortic balloon pump, *ECMO* extracorporeal membrane oxygenation, *LV* left ventricle

the use of IABP in patients with CS after STEMI, whereas the ACC/AHA and the ESC give a IIb recommendation for the use of alternative percutaneous left ventricular assist devices (LVADs) in patients with CS [3, 5, 9]. The different devices are discussed here and in Table 10.1.

Intra-Aortic Balloon Pump

The IABP is placed in the descending thoracic aorta via femoral arterial access. The pneumatic device inflates during diastole, raising diastolic blood pressure, and deflates during systole, lowering left ventricular afterload. Figure 10.3 demonstrates the typical IABP waveforms. The IABP can increase stroke volume and cardiac output up to 0.5–1.0 L/min [4]. The IABP is widely available and is the most commonly used mechanical support device, although it provides limited hemodynamic support [2].

Impella® 2.5, CP, and 5.0

The axial flow device is typically placed via femoral arterial access retrograde across the aortic valve and provides support by aspirating blood from the left ventricle and pumping it into the ascending aorta (Fig. 10.4). The 2.5 and CP can provide up to 2.5 L/min and 3.7–4.0 L/min of support, respectively, and both can be placed percutaneously. The 5.0 can provide up to 5.0 L/min of support, but requires a surgical cut down of either the femoral or axillary artery [2, 4].

TandemHeart®

This continuous flow centrifugal device is placed percutaneously and can deliver up to 4.0 L/min of circulatory support. The inflow cannula is placed in the left atrium via femoral venous access and transseptal puncture (Fig. 10.4). The outflow cannula is placed in the lower abdominal aorta or the iliac artery via femoral arterial access. Oxygenated blood is aspirated from the left atrium into the inflow cannula, and is then pumped into the lower abdominal aorta or the iliac artery via the outflow cannula. The TandemHeart® increases afterload because blood is pumped retrograde towards the left ventricle [2, 4].

Venoarterial Extracorporeal Membrane Oxygenation (v-a ECMO)

The percutaneous v-a ECMO system includes a centrifugal pump, heat exchanger, and oxygenator. The inflow cannula is placed in the right atrium via femoral venous access, and the outflow cannula is placed in the descending thoracic aorta via femoral arterial access. V-a ECMO can provide up to 4.0–7.0 L/min of biventricular circulatory support (bypasses both the right and left ventricle) as well as respiratory support. Limitations are that it does not directly unload the left ventricle, it increases afterload, and requires additional staffing [2, 4].

Temporary Surgical Mechanical Circulatory Support

When percutaneous MCS is inadequate, temporary surgical MCS with a surgically placed VAD can provide support for both the right and left ventricle with increased flow (up to 10 L/min) [13]. These surgically placed VADs can also be

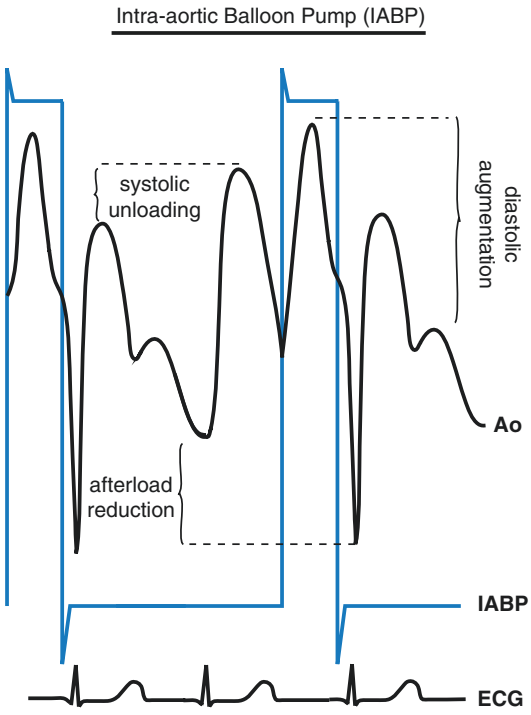

Fig. 10.3 Intra-aortic balloon pump (IABP) pressure waveforms, with IABP inflation set to 2:1. The IABP is inflated in early diastole at the timing of the dichrotic notch, augmenting blood pressure during diastole, thus augmenting coronary perfusion pressure. The balloon deflates in late diastole, lowering aortic end diastolic pressure, decreasing afterload on the left ventricle, and unloading the left ventricle during systole

left in place for weeks to months if necessary, providing a longer term temporary solution while waiting for recovery or as a bridge to transplant. The CentriMag® VAD (magnetically levitated rotor) and the Abiomed AB5000™ (pneumatically driven external ventricle) are two examples of temporary surgical MCS.

There are three outcomes of Mechanical Circulatory Support: (1) Recovery, i.e. improvement in hemodynamics such that MCS can be removed. (2) Implantation of a durable LVAD, or in rare circumstances, heart transplantation. (3) Progressive multisystem organ dysfunction and death. There are little data regarding the optimal patient selection and timing of percutaneous MCS, and we recommend that each patient be evaluated on an individualized basis with input from a multidimensional team.

Evidence Contour

There are several aspects in the management of patients with CS from an acute MI for which clinical equipoise still exists.

Multivessel Revascularization

Although the SHOCK trial demonstrated the benefit of early culprit vessel revascularization in CS from STEMI, the optimal revascularization strategy among individuals with multivessel disease remains unclear. A critical eye toward these data will note that the primary endpoint was not met and thus all additional analyses in the SHOCK trial were inherently exploratory. Nonetheless, since no other strategies have proven effective in CS, early revascularization remains the paradigm. Nearly ¾ of individuals who present with CS from acute MI have multivessel coronary disease [14]. Current ESC guidelines recommend multivessel PCI (class IIa) for individuals with CS who have multivessel coronary artery disease [9]. The ACC/AHA guidelines do not give an overt recommendation, but do recognize shock or severe heart failure as a clinical scenario in which acute revascularization of significant stenosis in noninfarct arteries can be justified [5]. The current evidence as it relates to patients with CS comes from nonrandomized studies looking at outcomes associated with multivessel PCI in CS from acute MI. Two of these trials demonstrated significant harm associated with multivessel PCI, one trial demonstrated significant benefit, and one demonstrated no change in mortality [14–17]. There is an ongoing prospective randomized control trial in Europe, the CULPRIT-SHOCK trial (Clinicaltrials.gov: NCT01927549), which seeks to answer this question.

Mechanical Circulatory Support

Although the use of percutaneous mechanical circulatory support is increasing, there are limited data supporting this practice. The Intra-aortic Balloon Pump in Cardiogenic Shock II (IABP-SHOCK II)

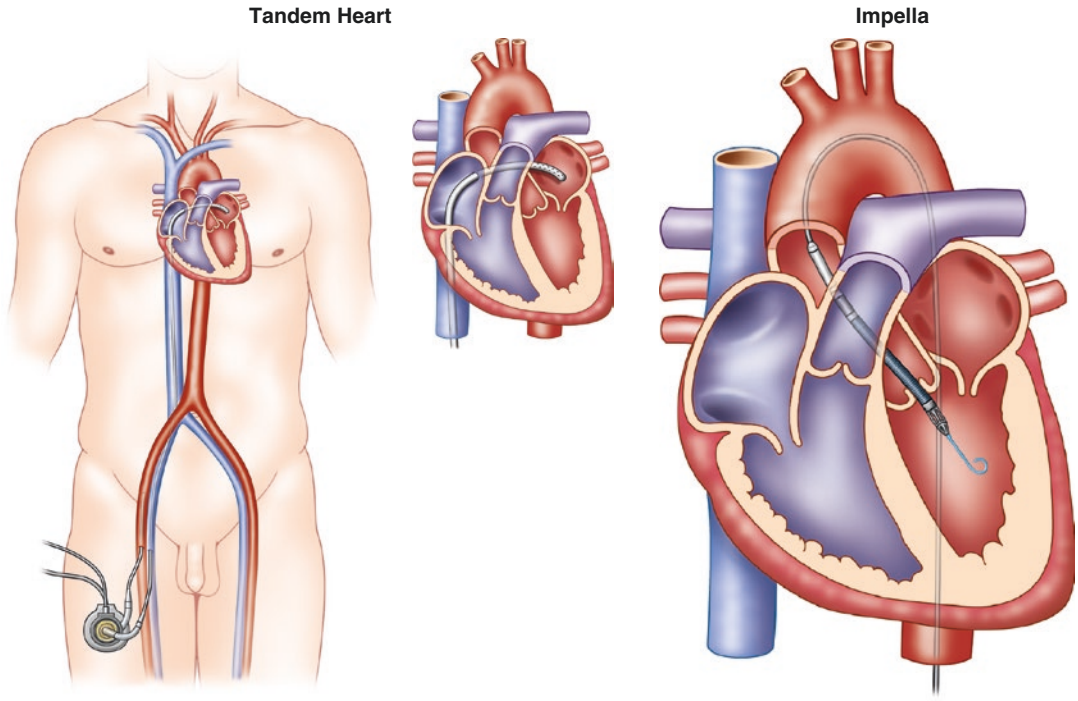

Fig. 10.4 Images of percutaneous LVADs: Tandem Heart® and Impella®

Trial randomized 600 patients with CS from acute MI to IABP versus no IABP. All participants were supposed to undergo early revascularization and receive guideline driven medical therapy. At 30 days there was no difference in the primary endpoint of all-cause mortality, and these results persisted at 12 month follow up [18, 19]. As a result, the ESC has recently changed their recommendation for IABP in CS from acute MI to a class III indication [9].

A meta-analysis was published in 2009 comparing the effect of percutaneous left ventricular assist devices (LVADs) versus IABP on hemodynamics and 30-day mortality [20]. The meta-analysis included two trials comparing the TandemHeart® to IABP and one comparing the Impella® to IABP. There was a significant improvement in hemodynamics (higher cardiac index, higher MAP, and lower PCWP) with percutaneous LVADs compared to IABP. However, there was no difference in 30 day mortality, and there was a significantly increased risk of bleeding in patients with percutaneous LVADs versus IABP. Importantly, the Impella® device used in the randomized control trial was the 2.5. It remains unclear if the higher flow Impella® devices (CP and 5.0) would be associated with lower mortality when compared to IABP. There are no randomized control trials that have evaluated the use of v-a ECMO in CS despite its widespread use for this indication.

Despite the lack of evidence, both the ACC/AHA and the ESC recommend consideration of mechanical circulatory support for patients with CS from acute MI [3, 5]. Given the high mortality rate associated with CS in acute MI, it is likely that the use of percutaneous mechanical circulatory support will continue notwithstanding the lack of hard outcome data. Additionally, these devices improve hemodynamic parameters and provide a critical bridge to further clinical decision making, including considerations of durable VADs, cardiac transplant evaluations, and goals of care discussions

with the patient's family and proxies. Future clinical trials are needed to better define patient selection, choice of mechanical support, and optimal timing for device placement.

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