Circulatory Support in the Management of Cardiogenic Shock

Cardiogenic shock is a life-threatening medical condition resulting from an inadequate circulation of blood due to primary failure of the ventricles of the heart to function effectively. As a result of the failure of the heart to pump enough nutrients to the body, blood pressure falls and organs may begin to fail.

Although there are many causes for cardiogenic shock, the most common cause is due to damage to the heart muscle, as a result of a large myocardial infarction. Despite advances in the management of myocardial infarction, cardiogenic shock complicates approximately 5 to 8 percent of ST elevation MI (STEMI) and 2.5 percent of non-STEMI cases. This translates to 40,000-50,000 cases per year in the United States. The mortality from cardiogenic shock continues to remain very high at 40-50 percent.

Diagnosis
The diagnosis of cardiogenic shock involves observing the following in the setting of an acute myocardial infarction.

Clinical criteria:
- SBP <90mm Hg for at least 30 minutes
- Supportive measures needed to maintain SBP >90mm Hg
- End-organ hypoperfusion
- Cool extremities
- Urine output <30ml/h
- Heart rate >60 beats/minute

Hemodynamic criteria:
- Cardiac index <2.2ml/min/m2 and PCWP >15mm Hg

Increasingly, it has been realized that cardiogenic shock is not a single entity but a spectrum ranging from pre-shock/mild shock, to severe shock and refractory shock. Mortality rises linearly with increasing severity of shock (Fig. 3).

Fig. 1 — Pathogenesis of Cardiogenic Shock

Continued on page 2

Management of Cardiogenic Shock

General measures: Along with general measures for acute myocardial infarction such as antiplatelet and antithrombotic therapy, arterial oxygenation and near-normal pH should be maintained to minimize ischemia. Mechanical ventilation should be instituted early, as positive end-expiratory pressure decreases preload and afterload. Mechanical ventilation also reduces work of breathing.

Pharmacological support includes inotropic and vasopressor agents, which should be used in the lowest possible doses. Unfortunately, these agents increase myocardial ATP consumption such that short-term hemodynamic improvement occurs at the cost of increased oxygen demand when the heart is failing and supply is already limited. Higher vasopressor doses are associated with poorer survival, and this is likely due to more severe underlying hemodynamic derangement and direct toxic effects. Still, use of inotropic and vasopressor agents are required to maintain coronary and systemic perfusion until circulatory support device is placed.

Immediate echocardiogram is indicated to assess the ventricular function and also to rule out mechanical complications such as acute mitral regurgitation, ventricular septal rupture or free wall rupture.

Rapid assessment of hemodynamics using pulmonary artery (Swan-Ganz) catheterization is frequently required to confirm the diagnosis of cardiogenic shock and to guide changes in therapy. Beyond just assessing pulmonary capillary wedge pressure, additional hemodynamic data, particularly cardiac power and pulmonary artery pulsatility index, add powerful diagnostic and prognostic value and are increasingly used in management algorithms (Fig. 3). Cardiac power (product of mean arterial blood pressure and cardiac output divided by 451) of <0.6 W represents an independent predictor of mortality in the setting of cardiogenic shock and has been used to guide extent of mechanical support.

Mechanical support: The main mechanical support strategies commonly utilized in the management of cardiogenic shock are intra-aortic balloon pump (IABP), Impella device, Tandem Heart device and venoarterial extracorporeal membrane oxygenation (VA-ECMO) (Fig. 2).

Although IABP has long been the mainstay of mechanical therapy, it provides minimal hemodynamic support, which may be insufficient to support more severe forms of cardiogenic shock. Further, a recent large randomized clinical trial demonstrated no benefit from routine use of IABP in patients with cardiogenic shock and as such cannot be recommended for routine use in cardiogenic shock.

Continued on page 3
Newer continuous flow devices such as Impella (left ventricle to aorta) and Tandem Heart (left atrium to femoral artery) offer a greater level of left ventricular support. VA-ECMO, femoral vein to femoral artery cannulation with extracorporeal oxygenation, has been used predominantly to provide circulatory support in severe cardiogenic shock coupled with respiratory failure.

While randomized clinical trials have been too small to demonstrate a mortality benefit with Impella compared to IABP in patients with cardiogenic shock, these studies have demonstrated superior hemodynamic support and maintenance of cardiac power. The FDA has recently approved Impella devices for use in cardiogenic shock after acute myocardial infarction or open-heart surgery. Limited randomized clinical trials for Tandem Heart also demonstrate superior hemodynamic support compared to IABP in the setting of cardiogenic shock. Finally, while VA-ECMO provides excellent circulatory volume support, left ventricular venting is often required to prevent increased myocardial oxygen demand secondary to increased afterload, which can precipitate further myocardial ischemia. Hence, its use is best reserved for patients with severe cardiogenic shock coupled with respiratory failure and severe persistent hypoxemia.

Revascularization: The survival benefit of early revascularization in cardiogenic shock compared to initial medical stabilization was demonstrated convincingly in the randomized SHOCK trial, which found a 13 percent absolute increase in one-year survival in patients assigned to early revascularization. This corresponded to a number needed to treat <8 patients to save one life. Following that, early revascularization, either percutaneous or surgical, became the standard of care in patients with cardiogenic shock.

As in MI without shock, earlier revascularization is better in cardiogenic shock. Presentation zero to six hours after symptom onset was associated with the lowest mortality among shock patients undergoing primary percutaneous coronary intervention (PCI). In addition, earlier institution of mechanical support with Impella device prior to revascularization has been shown to improve outcomes compared to subsequent implantation after PCI, giving rise to the concept of door to left ventricular unloading time in cardiogenic shock.

Conclusions
Cardiogenic shock continues to remain a life-threatening complication of acute myocardial infarction and is associated with very high mortality. BayCare hospitals’ use of novel hemodynamic markers such as cardiac power output, helps to identify the full spectrum of cardiogenic shock early. All BayCare hospitals have protocols for rapid hemodynamic stabilization using mechanical support – all the BayCare hospitals have IABP, most have Impella and major cardiac centers have V-A ECMO. BayCare cardiovascular service line is establishing a multidisciplinary “Shock Team” at each BayCare hospital to streamline and standardize care for the benefit of our patients. For those patients who need even further LV support, St. Joseph’s Hospital will begin the first left ventricular assist device program in BayCare this fall.