Evaluation and Management of ST-elevation Myocardial Infarction and Shock

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Abstract

Cardiogenic shock is the deadliest complication of acute ST-elevation myocardial infarction. Prompt recognition and intervention are critical for patient survival. The diagnosis of cardiogenic shock is primarily a clinical one based on signs and symptoms of low cardiac output and heart failure, and can be confirmed with placement of a pulmonary arterial catheter. Vasopressor and inotropic therapies are typically required, and in severe cases, an intra-aortic balloon pump can provide additional haemodynamic support. Although mortality for cardiogenic shock associated with ST-elevation myocardial infarction remains high, early reperfusion strategies primarily via percutaneous coronary intervention or coronary artery bypass graft surgery have led to improved outcomes.

Keywords

Cardiogenic shock, STEMI, intra-aortic balloon pump, PCI, CABG, rescue PCI

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Cardiogenic shock is the leading cause of death in patients with acute ST-elevation myocardial infarction (STEMI). Characterised by a state of low cardiac output leading to end-organ hypoperfusion, cardiogenic shock complicates approximately 5–8 % of STEMIs and is associated with a mortality rate approaching 50 percent.[1–3] Prompt recognition and therapeutic intervention for cardiogenic shock due to STEMI are critical for patient survival.

Aetiology

Ischaemic dysfunction of cardiac myocytes during STEMI can impair systolic and diastolic function of the right, left or both ventricles. When ischaemic injury is extensive, ventricular function can be impaired to such a degree that cardiogenic shock occurs, whereby cardiac output falls and elevated ventricular filling pressures lead to heart failure.[4] The decreased cardiac output then propagates a vicious cycle of progressively worsening coronary perfusion, myocyte dysfunction, and ultimately end-organ hypoperfusion.[5] This review focuses on ventricular dysfunction as the cause of shock, but cardiogenic shock can also occur from mechanical complications of STEMI such as papillary muscle, ventricular septum or free wall rupture.

Left ventricular dysfunction is implicated in the majority of cases of cardiogenic shock associated with STEMI.[6] Often, the infarction involves the anterior territory of the left ventricle. Predominant right ventricular dysfunction comprises approximately 5 % of cases and is usually due to an inferior MI with proximal occlusion of the right coronary artery.[7] In addition to the acute infarction, patients in cardiogenic shock often have suffered prior infarcts and tend to have severe three-vessel coronary disease, all of which leaves them prone to extensive ischaemic injury and subsequent ventricular dysfunction.[4,7]

Presentation and Diagnosis

Patients suffering from STEMI and cardiogenic shock present with signs and symptoms of hypoperfusion and heart failure. The diagnosis is made clinically and can be confirmed with placement of a pulmonary arterial catheter (PAC).

Common symptoms include chest pain and dyspnoea. On physical examination, patients are hypotensive and may show evidence of systemic hypoperfusion such as altered mental status or poor urine output. Laboratory studies may show an elevated lactate level and a rising creatinine, which may be due to both decreased renal perfusion and venous congestion. Classically, cardiogenic shock has been associated with cool extremities due to low cardiac output and compensatory systemic vasoconstriction. In practice, however, systemic vascular resistance is often not elevated and may even be low. This may be due to concomitant septic shock, particularly as the hypoperfusion from cardiogenic shock places patients at high risk for ischaemic bowel and subsequent translocation of gut microbes. In addition, myocardial infarction alone can lead to a systemic inflammatory response.[8] Most STEMI patients who develop cardiogenic shock do so not on arrival to the hospital, but within the first 24 hours of admission.[9]

Patients may also demonstrate signs of volume overload. Decreased oxygen saturation and rales due to pulmonary oedema may be present in patients with predominantly left ventricular dysfunction and may necessitate intubation, but up to one-third of such patients present without pulmonary congestion and chest X-ray may be clear.[10] Patients with mainly right-sided involvement tend to have clear lungs but may have distended neck veins and peripheral oedema or ascites.

Risk factors for cardiogenic shock associated with STEMI include older age, diabetes mellitus, ongoing angina, heart failure, low systolic blood pressure, tachycardia and left bundle branch block.[11,12] Of these risk factors, age appears to be most predictive. An increase in age by 10 years has been shown to be associated with a nearly 50 % higher probability of developing cardiogenic shock.[12]

Echocardiography should be performed to assess ventricular function and to exclude mechanical complications of STEMI. Echocardiography findings in cardiogenic shock include severe impairment of ventricular function, which may be predominantly right or left-sided or both and may be either systolic or diastolic or both. While a low left ventricular ejection fraction (LVEF) is a poor prognostic indicator, it does not completely correlate with the presence of cardiogenic shock. Often the LVEF is only moderately depressed and occasionally can be preserved.[13–16]

Placement of a PAC can both provide a definitive diagnosis of cardiogenic shock and guide resuscitative efforts. PAC haemodynamic measurements confirm the diagnosis of cardiogenic shock by showing a low cardiac index (<2.2 L/min/m^2) and elevated ventricular filling pressures. Mixed venous oxygen saturation is typically low, reflecting the decreased cardiac output and increased oxygen extraction from peripheral tissues. In addition, PACs can distinguish between left ventricular or right ventricular dysfunction as the primary cause of shock by demonstrating a corresponding pulmonary capillary wedge pressure (PCWP) greater than 15–18 or right ventricular end diastolic pressure (RVEDP) greater than 10–15 mmHg, respectively.[10] From the haemodynamic parameters provided by a PAC, systemic vascular resistance can be calculated to determine the potential co-existence of other types of shock. While PACs have not been proven to confer mortality benefit or lead to shorter hospitalisations, their utility specific to cardiogenic shock has not been evaluated.[17–19] In practice, they provide haemodynamic measurements that can prove invaluable in both diagnosis and management of cardiogenic shock.[20]

Therapy

The management for cardiogenic shock due to STEMI centres on early revascularisation and the provision of pharmacological and mechanical haemodynamic support. Re-establishing coronary perfusion can reverse the ischaemic injury to cardiac myocytes and potentially recover ventricular function. Therefore, revascularisation via percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) should be attempted on an urgent basis. Early presentation and early intervention are associated with significantly lower mortality rates at six months and 12 months as seen in the SHOCK trial.[14,21] It is the widespread adoption of early revascularisation that has led to the steady improvement in survival for cardiogenic shock over the last two decades, which previously was almost always fatal.[1,21] Revascularisation should be attempted even for delayed presentations of cardiogenic shock. Survival benefit has been demonstrated for PCI performed within 48 hours of STEMI onset and 18 hours after onset of shock.[22]

In settings where PCI and CABG are unavailable, fibrinolysis should be performed, although the mortality benefit conferred is modest and inferior to PCI or CABG.[23,24] If fibrinolysis does not achieve reperfusion, often considered "primary failure" of thrombolytic therapy, then rescue PCI should be undertaken as soon as possible, ideally within 12 hours of initial chest pain.[25–27] Among patients for whom fibrinolysis initially appears successful, a substantial proportion will develop recurrent ischaemia caused by threatened reocclusion of their coronary artery.[28] These patients, similarly to those who undergo rescue PCI, also derive survival benefit from early revascularisation with PCI.[29,30]

A comparison of outcomes between PCI and CABG in the setting of cardiogenic shock found no difference in survival at 30 days and one year. Notably, the patients in the CABG group were more likely to have three-vessel disease or left main disease.[31] Overall for patients in cardiogenic shock due to STEMI, PCI is performed much more commonly than CABG, particularly if a culprit lesion is found. Rarely, in cases involving multi-vessel or left main disease, PCI may be necessary to stabilise patients prior to CABG.

In addition to revascularisation, patients should be started on antithrombotic therapy including aspirin, heparin, P2Y12 inhibitors, and potentially glycoprotein IIb/IIIa inhibitors whereas beta blockers and other drugs with negative inotropic effects should be withheld. Although aspirin and heparin have not been specifically studied in cardiogenic shock, they should be continued given their established benefit in acute coronary syndromes and lack of obvious contraindication.[32,33] Antiplatelet therapy with P2Y12 inhibitors like clopidogrel, ticagrelor, or prasugrel are also indicated.[34–36] Glycoprotein IIb/IIIa inhibitors have been shown to independently reduce the 30-day mortality rate for patients in cardiogenic shock and should be considered particularly when P2Y12 inhibitors are not used.[37] In combination with antithrombotic therapy, the risk of major periprocedural bleeding is nontrivial and should be weighed carefully. For PCI, the risk has been reported as high as 5 %, although the increasing use of radial access has lowered the rate of vascular and bleeding complications.[38–42] Beta blockers and calcium channel blockers should be avoided given their negative inotropic effects and potential to prolong or worsen cardiogenic shock.[43]

While revascularisation is the definitive therapy for cardiogenic shock in STEMI, inotropic and vasopressor support is critical to maintain perfusion of vital organs. The ideal choice of first-line agent is not well-established, likely reflecting the complex physiology of cardiogenic shock. PACs are useful for tailoring vasopressor and inotropic therapy given the often uncertain and dynamic haemodynamic parameters in these settings. For patients who are severely hypotensive, norepinephrine is a reasonable first choice that exerts both inotropic and vasopressor activity. Dopamine at doses >10 mcg/kg/min is similar in effect, although it is associated with more arrhythmias and possibly higher mortality compared to norepinephrine.[44] However, the increased systemic vascular resistance brought on by both norepinephrine and dopamine raises afterload so that the heart must work harder to maintain cardiac output. For patients whose hypotension is less severe, inotropes such as dobutamine and milrinone should be considered. The vasodilation caused by both of these inotropes may be prohibitive in the severely hypotensive patient. In patients with milder cardiogenic shock, however, the vasodilatory effect and reduction in afterload may improve cardiac output such that on balance blood pressure may not actually decrease. The minimum required dose should be used for all of these agents as escalating doses increase the chance of arrhythmia and impose increased demand on an already struggling heart.

In addition to determining the optimal choice of inotropic or vasopressor support, a PAC can also guide fluid management.[20] The

overall goal of fluid management is to maximise cardiac output and minimise ventricular filling pressures. The optimal filling pressures are often a moving target and must be individualised to each patient in cardiogenic shock. Some patients in cardiogenic shock may be hypovolaemic with relatively low PCWP shown on PAC and these patients may benefit from a small volume challenge. Other patients present with pulmonary oedema with evidence of high filling pressures on PAC and require intravenous diuresis. Typically the goal PCWP ranges from 18–25 mmHg.[10]

Placement of an intra-aortic balloon pump (IABP) should also be considered to provide mechanical support of cardiac function, particularly for severe cardiogenic shock. The balloon pump is inserted into the proximal aorta and inflates during diastole to augment coronary perfusion and deflates in systole to reduce afterload by a vacuum effect. In the IABP SHOCK II trial, patients in cardiogenic shock from acute MI were randomised to either undergo IABP placement or not. There was no mortality difference between the two groups both at 30 days and at one year.[45,46] Nonetheless, the study authors acknowledge that their patient population represented primarily mild to moderately severe cardiogenic shock. IABP may provide mortality benefit for patients in rapidly decompensating and severe cardiogenic shock.

Early experience with other forms of mechanical support such as the percutaneous or surgically implanted left ventricular assist device (LVAD) has been relatively inconclusive thus far. The ventricular assist devices appear to result in more rapid haemodynamic improvement than IABPs but have not been shown to provide additional mortality benefit and yet are more invasive and associated with more complications.[47,48]

Patients with predominantly right ventricular dysfunction also derive benefit from early revascularisation and haemodynamic support but there are distinctive aspects to their management. These patients are highly preload dependent and often require large volume fluid resuscitation.[49,50] Fluids should be administered until the jugular venous pressure is greater than 15 mmHg and there is no longer improvement in blood pressure.[51] Nitrates and diuretics should generally be avoided as they both reduce preload of the right ventricle. If inotropic support is needed, dopamine and dobutamine are first-line agents.[52] Both agents also provide chronotropic support that is often necessary given the high frequency of bradycardia associated with right ventricular STEMIs. As low heart rates in this setting may substantially impair cardiac output, atropine or placement of a temporary pacemaker may also be required.[53,54]

Conclusion

Although cardiogenic shock remains a deadly complication of STEMI, early diagnosis and intervention have led to improved outcomes. The diagnosis of cardiogenic shock is primarily based on clinical signs and symptoms of low cardiac output and heart failure and confirmed with placement of a PAC. Vasopressor and inotropic therapies are typically required and in severe cases, an IABP can provide additional haemodynamic support. Ultimately, early revascularisation performed via PCI or CABG offers patients the greatest chance for survival. ■

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