Ischaemic cardiogenic shock

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Abstract

Ischaemia is the most common underlying cause of cardiogenic shock. Cardiogenic shock occurs in up to 10% of patients presenting with acute myocardial infarction and is the leading cause of death. Myocardial ischaemia results in both systolic and diastolic dysfunction and triggers a maladaptive feedback loop that can ultimately result in tissue hypoxia, multi-organ dysfunction and death. Myocardial dysfunction can be complicated by a systemic inflammatory response syndrome (SIRS) as a result of systemic hypoxia. Echocardiography is key to diagnosis and to exclude conditions requiring urgent surgical intervention. Serial assessment can be used to monitor response to interventions and/or complications. Resuscitative aims are immediate cardiorespiratory stabilization to facilitate urgent revascularization. Both pharmacological and mechanical supportive techniques are used. Mortality rates for patients who develop ischaemic cardiogenic shock remain high, and further research into strategies to prevent and treat the condition is required.

Keywords Acute myocardial infarction/ischaemia; cardiogenic shock; myocardial revascularization

Royal College of Anaesthetists CPD matrix: 1B04, 2C01, 2C03, 2C04, 3C00

Learning objectives

After reading this article you should be able to:

- define cardiogenic shock according to both clinical and haemodynamic parameters
- explain the pathophysiological processes that can cause or contribute to cardiogenic shock in the setting of myocardial ischaemia
- explain the processes of acute resuscitation of patients in ischaemic cardiogenic shock as they relate to pharmacotherapy, mechanical support and revascularization
- recognize the need for urgent revascularization in the setting of myocardial ischaemia

Definition and incidence

Shock is a clinical state in which there is an imbalance between cellular oxygen supply and demand resulting in tissue hypoxia. Cardiogenic shock (CS) is shock occurring as a result of primary cardiac pathology with inadequate cardiac output. It can be thought of as persistent hypotension and tissue hypoperfusion induced by heart failure after adequate correction of preload and major arrhythmia. It is defined primarily on the basis of haemodynamic parameters:

- persistent systemic hypotension
  - systolic blood pressure (SBP) <90 mmHg for ≥30 min or
  - mean arterial pressure (MAP) ≤60 mmHg or ≤30 mmHg below baseline
  - the need for vasopressors or intra-aortic balloon counterpulsation (IABP) to achieve these targets
- and a significantly decreased cardiac index (CI) (<1.8 litres/minute/kg without support or <2.0 litres/minute/kg with support) and
- adequate or excessive filling pressures (e.g. central venous pressure (CVP) >10–15 mmHg or pulmonary artery occlusion pressure (PAOP) >15–18 mmHg)[1–4]

Clinical correlates suggestive of cardiogenic shock include: cool, mottled peripheries; oliguria; elevated jugular venous pressure; and altered mental state. Elevated serum lactate (≥2 mmol/litre) suggests inadequate cellular oxygen supply.[1,2,5]

Ischaemia is the most common underlying cause of cardiogenic shock. Other potential causes include end-stage cardiomyopathy, pericardial tamponade, myocarditis, myocardial contusion, valvular heart disease, left ventricular outflow tract obstruction, dysrhythmia and septic shock with severe myocardial depression.[2]

Advances in early intervention and revascularization (percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG)) for myocardial infarction (MI) have seen a dramatic fall in overall mortality rates for all patients presenting with MI over recent decades. However, in the subset who develop cardiogenic shock, mortality has remained high at approximately 50%.[1–3,5]

Cardiogenic shock occurs in up to 10% of patients presenting with acute myocardial infarction and is the leading cause of death. The vast majority of patients who develop ischaemic CS will have suffered ST elevated myocardial infarction (STEMI), but it is also seen after non-ST elevated myocardial infarction (NSTEMI). Patients who develop CS after NSTEMI tend to do so later in the course of their hospital admission, and are older with more significant comorbid diseases. The mortality rate for CS is not different between STEMI and NSTEMI patients.[2]

Pathophysiology

Ischaemic CS can result from a large primary MI or may develop in the setting of delayed extension of the original infarction. Of patients who develop CS after MI, registry data suggest that only up to 30% are in CS at hospital presentation. More than 70% develop CS after presentation to hospital with a median time to onset of 7–10 hours after MI in STEMI, and over 70 hours after MI in NSTEMI.[2,6,7]

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Infarction or necrosis of large areas of myocardial tissue leads to impaired contractility, a fall in stroke volume (SV), and hence cardiac output and systemic MAP. The fall in MAP — particularly diastolic blood pressure (DBP) — further compromises myocardial perfusion. These changes provoke a host of neurohormonal responses aimed at rectifying the fall in cardiac output by attempting to increase circulating blood volume and perfusion pressure. Sympathetic nervous system activation and stimulation of the renin–angiotensin—aldosterone system cause tachycardia, widespread vasoconstriction and retention of salt and water. These compensatory responses are ultimately maladaptive. In addition to disordered systolic contraction, myocardial ischaemia also results in impaired diastolic relaxation with an increase in left ventricular end-diastolic volume (LVEDV) (systolic heart failure) and pressure (LVEDP) (diastolic heart failure), increased ventricular wall stress, a further fall in coronary artery perfusion and ischaemia or infarction of already compromised myocardium. Increased LVEDP may result in pulmonary oedema with resultant increased work of breathing (and oxygen consumption) and hypoxaemia. The combination of systolic and diastolic dysfunction and maladaptive responses results in a feedback loop that cycles towards ongoing systemic hypotension, worsening tissue hypoxia and lactic acidosis, multi-organ failure, and eventual death.[2,3]

An important component of the pathophysiology of ischaemic CS is the systemic inflammatory response syndrome (SIRS). Inadequate cardiac output results in widespread tissue hypoxia and triggers the release of inflammatory mediators that generate a SIRS response. There is evidence to suggest that the SIRS response contributes significantly to vascular endothelial dysfunction and ischaemia—reperfusion injury and results in further myocardial damage in areas of hypoperfusion. Strong associations have been demonstrated between higher levels of baseline inflammatory markers and increased incidence of CS or death in the setting of STEMI.[6]

Mechanical complications of myocardial ischaemia that result in cardiogenic shock are less common in the era of early revascularization. They occur in a bimodal distribution with most occurring within 24 hours of the onset of MI, and the remainder within the first week. Mechanical complications can include mitral regurgitation (MR) — either due to papillary muscle rupture or post-infarction LV remodelling; ventricular septal rupture; LV free wall rupture with tamponade; and LV aneurysm.[7]

Assessment and evaluation

Prompt assessment of the shocked patient and diagnosis of CS is critical to enable rapid implementation of appropriate life-saving therapy. As with the approach to all critically ill patients, assessment and management should proceed simultaneously — ideally in a fully equipped resuscitation area, and using a team-based approach. A short focused history, with concurrent resuscitation, should be obtained however this may not be possible as cerebral hypoperfusion and an acute confusional state or coma is common. Collateral history is often required. Other aetiologies of a shocked state should be considered.

Clinical assessment in CS usually reveals evidence of global hypoperfusion and end organ failure. A combination of pallor or cyanosis, cool peripheries, dysrhythmias, tachypnoea and oliguria is frequently present. Distended jugular veins, chest crepitations and hypoxia are indicative of predominantly left ventricular (LV) failure, while a clear chest and the presence of Kussmaul's sign* suggest right ventricular (RV) failure. A third or fourth heart sound is often heard on auscultation of the praecordium, and there may be a systolic murmur in the setting of ischaemic mitral regurgitation (MR) or ventricular septal defect (VSD). Initial bedside investigations must include a 12-lead ECG looking for signs of myocardial ischaemia, a chest X-ray to determine evidence of pulmonary oedema (and exclude other causes of shock), and rapid focused echocardiographic assessment of cardiac function (looking particularly at biventricular function and volume status; and for regional wall motion abnormalities (RWMA); mechanical complications of MI; or alternative cause of shock, e.g. pulmonary embolus, aortic dissection). An arterial blood gas, electrolyte panel, full blood count, coagulation profile and cardiac enzyme measurement complete the initial evaluation.[2,3] A group and screen should be taken in case of alternative diagnosis or complication requiring urgent transfusion.

Monitoring should include continuous ECG and oxygen saturation, invasive arterial and central venous pressures (central venous access also facilitates administration of potent vasoactive agents), and urine output assessment via an in-dwelling urinary catheter. Pulmonary artery catheterization is not recommended routinely in the management of patients with ischaemic CS but may be considered in a minority of patients — particularly in the setting of persistent hypotension despite inotropic support.[8]

Management

Reperfusion

The goal of early resuscitation and stabilization is to treat emergent threats to life and facilitate and expedite (not delay) reperfusion. Early revascularization of the occluded coronary vessel(s) is paramount to effective management of ischaemic CS with demonstrable mortality benefit. In settings where primary PCI (pPCI) or cardiothoracic surgical interventions are not readily available, both in-hospital and pre-hospital thrombolysis should be considered.[6,7,9]

Mechanical complications require urgent surgical intervention.

Resuscitation and stabilization

The principles of resuscitation and stabilization of the patient in CS are the same as those of any shocked patient — aiming to alleviate symptoms, stabilize haemodynamics and respiratory function, and identify and treat the underlying cause. Intubation and mandatory ventilation are often necessary to manage hypoxia, respiratory distress or obtundation, as well as to reduce the work of breathing. The haemodynamic effects of positive pressure ventilation (PPV) may assist by decreasing afterload in LV failure. However, the implications of PPV for the RV should also be considered (reduced preload and increased afterload). Furthermore, the choice of intubation drugs should

* Kussmaul's sign: a paradoxical rise in jugular venous pressure on inspiration in the spontaneously breathing patient.