Ischaemic cardiogenic shock

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Abstract

Recognition of cardiogenic shock in the setting of myocardial ischaemia has important prognostic and therapeutic implications. Mortality remains high in the order of 50%, despite introduction of urgent revascularization. Resuscitative efforts should focus to avoid multi-organ dysfunction with further spiralling instability that commonly is irreversible. To interrupt these processes, timely recognition and restoration of adequate perfusion is mandatory. The therapeutic means to achieve this beyond early revascularization have been widely debated, in particular the extent of pharmacological support and the timing of mechanical support form key components of modern intensive care treatment. Further research work on optimal support and patient selection for more advanced therapies is required.

Keywords Acute myocardial infarction; 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:

- identify patients with cardiogenic shock on clinical assessment among patients with cardiac ischaemia
- explain the pathophysiology and potential interventions to reverse the low cardiac output state
- formulate an initial management plan for a patient in cardiogenic shock in regards to prioritization and initial resuscitation
- evaluate role of early revascularization in the setting of myocardial ischaemia

Definition

In cardiogenic shock (CS) the heart is failing to provide adequate cardiac output to maintain end organ function. Most commonly accepted parameters for diagnosis of CS have been described as follows[1–3]

- persistent hypotension
  - systolic blood pressure <80–90 for mmHg ≥30 minutes or mean arterial pressure 30 mmHg lower than baseline
  - need for vasopressors to achieve these targets
- severe reduction in cardiac index
  - < 1.8 L/min/m^2 without support or <2.0–2.2 L/min/m^2 with support
- adequate or elevated filling pressures
  - left ventricular (LV) end-diastolic pressure (estimated by pulmonary occlusion pressure) >18 mmHg or
  - right ventricular (RV) end-diastolic pressure (estimated by central venous pressure) >10–15 mmHg.

The limitation of these predominant haemodynamic parameters is an over-reliance on pulmonary artery catheter measurements. In most circumstances they are not readily available nor is a pulmonary artery catheter routinely used for management. For practical purposes a patient in shock, peripherally cold with a raised arterial lactate level and a bedside echocardiography showing severe systolic dysfunction with likely elevated filling pressures, should be managed as cardiogenic shock in the absence of an alternate diagnosis.

Most commonly CS will follow acute myocardial infarction (80%),[4] typical with an ST elevation myocardial infarction (STEMI). Delayed cardiogenic shock may also occur with non-ST elevation myocardial infarction (NSTEMI) with or without significant pre-existing myocardial disease.[2]

Differential diagnoses that may also present with shock, a low cardiac output state and elevated CVP include non-ischaemic cardiac causes such as end-stage cardiomyopathy, cardiac tamponade, myocarditis, cardiac contusion, valvular heart disease and outflow tract obstructions (left or right ventricular). Important non-cardiac causes are pulmonary embolism, severe asthma or septic shock with severe cardiomyopathy.

Cardiac mortality in the western world has steadily fallen; however, this is less so for CS as the last decades have shown mortality rates around 50% in comparison to historical data. This suggests an even higher mortality rate up to 80% pre early vascularization.[3] The percentage of patients with AMI who develop CS has remained consistent between 5 and 10%. Overall the degree of haemodynamic derangement indicates severity and prognosis and does not principally differ in STEMI or NSTEMI.

Pathophysiology

Ischaemic CS frequently develops in hospital after an ischaemic insult; however, up to 30% of patients are in CS at the time of hospital presentation, typically with STEMI.[3] Large areas of myocardium affected by ischaemia or necrosis lead to impaired contractility, fall in stroke volume and therefore cardiac output. This ultimately leads to a fall in systemic mean arterial pressure (MAP). Particularly the fall in diastolic blood pressure 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.[4] 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 and/or right ventricular end-diastolic volume and pressure. Increased wall tension leads to increased oxygen requirements (according to the law of Laplace) and a further fall of coronary artery perfusion pressure of an

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already compromised myocardium. Differential right and left ventricular output with increased left ventricular end-diastolic pressure will result in pulmonary oedema with consequently increased work of breathing and hypoxaemia. The combination of systolic and diastolic dysfunction and maladaptive responses results in ongoing systemic hypotension, worsening tissue hypoxia, lactic acidosis, multi-organ failure, and eventual death.[1]

Systemic inflammatory response syndrome (SIRS) is an important component of the pathophysiological response of ischaemic CS. The principal triggers for this seem tissue hypoxia and early multi-organ failure leading to release of inflammatory mediators that generate a SIRS response. There is evidence to suggest that SIRS also contributes significantly to vascular endothelial dysfunction and itself to ischaemic reperfusion injury and further myocardial damage in areas of hypoperfusion.[3] Strong associations have been demonstrated between higher levels of baseline inflammatory markers and an increased incidence of CS or death in the setting of STEMI.

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, either due to papillary muscle rupture or post-infarction LV remodelling; ventricular septal rupture; LV free wall rupture with tamponade; and LV aneurysm.[2]

Recognizing ischaemic cardiogenic shock

Clinical gestalt	Pale, clammy, cold peripheries, increased capillary refill time Tachycardia, hypotension, jugular venous distension
ECG	STEMI or STEMI-equivalents[7]: posterior MI, acute left main occlusion Cardiac arrhythmias (heart block, ventricular dysrhythmias) Caution: mimics of ischaemia e.g tako-tsubo cardiomyopathy, myocarditis
Echocardiography	Bedside study severe LV dysfunction reduced cardiac output Regional wall motion abnormality may indicate site of culprit lesion
End organ dysfunction	Impaired mentation, seizure (significant cerebral hypoperfusion) Oliguria or anuria Troponin may be normal on initial presentation ALT rapid rise over time indicator of ischaemic hepatitis
Lactate	>2 mmol/L may rise rapidly

Table 1

Assessment and evaluation

Patients with STEMI are by far the largest group of patients at risk of developing CS, which may be occurring at presentation or a period of time (7–10 hours) post insult. NSTEMI patients may also develop CS. This is often delayed and in hospital over hours to days.[5] A third patient group can be differentiated with a pre-existing grossly abnormal cardiac function (e.g. ischaemic cardiomyopathy, HOCM) with additional acute myocardial ischaemia.

Identified patients mandate immediate transfer to an acute care environment for simultaneous assessment and management. It is vital to have capabilities for invasive monitoring and a team to provide cardiorespiratory support including: intubation, central venous access, and advanced cardiac life support if required.

Priority needs to be given in establishing the cause of shock and diagnosing cardiac ischaemia. Generally, a focused history will reveal preceding ischaemic symptoms and/or symptoms suggestive of rapidly progressing heart failure. Global hypoperfusion and end-organ dysfunction are the key clinical features[1,2,6] on assessment as outlined in Table 1. In addition, features suggesting prominent LV failure are hypoxia, chest crepitations and third or fourth heart sound which are often heard on auscultation. A prominent pansystolic murmur should alert to a potentially acute mitral regurgitation. In contrast, clear chest auscultation and the presence of Kussmaul's sign suggest RV failure which, in isolation, leads less commonly to CS.

Initial bedside investigations must include serial 12-lead ECGs looking for signs of myocardial ischaemia or evolving infarction.[7] A chest X-ray will determine pulmonary oedema and assist in the exclusion of other causes of shock. Focused echocardiography is used to look for RV-, LV dysfunction and regional wall motion abnormalities, which suggests the coronary artery territory involved; further mechanical complications of MI; or alternative cause of shock such as pulmonary embolus or even aortic dissection may be diagnosed. An arterial blood gas, electrolyte panel, full blood count, coagulation profile and cardiac enzyme measurement completes the initial evaluation. Normal troponin early in the presentation does not exclude coronary artery occlusion.[3]

Monitoring should include continuous ECG and pulse oximetry, 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 routine, but may be employed if uncertainty exists regarding the cardiogenic component of the shock state as a diagnostic tool, while most clinicians will choose alternative measures to monitor cardiac output.

Management

Resuscitation

Patients in ischaemic CS require timely resuscitation to stabilize their circulatory and respiratory function. Senior judgement is advised for patients presenting in CS with criteria for urgent revascularization on ECG. This will assist with determining and prioritizing tasks pre percutaneous coronary intervention (PCI) to stabilize the patient. The goal is to minimize time to PCI.

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