Chapter Eleven

Acute Myocardial Infarction: Complications

CARDIOGENIC SHOCK

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DEFINITION

Cardiogenic shock remains the leading cause of death in patients hospitalized with myocardial infarction. A primary cardiac dysfunction resulting in inadequate tissue perfusion is the underlying prerequisite. Clinically, cardiogenic shock is defined as: (1) sustained systolic blood pressure <90 mmHg (12 kPa) without hypovolemia, in combination with (2) signs of organ hypoperfusion (e.g. oliguria, impaired consciousness) and (3) signs of sympathetic activation (e.g. cool extremities, sweating). Diagnostic specificity is improved by the finding of low cardiac index (<2.2 l/minute) despite elevated left ventricular filling pressure (>15 mmHg [2 kPa]).

EPIDEMIOLOGY

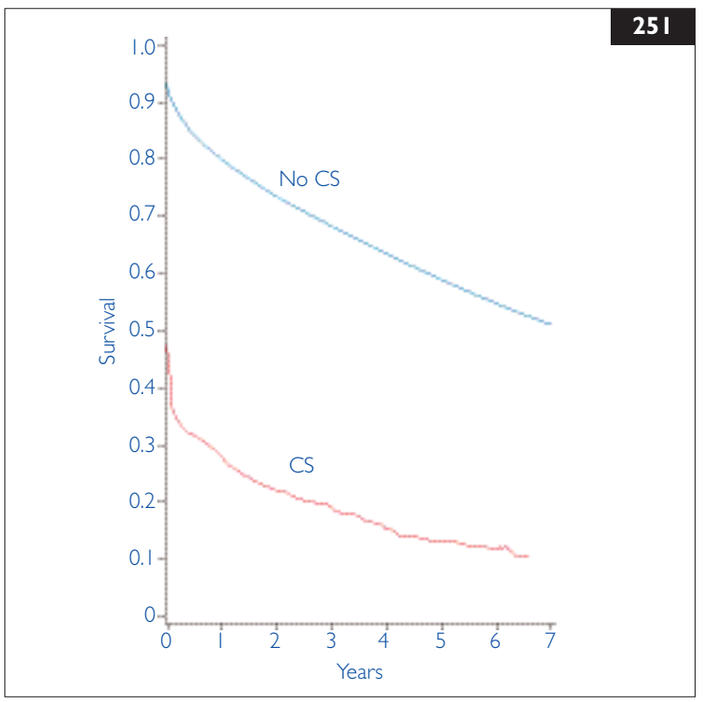
Cardiogenic shock occurs in 7–10% of acute myocardial infarction (AMI). Risk factors include older age, diabetes, prior infarction, and female gender. Although large infarctions and anterior infarctions are predominant, shock can result from an infarct in any location. In fact, almost one-third of cardiogenic shocks are complications to a non-Q wave infarction. Median delay from onset of symptoms is 7 hours with a trend towards later shock development in non-ST elevation AMI. The majority of shocks therefore develop after hospital admission, 60–75% within the first 24 hours. Short-term mortality exceeds 60–70% (**251**) and is even higher in older patients and patients who develop shock more than 48 hours after AMI.

PATHOGENESIS

Left ventricular failure is the most common cause of cardiogenic shock, accounting for 75–80% of cases. Isolated right ventricular failure occurs in 3%, while mechanical complications such as severe mitral regurgitation, ventricular septal rupture, or cardiac tamponade account for 10–15% of cases of cardiogenic shock (see Chapter 11, Mechanical Complications). Ventricular dysfunction results in low stroke volume reducing cardiac output. Myocardial perfusion is compromised by systemic hypotension and increased left ventricular end-diastolic pressure (LVEDP). In some patients, hypotension and myocardial depression may be further aggravated by a systemic inflammatory response. Together with neurohormonal activation, these changes contribute to a cycle of worsening ischemia and organ perfusion.

CLINICAL PRESENTATION

The typical patient with cardiogenic shock presents with hypotension, sinus tachycardia, cool extremities, oliguria, pulmonary rales, and respiratory distress due to pulmonary congestion. While the clinical appearance of this syndrome is often easily recognizable, it must be emphasized that shock can be



251 Kaplan–Meier survival curves for consecutive patients with acute myocardial infarction with (n=444) and without (n=6226) cardiogenic shock (CS). (From Lindholm MG *et al.* [2003]. Cardiogenic shock complicating acute myocardial infarction: Prognostic impact of early and late shock development. *Eur. Heart J.* **24**:258–265. With permission from the publisher, WB Saunders.)

present or emerging without the presence of these classic features (e.g. a significant proportion of patients with left ventricular failure show no signs of pulmonary congestion). Similarly, hypotension, which traditionally has been considered the hallmark of circulatory failure, may not necessarily be the dominating problem in the early phases of shock because blood pressure may be maintained initially by a high sympathetic drive. This early phase is termed preshock and the majority of patients with fully developed shock have passed through this preshock period. Thus, the presence of AMI, sinus tachycardia, cool extremities, and low urine output, indicative of a low cardiac output state, should be considered important warning signals of evolving cardiogenic shock even in patients with normal systemic blood pressure.

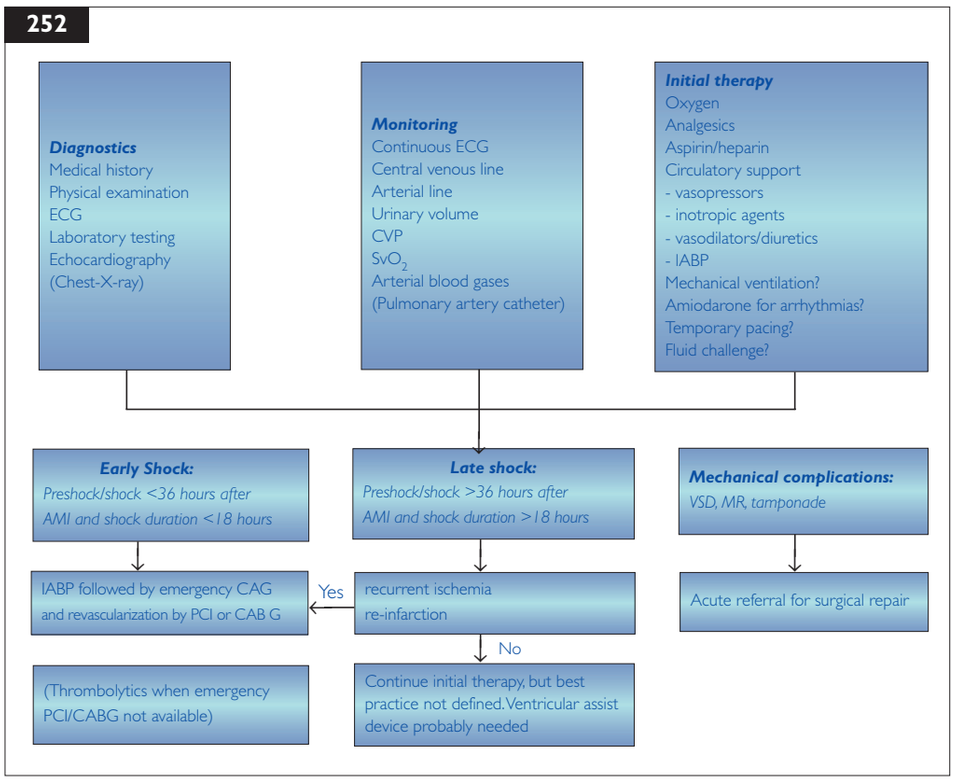
DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Cardiogenic shock is an emergency condition. Consequently, diagnostic verification, initial hemodynamic stabilization, and plans for definitive therapy must be dealt with simultaneously (**252**). A medical history, electrocardiogram (ECG), physical examination, and echocardiography constitute the initial diagnostic procedures. Echocardiography is necessary to confirm the diagnosis of primary ventricular failure as opposed to mechanical complications or other noncardiac causes of shock (e.g. septic shock, aortic dissection, pulmonary emboli) needing a different treatment strategy (**253**). Invasive hemodynamic monitoring is used to confirm the presence of low cardiac output, to exclude volume depletion and primary right ventricular infarction, and to estimate filling pressures and treatment response.

MANAGEMENT

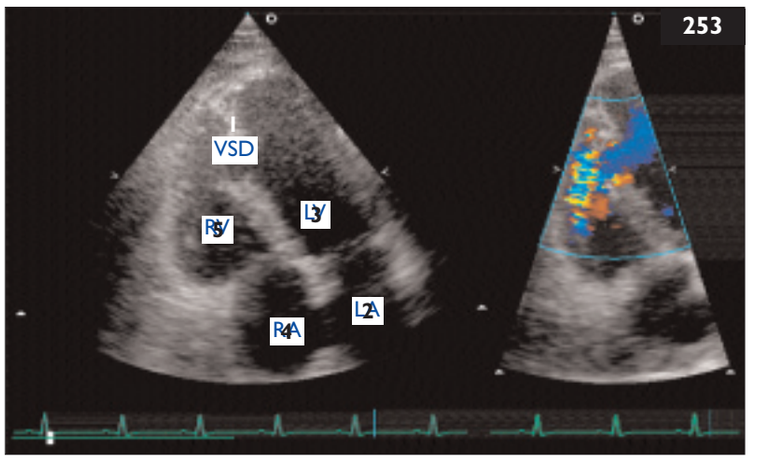
Multiple data suggest that an aggressive hemodynamic- and revascularization-based approach is associated with improved outcome in cardiogenic shock. Initial therapy of cardiogenic shock therefore optimally includes: (1) early identification, (2) intensive care monitoring including a low threshold towards mechanical ventilation, (3) circulatory support with sympathomimetic inotropic and vasopressor agents, (4) coronary reperfusion (**254**), and (5) general measures including correction of acidosis and significant arrhythmias and discontinuation of negative inotropes, anti-hypertensive, or nephrotoxic agents. Mechanical circulatory support with an intra-aortic balloon pump (IABP) is usually indicated and should routinely be performed before attempting coronary reperfusion (**255**).

In normovolemic, hypotensive patients, dopamine or norepinephrine are the initial drugs of choice. Due to their vasodilatory properties, inotropes such as dobutamine or the phosphodiesterase inhibitor milrinone should await correction of overt hypotension. Peripheral vasodilators such as nitroglycerin or nitroprusside may be combined with inotropes in patients with high filling pressures. IABP improves coronary perfusion, reduces

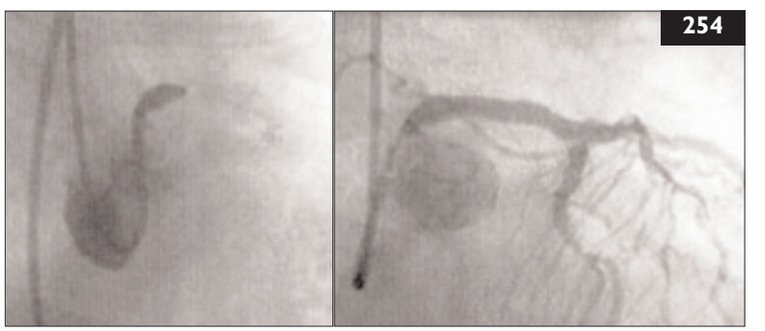


252 Management of cardiogenic shock complicating acute myocardial infarction. AMI: acute myocardial infarction; CABG: coronary artery bypass grafting; CAG: coronary angiography; CVP: central venous pressure; IABP: intra-aortic balloon pump; MR: mitral regurgitation; PCI: percutaneous intervention; SvO_2: central venous oxygen saturation; VSD: ventricular septal defect.

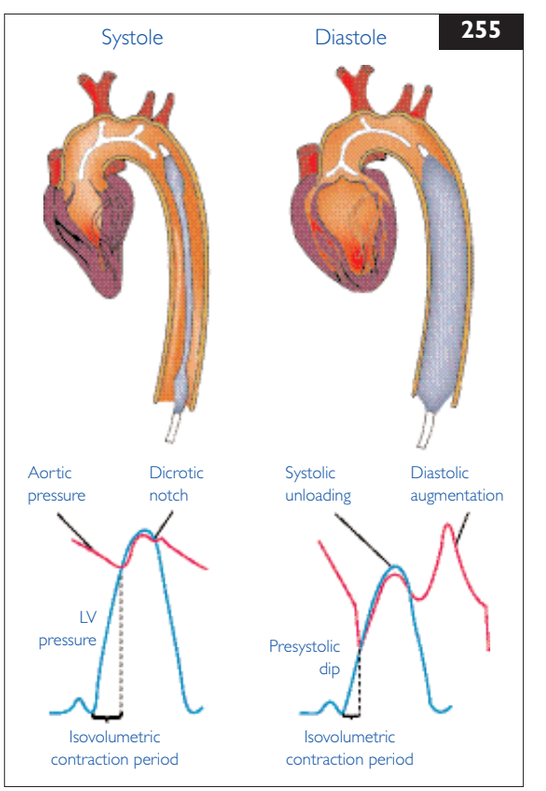
253 Left: Echocardiographic four-chamber view showing a large ventricular septal defect (1) complicating an acute myocardial infarction. 2: left atrium; 3: left ventricle; 4: right atrium; 5: right ventricle. **Right**: Doppler image of the same patient showing a large left-to-right shunt.



254 A 75-year-old female was admitted with severe chest pain. At arrival she was pale with cool extremities and an arterial oxygen saturation of 84%. The arterial blood pressure was 82/53 mmHg (10.9/7.1 kPa), heart rate 102 bpm, and electrocardiogram showed an anterior ST elevation acute myocardial infarction. After initial respiratory/circulatory stabilization, emergency coronary angiography was performed within 3 hours of start of symptoms. The angiography shows an occluded left main coronary artery (left). The lesion was immediately managed by percutaneous transluminal coronary angioplasty and stenting of the left main artery (right).



255 The principle of intra-aortic balloon pump. Initiation of balloon inflation is timed to the arterial dicrotic notch, producing an augmentation in proximal aortic diastolic pressure. Deflation of the balloon is timed to begin just before the onset of the next ventricular systole, which produces the systolic unloading effect (presystolic dip). LV: left ventricle. (Modified from Cercek and Shah [2001]. In: *Cardiology*. MH Crawford, JP Di Marco, WJ Paulus (eds). Mosby, London.)



afterload, increases cardiac output without increasing oxygen demand, and may allow reduction in pharmacologic vasopressor therapy. In addition, IABP insertion reduces the incidence of serious complications during invasive procedures.

These initial treatment measures have not been shown to improve prognosis in cardiogenic shock and should only be regarded as temporary means to establish adequate tissue perfusion while awaiting the effect of a coronary reperfusion. Therefore, in most patients with early shock due to ventricular failure and short shock duration, the next treatment goal is myocardial revascularization. In clinical practice this condition may be defined as shock duration <18 hours and onset of shock <36 hours after AMI. This strategy is supported by data from the randomized SHOCK trial, where emergency invasive revascularization tended to reduce 30-day mortality and significantly reduced 6-month and 12-month mortality (**256**). In particular, patients <75 years old seem to benefit from invasive therapy, with a 12-month mortality of 48% as compared to 77% in medically treated patients. Since the effect of thrombolysis is reduced in cardiogenic shock, thrombolytic therapy should be reserved for patients who cannot rapidly be transferred for emergency coronary revascularization with coronary angioplasty or bypass surgery. To improve the chances of reperfusion, thrombolytics should then be administered on top of proper pharmacologic and mechanical (IABP) circulatory support.

PROGNOSIS

Cardiogenic shock complicating AMI continues to be a very serious condition (*Table 32*). Thirty-day mortality in medically treated patients is at least 60–70%. With best practice therapy, including mechanical circulatory support and early revascularization, 30-day mortality is still >30–40%. For the few patients surviving 30 days, the long-term mortality rate is slightly higher than in patients with myocardial infarction and an in-hospital diagnosis of heart failure without cardiogenic shock. The majority of these patients are classified in NYHA functional class I/II.

SUMMARY

Several sets of data suggest that if cardiogenic shock is recognized early and managed with rapid initiation of up-to-date supportive measures and emergency invasive revascularization, outcome can be improved. On the other hand, it is likely that there are groups of older patients, and patients with long lasting cardiogenic shock and fully developed multi-organ failure, where invasive therapy is futile. Therefore, management of cardiogenic shock needs to be individualized and treatment strategy carefully selected.

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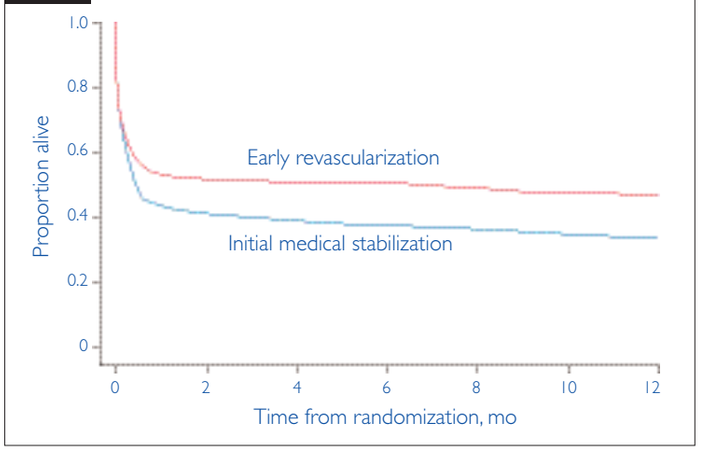

256 One year Kaplan–Meier survival curve for patients randomized to early emergency revascularization (n=152) and initial medical stabilization (n=149)(p<0.04). Data from the SHOCK trial. (From Hochman *et al.* [2001]. One-year survival following early revascularization for cardiogenic shock. *JAMA* **285**:190–192. Copyrighted 2001, American Medical Association.)

Table 32 principles in cardiogenic shock complicating acute myocardial infarction

- Cardiogenic shock is an emergency.
- Most shock patients slowly slide into shock after admission to hospital.
- Act immediately on signs of organ hypoperfusion.
- Echocardiography and intensive care monitoring/therapy are standard.
- Pharmacological and mechanical circulatory support are generally both needed.
- Use IABP/dopamine as first-line therapy in hypotensive patients.
- Add inodilators/vasodilators when mean arterial pressure >65 mmHg (8.7 kPa).
- Every patient with early shock should be evaluated for emergency invasive revascularization.
- Insert IABP before initiation of CAG/PCI.
- In the initial clinical setting, left ventricular ejection fraction carries little prognostic information.