Cardiogenic Shock

Joshua B. Moskovitz, MD, MPH, MBA*, Zachary D. Levy, MD,
Todd L. Slesinger, MD

KEYWORDS

• Cardiogenic shock • Heart failure • Congestive heart failure • Acute heart failure
• Cardiac failure

KEY POINTS

- Cardiogenic shock is defined as a state of hypoperfusion and end-organ dysfunction resulting from profoundly decreased cardiac output.
- Acute MI and left ventricular failure is the most common cause overall; less commonly, shock may be precipitated by acute valvular dysfunction and aortic dissection, both of which are surgical emergencies.
- Patients in cardiogenic shock require urgent evaluation for reperfusion therapies, including coronary artery bypass graft and PCI.
- Norepinephrine is the preferred agent for patients in cardiogenic shock who require pressor support; dobutamine and milrinone are second-line adjuncts that may worsen hypotension when used in isolation.
- In the emergency setting, IABP and percutaneous LVAD placement may be used as temporizing measures to improve patient hemodynamics while awaiting definitive treatment.

INTRODUCTION

Overall mortality in acute coronary syndrome (ACS) has dropped significantly in the past several decades, from 10.4% in 1990 to 6.3% in 2006.[1] The improvement in survival among patients with ACS can be attributed, in part, to advances in pharmacologic and mechanical interventions.[2–4] Many of these therapies target cardiogenic shock, a relatively common complication of ACS with an associated mortality rate between 50% and 80%.[2] Despite the need for well-defined, evidence-based treatment algorithms in this critically ill patient population, there is a paucity of data. This article describes the pathophysiology of cardiogenic shock, organizes treatment considerations, and catalogues advances in current practice.

No financial disclosures or conflicts of interest.
Department of Emergency Medicine, Hofstra North Shore-LIJ School of Medicine, 300 Community Drive, Hempstead, NY 11030, USA
* Corresponding author. Department of Emergency Medicine, North Shore University Hospital, 300 Community Drive, Hempstead, NY 11030.
E-mail address: joshmoskovitz@gmail.com

Emerg Med Clin N Am 33 (2015) 645–652
http://dx.doi.org/10.1016/j.emc.2015.04.013 **emed.theclinics.com**
0733-8627/15/$ – see front matter

PATHOPHYSIOLOGY

Cardiogenic shock is best described as a state of heart failure that results in inadequate cardiac output, hypoperfusion, and end-organ dysfunction. Defining features of cardiogenic shock appear in **Table 1**. The diagnosis was historically made via pulmonary artery catheterization, but noninvasive echocardiography is increasingly used.[5,6] In addition to hypotension, clinical signs may include cool extremities, decreased urine output, mottled skin, and altered mental status.

Ultimately, cardiogenic shock involves left and/or right ventricular dysfunction. The causes of ventricular failure are numerous, including acute myocardial infarction (MI), myocarditis/pericarditis, cardiomyopathies, acute or chronic valvular dysfunction, aortic dissection, myocardial contusion, and myocardial depression from septic shock.[7] Mechanical etiologies of cardiogenic shock (including valvular rupture and aortic dissection) are true surgical emergencies, requiring prompt diagnosis and surgical referral. These conditions often carry a poor prognosis.[8] The most common overall cause of cardiogenic shock, however, is left ventricular dysfunction in the setting of ACS involving the left-sided coronary arteries.[7] This article focuses predominantly on MIs as a cause of cardiogenic shock, and the various treatment options that emergency physicians should consider.

The primary insult is typically an infarct causing either reversible ischemia or irreversible injury to the left ventricle. As a result, coronary perfusion decreases, thereby decreasing cardiac output. The drop in cardiac output results in hypoperfusion, triggering catecholamine release to improve contractility and blood pressure. This, in turn, increases myocardial oxygen demand.[5] The result is a cycle of decreasing myocardial blood supply with increasing myocardial oxygen demand, a chain reaction that can manifest as rapid clinical deterioration.

Right ventricular MI accompanies inferior wall ischemia in up to 50% of cases, and generally does not exist in the absence of left ventricular MI. Hemodynamic stability in right ventricular MI is variable; some patients remain asymptomatic, whereas others experience severe hypotension and cardiogenic shock. Right ventricular MIs are more often complicated by arrhythmias, including complete atrioventricular or sinoatrial blocks.[9] It is important to consider right ventricular extension when evaluating a patient with inferior wall ischemia, and right precordial leads may be necessary when obtaining an electrocardiogram to evaluate for extension of an inferior wall MI. ST-segment elevation in lead V4R greater than 1 mm is reportedly 100% sensitive and 87% specific for right ventricular infarction.[9]

Several risk factors have been associated with increased mortality in cardiogenic shock in the setting of MI, include advanced age (>75) and increased serum lactate (>6.5 mmol/L).[10] Indeed, elevated serum lactate has previously been identified as an independent predictor of the development of cardiogenic shock.[11] Failed reperfusion and history of prior MI have also been identified as high-risk features.[12]

Table 1
Definition of cardiogenic shock

Persistent hypotension	Systolic BP <90 or MAP >30 below baseline
Severe reduction in cardiac index	<1.8 L/min/m^2 without support
Adequate or elevated filling pressure	LV end diastolic pressure >18 mm Hg RV end diastolic pressure >10–15 mm Hg

Abbreviations: BP, blood pressure; LV, left ventricular; MAP, mean arterial pressure; RV, right ventricular.