Cardiogenic Shock

Semhar Z. Tewelde, MD[a], Stanley S. Liu, MD[b], Michael E. Winters, MD[c,*]

KEYWORDS

• Cardiogenic shock • Hypoperfusion • Inotropic medications • Vasopressor medications
• Percutaneous coronary intervention • Echocardiography • Dobutamine
• Intra-aortic balloon pump counterpulsation

KEY POINTS

- Bedside echocardiography should be performed in the initial evaluation of patients with cardiogenic shock (CS) to assess intravascular volume, determine the left ventricular ejection fraction, and diagnose a pericardial effusion or obstructive lesions.
- Initial resuscitation of the patient with CS is aimed at restoring cardiac output and tissue perfusion.
- Management of the patient with CS includes a combination of medical therapies, reperfusion therapy, and mechanical support.
- Vasopressor and inotrope medications can increase myocardial oxygen consumption and increase the risk of arrhythmias.
- Consider mechanical support devices in patients with CS who are not responding to pharmacologic therapy.

INTRODUCTION

Cardiogenic shock (CS) is commonly defined as a physiologic state in which cardiac pump function is inadequate to perfuse the tissues. If CS is not rapidly recognized and treated, tissue hypoperfusion can quickly lead to organ dysfunction and patient death. Patients with CS can present with a myriad of symptoms and physical examination findings that range from subtle hemodynamic alterations to overt cardiovascular collapse. Once diagnosed, patients with CS require rapid initiation of therapy to prevent unnecessary increases in morbidity and mortality. Treatment of patients with CS is challenging, as many will require a combination of vasoactive medications and mechanical support. This article reviews the most common etiologies of CS, along with critical components in the diagnostic evaluation of patients with CS. Medical management and mechanical support of patients with CS also is discussed.

DIAGNOSIS

Commonly accepted hemodynamic criteria for the diagnosis of CS are listed in **Box 1**. In addition to these hemodynamic criteria, patients with CS also exhibit signs and symptoms of pulmonary congestion and tissue hypoperfusion. These signs and symptoms can include dyspnea, rales, elevated jugular venous pressure (JVP), altered mental status, narrow pulse pressure, reduced urine output (less than 20 mL/h), cool and clammy skin, and elevated lactate levels.[1,2]

EPIDEMIOLOGY

Many patients with CS die before hospital arrival. As a result, it is difficult to determine the true incidence of CS. What is clear, however, is that the proportion of intensive care unit admissions with CS has doubled from 4% to 8% over the past 15 years.[3] Currently, CS complicates

Disclosure: The authors have no relevant financial relationships to disclose.
[a] Department of Emergency Medicine, University of Maryland School of Medicine, 110 South Paca Street, 6th Floor, Suite 200, Baltimore, MD 21201, USA; [b] Division of Cardiovascular Medicine, University of Maryland School of Medicine, 110 South Paca Street 7-N-127, Baltimore, MD 21224, USA; [c] Emergency Medicine/Internal Medicine/Critical Care Program, University of Maryland School of Medicine, 110 South Paca Street, 6th Floor, Suite 200, Baltimore, MD 21201, USA
* Corresponding author.
E-mail address: mwinters@em.umaryland.edu

Cardiol Clin 36 (2018) 53–61
https://doi.org/10.1016/j.ccl.2017.08.009

Box 1
Diagnostic criteria for cardiogenic shock

- Hypotension
  - Systolic blood pressure less than 90 mm Hg OR
  - A reduction in mean arterial pressure of 30 mm Hg or more from the patient's baseline
- Reduced Cardiac Index
  - Less than 2.2 L/min/m^2 body surface area for patients receiving vasoactive or mechanical support
  - Less than 1.8 L/min/m^2 body surface area for patients not receiving vasoactive or mechanical support
- Adequate Filling Pressure
  - Pulmonary artery wedge pressure greater than 15 mm Hg

Adapted from Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. Circulation 2008;117:686; with permission.

approximately 8% to 9% of patients with an ST-segment elevation myocardial infarction (STEMI), whereas the incidence of CS in patients with a non-ST-segment myocardial infarction (NSTEMI) is approximately 2.5%.[4–6] Mortality for patients with CS is unchanged in recent years and remains unacceptably high at approximately 50%.[7]

ETIOLOGIES

The SHOCK Trial Registry is the largest data set from which to assess the various cardiac etiologies of CS.[8] By far, the most common cardiac cause of CS is acute left ventricular failure in the setting of an STEMI.[8] Most often this is due to anterior wall myocardial infarction and accounts for almost 79% of patients with CS.[8] Mechanical complications of ischemic heart disease include severe mitral regurgitation (7%), ventricular septal rupture (4%), right ventricular failure (3%), and tamponade (1.4%).[8] Of these cardiac causes, ventricular septal rupture carries the highest mortality.

CS can also result from nonischemic cardiac conditions. These conditions are listed in **Table 1**. It is important to consider these nonischemic etiologies in patients presenting with typical signs and symptoms of CS but with nonspecific findings on the electrocardiogram (ECG) and negative laboratory values for myocardial infarction.

Table 1
Nonischemic etiologies of cardiogenic shock

Etiology	Examples
Pharmacologic	Beta blockers Calcium channel blockers Digoxin toxicity
Primary ventricular dysfunction	Acute myocarditis Stress cardiomyopathy (ie, Takatsubo cardiomyopathy) Nonischemic cardiomyopathy (eg, sarcoidosis, amyloidosis, hemochromatosis)
Outflow obstruction	Valvular stenosis Left ventricular outflow obstruction (eg, in hypertrophic cardiomyopathy)
Acute valvular regurgitation	Trauma Degenerative disease Endocarditis
Endocrine	Severe hypothyroidism
Pericardial disease	Cardiac tamponade Pericardial constriction
Tachyarrhythmias	Supraventricular/atrial tachyarrhythmias Monomorphic VT Polymorphic VT (ie, Torsades de Pointes)
Bradyarrhythmias	Sinus node dysfunction (eg, sick sinus syndrome) AV node dysfunction (eg, AV nodal block)

Abbreviations: AV, atrioventricular; VT, ventricular tachycardia.

INITIAL EVALUATION

Emergent evaluation of the patient with suspected CS is critical. The immediate goals of the initial evaluation are to identify signs of tissue hypoperfusion and determine the etiology of shock. Select findings from the physical examination, ECG, chest radiograph (CXR), laboratory studies, and bedside echocardiogram are extremely helpful in achieving these initial goals.

Physical Examination

A focused physical examination should be performed in patients with suspected CS. Pertinent findings from the examination are listed in **Box 2**. Although physical examination findings of abnormal cardiac function often lack sensitivity, the presence of an elevated JVP, an S3, and a