Interventional Cardiology

Cardiogenic shock

Abstract

Introduction: The cardiogenic shock is a state of endorgan dysfunction, secondary to insufficient cardiac output despite adequate preload, as a result of left ventricular, right ventricular or biventricular dysfunction. Cardiogenic shock occurs in up to 10% of patients presenting with acute myocardial infarction and is the leading cause of death.

Objective: To conduct a review of cardiogenic shock.

Methodology: The search was performed in the databases PUBMED/MEDLINE, EMBASE and Google Scholar with the search terms: Cardiogenic shock and Epidemiology or Pathophysiology or Diagnosis or Treatment. We selected the most relevant studies on cardiogenic shock.

Results: We provided a general description of the definition, epidemiology, clinical manifestations, causes, pathophysiology and treatment of cardiogenic shock. The clinical syndrome of cardiogenic shock has been described as: a systolic blood pressure of less than 90 mm Hg, or greater than 30 mm Hg below baseline BP, for at least 30 minutes, with signs of a reduced cardiac output. The most common cause of cardiogenic shock is acute coronary syndrome, accounting for about 70% to 80% of cardiogenic shock cases. The basic treatment measures include initial stabilization with volume expansion to obtain euvolaemia, vasopressors, and inotropes plus additional therapy for the prevention or treatment of multiorgan system dysfunction. Norepinephrine is associated with fewer arrhythmias and may be the vasopressor of choice in many patients with cardiogenic shock. Coronary reperfusion is the main evidence-based therapeutic intervention for patients with acute MI presenting with cardiogenic shock. Traditionally, intra-aortic balloon pumps have been the main support system, there has been interest in improved mechanical support devices because these may make revascularization safer.

Keywords: Cardiogenic Shock ▪ Epidemiology ▪ Pathophysiology ▪ Diagnosis Treatment

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Introduction

The term shock was first used in 1743 but Harrison in 1935, and Blalock in 1940, were the first to classify shock according to cause: cardiogenic, oligemic, vasogenic, and neurogenic [1,2]. The cardiogenic shock is a state of endorgan dysfunction, secondary to insufficient cardiac output despite adequate preload, as a result of left ventricular, right ventricular or biventricular dysfunction [2,3]. Cardiogenic shock is defined as systemic tissue hypoperfusion due to inadequate cardiac output despite adequate circulatory volumen [3]. The clinical syndrome of cardiogenic shock has been described as: a systolic blood pressure of less than 90 mm Hg, or greater than 30 mm Hg below baseline BP, for at least 30 minutes, with signs of a reduced cardiac output. Signs of reduced cardiac output may be manifested as reduced urine output (<20 mL/h), impaired cognitive function, and evidence of peripheral vasoconstriction. The diagnosis is confirmed when cardiac index is less than 2.2 L/m2 body surface area, and pulmonary capillary wedge pressure greater than 15 mm Hg [3,4]. In this review, we offer an overview of cardiogenic shock.

Epidemiology

Cardiogenic shock occurs in up to 10% of patients presenting with acute myocardial infarction and is the leading cause of death [5]. The most common cause of cardiogenic shock during acute myocardial infarction is left ventricle failure (78.5%), followed by severe mitral regurgitation (6.9%), ventricular septal rupture (3.9%), right ventricle failure (2.8%), and cardiac tamponade (1.4%) [6].

In a recent large cohort of 21210 patients with ST segment elevation myocardial infarction, cardiogenic shock was observed in 8.9% of patients with the incidence increasing over time and high mortality of 45%–70% [7]. Cardiogenic shock was observed in one out of ten STEMI patients and is most often present already on admission (56%)[8].

Cause of cardiogenic shock

The most common cause of cardiogenic shock is acute coronary syndrome, accounting for about 70% to 80% of cardiogenic shock cases. Other causes of cardiogenic shock predominantly include the decompensation of chronic heart failure and right ventricular failure in about 5% of cases [9]. Cardiogenic shock can be caused by an acute cardiac condition or a systemic illness that triggers a chronic cardiac condition associated with minimal cardiac reserve. Unstable angina, postcardiotomy syndrome, valvular heart disease, myocardial disease (such as myo-carditis), LV outflow obstruction in hypertrophic cardiomyopathy, stress-induced cardiomyopathy, pericardial tamponade, congenital lesions, and mechanical injury to the heart have all been implicated in the pathogenesis of cardiogenic shock [10].

Pathophysiology

The essential feature is that decreased coronary blood flow results in decreased cardiac output. This decrease in cardiac output leads to hypotension and progressively more cardiac ischemia and dysfunction [11]. Cardiogenic shock is associated with primary left ventricular dysfunction when > 40% of myocardium is damaged. Initially renal compensatory mechanism causes fluid retention to increase preload. Vasoconstriction to sustain a blood pressure increases afterload, further impairing performance of the heart and increases the myocardial oxygen and nutritional demand. Increased demand and inadequate perfusion worsens ischemia, and the vicious cycle sets in, which if not interrupted, result in irreversible cardiogenic shock and ends in death [11,12].

Hemodynamics

Hemodynamic monitoring with a pulmonary artery catheter plays a crucial role in the management of patients with cardiogenic shock. It is important to define hemodynamics, assess interaction between the right ventricle and left ventricle and guide selection of pressors and inotropes. Hemodynamic data, such as cardiac power output (CPO) have prognostic value.

CPO derived from CO and MAP and calculated as CPO=CO*MAP/451 [W], reflects the cardiac hydraulic pumping ability and was found to be the strongest hemodynamic correlate of in-hospital mortality [13].

Risk stratification

A number of risk stratification tools have been proposed in cardiogenic shock. Some of the tools were derived from critically ill patients in a general intensive care unit. The APACHE II (Acute Physiology and Chronic Health Evaluation) score is derived from 13 variables obtained in the first 24 hours of admission to an intensive care unit. The SAPS II (Simplified Acute Physiology Score) score includes 12 physiologic variables. These scores can predict in hospital mortality. The Card Shock score was derived by European investigators and uses 7 variables, each of which individually predicts in-hospital mortality. The score ranges from 0 to 9, and is highly sensitive with an area under the curve of 0.83. The in hospital mortality risk increases with the score, and patients with a score of 9 have 100% mortality [14].

Clinical presentation

In addition to signs and symptoms of AMI, patients may present with respiratory difficulties, diaphoresis, and cold and clammy extremities. Signs of end-organ damage may present as oliguria, altered mental status, and severe dyspnea. An S3 gallup on auscultation or a dyskinetic segment of the ventricle may be felt on palpation [15].

Diagnosis

Patients with cardiogenic shock also exhibit signs and symptoms of pulmonary congestion and tissue hypoperfusion. These signs and symptoms can include dyspnea, rales, altered mental status, elevated jugular venous pressure, reduced urine output, narrow pulse pressure, cool and clammy skin, and elevated lactate levels [16]. Criteria for the diagnosis of cardiogenic shock are listed in Table 1.

Table 1: Diagnostic criteria for cardiogenic shock.
Diagnostic criteria
Adequate Filling Pressure: Pulmonary artery wedge pressure greater than 15 mm Hg.
Reduced Cardiac Index: Less than 2.2 L/min/m2 body surface área for patients receiving vasoactive or mechanical support. Less than 1.8 L/min/m2 body surface área for patients not receiving vasoactive or mechanical support
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

Although physical examination, laboratory, electrocardiographic, and echocardiographic testing remain the mainstay in the initial evaluation of a patient suspected of having cardiogenic shock, increasing emphasis on hemodynamic evaluation has the potential for earlier recognition and more appropriate management of cardiogenic shock with subsequent improvement on outcomes [17]. The severity of cardiogenic shock can range from pre-shock with clinical evidence of hypoperfusion, despite SBP > 90 mmHg, to refractory shock, in which there is ongoing hypoperfusion despite ≥ 2 vasopressors and treatment of the underlying cause. Some patients with cardiogenic shock may not have hypotension [18].

Management

Inotropes and vasopressors

The basic treatment measures include initial stabilization with volume expansion to obtain euvolaemia, vasopressors, and inotropes plus additional therapy for the prevention or treatment of multiorgan system dysfunction [19]. Norepinephrine is associated with fewer arrhythmias and may be the vasopressor of choice in many patients with cardiogenic shock [20]. Because catecholamines increase myocardial oxygen consumption and vasoconstrictors may impair microcirculation as well as tissue perfusion, their use should be restricted to the shortest possible duration and the lowest possible dose [19]. Vasopressin is another agent utilized in many centers as a second-line therapy. It is an endogenous vasopressor stored mainly in the posterior lobe of the pituitary gland and myocardium [21]. Levosimendan is a calcium sensitiser and ATP-dependent potassium-channel opener. However, some clinical observations indicate that levosimendan can improve haemodynamics in the context of cardiogenic shock after acute coronary syndromes, when combined with catecholamines, to maintain adequate perfusion pressures [22,23]. Inotropic and vasopressor agents have been recommended and used for several years in the treatment of patients in shock, but they remain controversial. Despite its beneficial effect on myocardial contractility, the side effects of inotropic therapy (arrhythmiasand increased myocardial oxygen consumption) may be associated with increased mortality [24].

Revascularization

Coronary reperfusion is the main evidence-based therapeutic intervention for patients with acute MI presenting with cardiogenic shock. Approximately 80% of patients who have cardiogenic shock present with multivessel coronary artery disease, and mortality is higher with multivessel disease than with single-vessel disease [25]. Multivessel revascularization (ie, performing PCI on culprit and nonculprit related vessels) has the theoretic benefit of restoring blood flow to ischemic territories [26]. The ACC/AHA/SCAI guidelines recommend that in patients presenting with STEMI complicated by cardiogenic shock, emergency revascularization with either PCI or CABG irrespective of the time delay from myocardial infarction onset [27].

Mechanical Circulatory Support

Traditionally, intra-aortic balloon pumps have been the main support system. Aortic balloon counterpulsation during diastole augments diastolic coronary perfusion and balloon deflation during systole reduces afterload and may improve haemodynamic parameters [28]. Veno-arterial extracorporeal membrane oxygenation bypasses both the right and left heart and can fully replace cardiorespiratory function even with no intrinsic cardiac function in cardiac arrest or previously considered non-supportable ischaemic cardiogenic shock, allowing time for cardiac recovery or at least a potential bridge to implantation of permanent ventricular assist devices or heart transplantation [28,29]. Despite successful revascularization, mortality in patients with cardiogenic shock remains very high. There has been interest in improved mechanical support devices. These may make revascularization safer [29].

Stabilization and resuscitation strategy

If hypovolemia is present, conservative boluses of crystalloids (250-500 mL) are reasonable while the patient is being stabilized for cardiac catheterization. Oxygen goals vary depending on patient comorbidities, but in the acute care setting blood oxygen saturations of >90% are acceptable. When non-invasive forms of oxygenation and ventilation are inadequate, invasive ventilation is required. Therefore, a low tidal volume strategy is recommended when mechanically ventilating patients in CS [30]. Continuous renal replacement therapy should be considered with stage

2 kidney injury or when life-threatening changes in fluid, electrolyte, and acid-base balance precipitates the need for dialysis.

Conclusion

Cardiogenic shock is a potentially fatal complication of acute myocardial infarction and other heart diseases. This article provides general information about cardiogenic shock.

Conflicts of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that be construed as a potential conflict of interest.

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