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Editorial

Hemodynamics and Kinetics of Heart Failure with Preserved Ejection Fraction Shock

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Abstract

The classical paradigm of cardiogenic shock is severe impairment of left ventricular, right ventricular, or biventricular contractility resulting in decreased cardiac output and end-organ failure. In patients with preserved ejection fraction, cardiogenic shock results from impaired left ventricular filling leading to decreased cardiac output and end-organ hypoperfusion. Heart failure with preserved ejection fraction (HFpEF) comprises a heterogenous group of myocardial and systemic metabolic derangements. Cardiogenic shock with preserved left ventricular ejection is thought to be less common than with reduced left ventricular ejection fraction, and therapeutic approaches are not well standardized. We aim to review the pathophysiology of cardiogenic shock in HFpEF, define various etiologies that culminate in the HFpEF shock state, and present our algorithmic approach to managing these complex patients.

Keywords: cardiogenic shock, heart failure with preserved ejection fraction, HFpEF, hemodynamics, restrictive, hypertrophic, amyloidosis

Background

The classical paradigm of cardiogenic shock is severe impairment of left ventricular, right ventricular, or biventricular contractility resulting in decreased cardiac output and end-organ failure. Large acute myocardial infarction, acutely decompensated heart failure, and acute myocarditis are common causes of cardiogenic shock.[1]

In restrictive and hypertrophic cardiomyopathies, cardiogenic shock results from impaired left ventricular filling and not from impaired ventricular contractility. In turn, impaired left ventricular filling leads to decreased cardiac output and end-organ hypoperfusion.[2]

Heart failure with preserved ejection fraction (HFpEF) comprises a heterogenous group of myocardial and systemic metabolic derangements. HFpEF includes patients with typical risk factors for diastolic dysfunction such as aging, systemic hypertension, obesity, type 2 diabetes, or coronary artery disease, but also includes patients with restrictive and infiltrative cardiomyopathies, constrictive pericarditis, hypertrophic (obstructive and non-obstructive) cardiomyopathy, valvular heart disease, myocarditis, and complications of heart transplantation (rejection and cardiac allograft vasculopathy) (Figure 1). Differences in etiopathogenesis, natural history, and prognosis present challenges in epidemiologic surveillance and prognostication.[3] Cardiogenic shock with preserved left

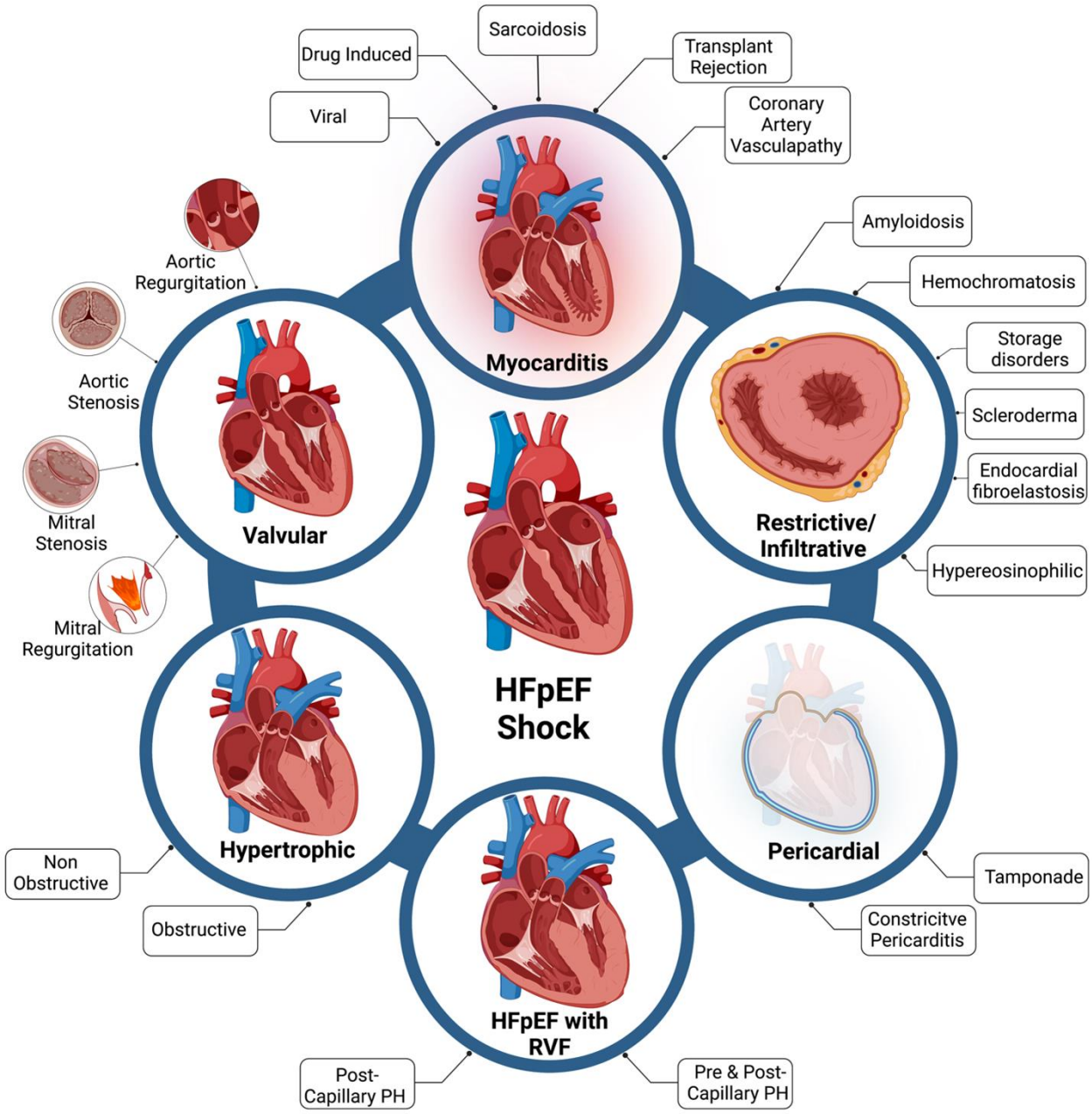

Figure 1. Various etiologies of cardiogenic shock in patients with preserved left ventricular ejection fraction.

ventricular ejection is thought to be less common than with reduced left ventricular ejection fraction, and therapeutic approaches are not well standardized. We aim to review the pathophysiology of cardiogenic shock in HFpEF, define various etiologies that culminate in the HFpEF shock state, and present our algorithmic approach to managing these complex patients.

Pathophysiology

Recognition of the clinical phenotypes of cardiogenic shock with preserved left ventricular ejection fraction has important clinical implications (Figure 1). The management and prognosis of primary myocardial pathology, like restrictive or hypertrophic cardiomyopathy, differs from that of cardiogenic shock due to the pleiotropic manifestations of advanced heart failure with preserved ejection fraction.

Restrictive and Hypertrophic Cardiomyopathies

Restrictive left ventricular or biventricular filling with reduced stroke volume may result in a low cardiac output state and end-organ hypoperfusion. Atrial arrhythmias resulting

from atrial dilation and dysfunction can impair left ventricular filling and further decrease stroke volume.[4] Excessive cardiac preload reduction due to aggressive diuresis or bradyarrhythmia may cause systemic hypoperfusion, end-organ dysfunction, and cardiogenic shock.[5] At times, various pathologies that lead to different types of HFpEF coexist and often accelerate the progression to shock. For example, 15% of patients with severe aortic stenosis and approximately 30% of patients with low-flow, low-gradient aortic stenosis have transthyretin cardiac amyloidosis.[6] Severe aortic stenosis promotes left ventricular hypertrophy that impairs left ventricular filling and, together with a fixed outflow obstruction, leads to a low cardiac output state and, thereby, cardiogenic shock. Hypertrophic cardiomyopathy with a dynamic left ventricular outflow tract obstruction and systolic anterior motion of the mitral valve may precipitously lower cardiac output and trigger cardiogenic shock presentation.[7]

Cardiomyopathy Due to Comorbidities

Aging, obesity, hypertensive heart disease, type 2 diabetes, and coronary artery disease promote left ventricular remodeling and atrial dilation. Left atrial non-compliance due to chronically elevated left ventricular filling pressure decreases pulmonary vascular compliance and causes pulmonary venous hypertension. At the same time, in other patients, this leads to an increase in pulmonary vascular resistance with the development of pre- and post-capillary pulmonary hypertension. In some instances, this results in right ventricle-pulmonary artery uncoupling, and right ventricular failure (RVF) ensues. Atrial arrhythmias due to left atrial dilation and atrial functional mitral regurgitation due to left atrial annular dilation can perpetuate the vicious cycle from pulmonary hypertension (PH) to RVF. Progression of RVF in patients with severe PH due to left-sided heart disease results in a clinical syndrome that manifests as cardiogenic shock in severe states. Furthermore, worsening renal dysfunction due to systemic venous congestion may exacerbate RVF.[8-10]

Constrictive pericarditis is a result of loss of pericardial compliance, which leads to a decoupling of the intrapericardial and intrathoracic pressure and interventricular interdependence. While this is an entirely separate entity from a classic HFpEF, the presentation is often similar to elevated filling pressures, heart failure syndrome, and the appearance of preserved ejection fraction on transthoracic echocardiogram; therefore, we thought it deserved mention in this manuscript.[2]

Similarly, acute myocarditis is distinct from typical HFpEF syndromes; however, patients can present with heart failure syndrome and overt cardiogenic shock. Acute myocarditis, acute viral infection, or other etiologies lead to an acute inflammatory response within the myocardium, increased LV wall thickness, impaired filling, and impaired contractility. Myocarditis is also often manifested by atrial and ventricular arrhythmias, which further worsen myocardial oxygen demand and ventricular filling and contractility, leading to a low cardiac output state.[11]

Various valvular pathologies may lead to cardiogenic shock, but myocardial dysfunction is often not the primary etiology of shock in these cases. Since the ejection fraction is preserved in most of these cases and clinical presentation may be similar, we mention these etiologies here but will not discuss them in detail.

Clinical Presentation

The clinical manifestations of cardiogenic shock with reduced and preserved left ventricular ejection fraction are indistinguishable. They include hypotension, decreased urine output, altered mental status, and respiratory compromise. Renal and hepatic dysfunction and lactic acidosis are laboratory evidence of hypoperfusion.[2] While most of the etiologies of HFpEF have an insidious onset of symptoms, myocarditis often presents as more of an acute presentation, differentiating it from other pathologies.

Echocardiogram and Hemodynamics

Patients with restrictive and hypertrophic cardiomyopathy have increased left ventricular thickness, small cavity size, and bi-atrial enlargement. Left ventricular outflow tract obstruction may be present at rest in patients with hypertrophic cardiomyopathy or with exacerbating maneuvers such as Valsalva, exercise, hypotension, or hypovolemia.

The hemodynamic parameters of cardiogenic shock due to restrictive or constrictive cardiomyopathy include depressed cardiac index and equalization of elevated left atrial and right atrial pressures (RAP) and elevated pulmonary artery pressures (PAP).[12] Hemodynamic parameters of cardiogenic shock due to hypertrophic cardiomyopathy are often mildly elevated RAP but with severely elevated PAP and PAWP and depressed cardiac index. Hemodynamic parameters of cardiogenic shock due to predominant right ventricular failure are elevated RAP out of proportion to left atrial pressure, elevated PAP and pulmonary vascular resistance (PVR), and depressed cardiac index.[13]

Diagnosis and Management

A bedside echocardiogram provides an initial non-invasive assessment of cardiac performance, including diastolic and

systolic ventricular function, valvular heart disease, and pericardial alterations. Insertion of a balloon tip pulmonary artery catheter allows serial measurements of right and left filling pressures and cardiac output and thereby helps guide fluid resuscitation, diuretic therapy, and initiation/titration of inotropes, pressors, or mechanical circulatory support. Right heart catheterization (RHC) is a crucial part of investigating patients with undifferentiated HFpEF shock, as it allows phenotyping of patients regarding univentricular versus biventricular shock and degree of pulmonary vascular involvement. RHC also allows real-time monitoring of response to therapy and helps guide volume management, inotropes, pressor use, pulmonary vasodilator therapy, and mechanical circulatory support.[2] When RHC cannot be rapidly performed, a central venous catheter may be used as a rudimentary tool to guide fluid management based on central venous pressure and inopressor therapy based on central venous oxygen saturation. Central venous pressure and calculated cardiac index based on central venous oxygen saturation help differentiate cardiogenic shock from distributive shock due to sepsis or hypovolemic shock.[14] However, relying solely on central venous catheters, compared to RHC, may lead to false assumptions and inadequate or frankly incorrect tailoring of therapy. Early institution of invasive mechanical ventilation can improve hypoxemia, coronary perfusion, and end-organ hypoperfusion by lowering the metabolic cost of breathing in patients with limited oxygen delivery.[15] Cardioversion for atrial arrhythmias in the setting of hemodynamic instability and atrial pacing in the setting of intact AV node or ventricular pacing in patients with complete AV block improves cardiac output by restoring atrial contribution to left ventricular filling and improves chronotropic response. Renal replacement therapy to correct acidosis and volume overload improves end-organ function and prevents pulmonary injury.[16]

Institution of temporary mechanical circulatory support in refractory cardiogenic shock hinges on the reversibility of the shock state, a life expectancy > 1 year, or a possible exit strategy such as candidacy for heart transplantation or chance for myocardial recovery. The type of mechanical circulatory support for restrictive and hypertrophic cardiomyopathies depends on several factors: local interventional or surgical expertise, vascular access site availability, and extent of left, right, or bi-ventricular contribution to the shock state. Left atrial unloading with tandem heart versus veno-arterial extracorporeal oxygenation (V-A ECMO) or bi-atrial unloading using multistage cannula left atrial V-A ECMO can provide temporary support as a bridge to recovery or heart transplantation (HT). Durable mechanical circulatory support with left ventricular assist devices is challenging in restrictive cardiomyopathies due to a small left ventricular cavity leading to suction events and right ventricular failure. Total artificial heart as a bridge to HT is an option in highly selected individuals.[17]

Right ventricular failure is a therapeutic challenge in heart failure with preserved ejection fraction. Management includes inotropic support and, in select cases, V-A ECMO as a bridge to recovery when reversible causes are identified. Pulmonary vasodilators or isolated right ventricular assist device support may worsen pulmonary edema or cause pulmonary hemorrhage due to high PAP, PAWP, and impaired left ventricular diastolic function.

Conclusions

The first step in managing cardiogenic shock with preserved ejection fraction is to recognize the underlying disorder: hypertrophic or infiltrative cardiomyopathy, valvular or pericardial disease or end-stage heart failure with preserved ejection fraction, right ventricular failure and pulmonary hypertension, or acute myocarditis. Echocardiographic and invasive hemodynamic assessment should guide the management of patients in cardiogenic shock with preserved ejection fraction. A multidisciplinary team-based approach for cardiogenic shock management allows rapid triage and a timely escalation of support for appropriate candidates.

Disclosures

The authors report no relevant disclosures.

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