Mechanical Circulatory Support for High-Risk Pulmonary Embolism

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KEYWORDS

- Mechanical circulatory support • Right ventricular failure • Cardiogenic shock
- Pulmonary embolism

KEY POINTS

- Temporary mechanical circulatory support (MCS) devices have a role in treating high-risk patients with pulmonary embolism (PE) with cardiogenic shock.
- Mechanical circulatory device selection should be made based on center experience and device-specific features.
- All current devices are effective in decreasing right atrial pressure and providing circulatory support of 4 to 5 L/min.
- The pulmonary artery pulsatility index may prove to be an unreliable method to assess right ventricular function in high-risk patients with PE with cardiogenic shock.
- Careful clinical evaluation on an individual patient basis should determine the need for MCS.

Venous thromboembolism (VTE) is responsible for the hospitalization of more than 250,000 Americans annually and represents a significant risk for morbidity and mortality. The incidence of pulmonary embolism (PE) is approximately 60 to 70 per 100,000 people.[1] Mortality rates for patients with acute PE may exceed 15% within the first 3 months of presentation.[2]

Acute high-risk (also called "massive") PE is an important cause of acute right ventricular (RV) failure that predisposes to cardiogenic shock and sudden cardiac death. Guidelines for treating acute PE have been published and include treatment guidelines for patients with high-risk PE and cardiogenic shock.[3] However, the evidence base for management of these patients is poor and there is little consensus regarding the appropriate management for treatment of acute RV failure–induced cardiogenic shock due to PE.

High-risk PE is defined as PE that causes sudden cardiac death or cardiogenic shock (CS) with an associated systolic blood pressure (BP) lower than 90 mm Hg or the need for vasopressor support. In its most severe form, high-risk PE can present as continuous hypotension, profound bradycardia, or pulselessness. Intermediate-risk (also termed "submassive") PE is characterized by evidence of RV dysfunction or myocardial necrosis in the presence of systemic normotension. Other characteristics of intermediate-risk PE are RV strain and the elevation of cardiac markers.[4]

Evaluation of RV function is complex and requires a multimodality approach, including clinical assessment, laboratory studies such as

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brain natriuretic peptide and troponin, echocardiography, computed tomography/MRI, clinical risk scores, and right heart catheterization with hemodynamic measurements.[5,6]

MANAGEMENT OF PULMONARY EMBOLISM

Unfractionated heparin, fondaparinux, and low-molecular-weight heparin, acting as a bridge to vitamin K antagonists or direct oral anticoagulants, are the mainstays of treatment for patients without hemodynamic compromise. Unstable patients may require escalation of treatment over and above isolated anticoagulation therapy. Risk stratification is critical to guide effective PE treatment in the acute phase. Clinical findings at presentation may facilitate assessment of disease and treatment prognosis. Identifying the presence and severity of RV dysfunction from acute pressure overload represents one of the most important factors in an effective PE risk stratification protocol. Early fully therapeutic anticoagulation is the most important therapy in low-risk and intermediate-risk patients with PE to prevent progression to high-risk status.

PULMONARY EMBOLISM SEVERITY INDEX, SIMPLIFIED PULMONARY EMBOLISM SEVERITY INDEX, AND PULMONARY ARTERY PULSATILITY INDEX

Severity indices to predict PE 30-day mortality risk have been established and are used as part of the patient workup and decision making. PE severity index (PESI) is clinically based and represents the most extensively validated score to date.[7] This score accounts for the severity of PE as well as the patient's existing comorbidities. The PESI score uses 11 clinical criteria, including history of heart failure, systolic BP, heart rate, and O2 saturation, to predict 30-day outcomes of patients with PE.

The simplified PESI (sPESI) score, which uses 8 clinical criteria, also has been validated.[8] Risk stratification via circulating biochemical markers, such as natriuretic peptides and cardiac troponin I or T levels, also may be helpful in determining risk for normotensive patients with PE.[9] Combination of the sPESI with troponin testing provides additional prognostic information.[10] The pulmonary artery pulsatility index (PAPI) is another well-established index used to detect RV failure in myocardial infarction and to predict right-sided heart failure after left ventricular assist device (LVAD) implantation.[11] The PAPI is calculated as pulmonary pulse pressure divided by right arterial (RA) pressure.

One of the greater challenges in treating patients with chronically or acutely compromised RV function is to determine and quantify right heart function and predict outcome. The rationale of the PAPI hemodynamic index is to assess RV function by indexing RV systolic function (pulse pressure) to RV preload (mean RA pressure). Although it has been used to predict outcomes in patients with LVAD, there are no data to support the use of PAPI in patients who suffer acute high-risk and intermediate-risk PE. Unlike in chronic RV failure, as is the case in LVAD candidate patients and in acute RV infarction, in acute PE, the RV myocardium is not directly affected. The increase in cardiac biomarkers represents an increase in afterload and RV strain with minimal damage to the myofibrils themselves.[12,13] Thus, the RV may be more capable of generating enough contractility to generate higher pulse pressure compared with patients with chronic RV failure or acute RV infarction.

PAPI is inversely dependent on preload. In the acute PE setting, preload may remain relatively low or mildly increase unless rapid volume expansion therapy is administered. Moreover, even with volume expansion, the amount of increase in preload is difficult to predict, as it depends on multiple, inaccessible factors such as RA/RV compliance and rigidity, the effect of ventricular interdependence, intravascular volume status, and more.[14] Last, in our experience, PAPI is unreliable in patients with Impella RP (discussed subsequently; Abiomed Inc., Danvers, MA) due to altered PA wave forms, which can result in inaccurate PA systolic and diastolic measurements. In a small series of 4 patients from Detroit Medical Center with high-risk PE treated with Impella RP there was poor correlation between PAPI and the patients' clinical status (Table 1, Elder M, unpublished data, 2017).

MANAGEMENT OF RIGHT VENTRICULAR FAILURE

The ability of the RV to immediately adapt to the acute increase in afterload associated with high-risk PE is limited, because the nonpreconditioned, thin-walled RV is usually unable to generate a mean pulmonary artery pressure higher than 40 mm Hg. An acute increase of RV afterload generates RV strain and dilation which, per the Starling law, generates more contractility until the dilatation is extreme and contractility falls.[15]

Current guidelines for acute RV failure management include saline infusion as the first step, with an emphasis to avoid excessive