Perspective

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Hemodynamic Stress and Acute Heart Failure in Valvular Heart Disease

Stefan Brenner*

Department of Cardiovascular Science, University of Gottingen, Gottingen, Germany

***Corresponding author:** Stefan Brenner, Department of Cardiovascular Science, University of Gottingen, Gottingen, Germany; Email: brennerS@gmail.com

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Introduction

Acute Heart Failure (AHF) represents a spectrum of conditions resulting from the interaction of an acute precipitant with cardiac and patient comorbidities. AHF may result from multiple precipitants that add to acute hemodynamic stress leading to chronic valvular injury or may occur following significant new valvular injury. In addition, there are no rigorous randomized controlled trials of Valvular Heart Disease (VHD) and AHF, with most data coming from observational studies. Therefore, unlike chronic conditions, current guidelines are unclear as to when patients with severe VHD present with AHF. Valve damage typically develops gradually over time, but clinical manifestations or exacerbations during hemodynamic stress from a superimposed precipitant may vary in severity and interact in a complementary manner.

Description

In addition, changes in the underlying cardiovascular system during the progression of heart failure can lead to ventricular dysfunction, especially Mitral Regurgitation (MR) and Tricuspid Regurgitation (TR), and the coexistence of these two conditions can complicate the clinical picture. Unlike organic regurgitation, in which the valve apparatus is structurally normal, secondary MR and RT develop through structural changes in the shape of the ventricles or atria. Secondary atrioventricular regurgitation can occur at any level of Left Ventricular Ejection Fraction (LVEF) and is a dynamic lesion, with varying severity depending on the load. Acute ventricular failure may be the cause of acute heart failure in cases of new acute valvular dysfunction or pre-existing moderate ventricular failure exacerbated by endocarditis, prosthetic valve thrombosis, etc. Furthermore, the hemodynamic impact of even moderate VHD may be exacerbated by the coexistence of various predisposing factors such as acute coronary syndrome, hypertension, arrhythmias, fluid overload, or renal dysfunction. In left ventricular heart disease, left ventricular pressure and/or volume overload result in left atrial pressure overload and passive pulmonary venous hypertension. This sudden increase in pulmonary venous pressure leads to rupture of the alveolar capillary membrane resulting in acute reversible alveolar edema. Pulmonary Hypertension (PH) results in right ventricular pressure overload resulting in right ventricular hypertrophy and dilatation and thus valve annulus dilatation with secondary TR leading to progressive deterioration of right ventricular function. Pulmonary hypertension due to left heart disease, including ischemic heart disease, represents group 2 PH defined by a mean pulmonary artery pressure of 20 mmHg and a pulmonary capillary wedge pressure of 15 mmHg. In the hemodynamic setting of postcapillary HP, isolated HP is defined by a Pulmonary Vascular Resistance (PVR) of 2 Wood Units (WU) and combined HP by a PVR of 2 WU. Severe HP is defined as >5 WU. In patients with VHD undergoing intervention, increased PVR, especially if >5 WU, is associated with increased disease burden and a worse prognosis. Furthermore, PH recovery after correction of VHD is often incomplete and persistent PH is associated with adverse outcomes. The prevalence of PH increases with the severity of left-sided VHD and severity of symptoms. In Mitral Stenosis (MS), PH is associated with symptom severity and valve area and is associated with long-term prognosis. The prevalence of PH on primary MRI may vary depending on clinical severity and may be as high as 64% in New York Heart Association (NYHA) class III/IV patients. Early surgical treatment is recommended in these patients because pre-existing PH is associated with reduced postoperative left ventricular systolic function and nearly doubles postoperative mortality. Acute HF in the setting of VHD poses several diagnostic challenges, including difficulty in assessing the severity of VHD due to rapid changes in loading conditions and the intervening acute precipitants and associated comorbidities, making it difficult to determine whether VHD is the sole contributor to the patient's clinical deterioration.

Conclusion

Furthermore, therapeutic interventions in patients with VHD and AHF are not based on rigorous evidence as there are no randomized controlled trials in this setting and, even more so, patients with severe VHD are often excluded from randomized trials of AHF. Therefore, a clear strategy regarding the timing of intervention or the type of cannot yet be defined. However, because AHF patients with VHD, especially those with multiple organ dysfunction or severe comorbidities, may be at very high or very high surgical risk, percutaneous strategies should be integrated into the treatment spectrum, at the discretion of the cardiology team to determine the best treatment option for each individual case.