Acute Valvular Heart Disease

Varun Maheshwari, MD, Brian Barr, MD, Mukta Srivastava, MD, FSCAI*

KEYWORDS

• Aortic regurgitation • Aortic stenosis • Mitral stenosis • Mitral regurgitation
• Prosthetic valve dysfunction

KEY POINTS

- A targeted history, physical examination, and basic initial workup can provide early recognition of decompensated valvular heart disease.
- Echocardiography is diagnostic in acute valvular disease in determining etiology and defining severity.
- Early hemodynamic assessment in valvular disease is key to appropriate initiation of medical therapy, which focuses on improving hemodynamics, peripheral perfusion, and relieving vascular congestion.
- Mechanical circulatory support has a role in the treatment of decompensated valvular disease.
- Prosthetic valve dysfunction can cause acute obstruction, regurgitation, and hemolytic anemia; these conditions should be easily recognizable and well-understood.

INTRODUCTION

Valvular heart disease (VHD) is a common phenomenon in the developed world, affecting a large proportion of adults to varying degrees of severity.[1,2] Although the majority of patients have stable valvular disease, with the increasing prevalence in the population, decompensated illness as a result of valvular disease is increasingly recognized. Owing to improved outcomes as the result of cardiac surgery and interventional therapies, the prognosis for VHD has improved over the past several decades. We aim to delineate the initial diagnosis and management of these patients and provide a context for clinicians to provide evidence-based, effective treatment modalities in acutely decompensated VHD.

INITIAL EVALUATION

Medical stabilization of decompensated VHD requires prompt recognition. Early evaluation of hemodynamics and the recognition of an initial appropriate evaluation including a targeted physical examination and history is important. Hypotension, pulmonary congestion, poor peripheral perfusion, altered mentation, and oliguria are signs concerning for cardiogenic shock and heart failure, which may be the result of acutely decompensated VHD. Assessment of respiratory status and anticipating a need for airway management with either intubation or noninvasive positive pressure ventilation is important. A history of intravenous drug use, ischemia, blunt cardiac injury, known VHD, or congenital valvular disease may provide clues regarding etiology. Cardiac auscultation may be helpful; murmurs can provide, in some cases, a clue to the severity of the disease process.

Several noninvasive tests can be undertaken while the patient is being stabilized. Laboratory work, including cardiac troponin and brain natriuretic peptide can provide data regarding volume status and evidence of myocardial ischemia. Chest radiography will confirm pulmonary

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Department of Medicine, Division of Cardiology, University of Maryland School of Medicine, 110 South Paca Street, 7th Floor, Baltimore, MD 21201, USA
* Corresponding author. 110 South Paca Street, 7N -118, Baltimore, MD 21201.
E-mail address: msrivast@som.umaryland.edu

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congestion and electrocardiography (ECG) will allow for the evaluation of myocardial ischemia. Early echocardiography can evaluate the presence and severity of disease. For each valvular pathology, further therapy should be tailored as discussed in this article.

AORTIC REGURGITATION

Etiology and Pathophysiology

Acute aortic regurgitation (AR) is among the most dangerous acute valvular pathologies and can result in rapid clinical deterioration. Although AR can have several distinct etiologies (**Table 1**), acute AR is commonly the result of trauma, infective endocarditis, or thoracic aortic dissection extending retrograde toward the aorta.[3,4] Infective endocarditis can cause leaflet perforation and perivalvular abscesses resulting in acute AR. Aortic dissection can cause acute AR, either through direct retrograde extension into the leaflet or through dilation of the sinuses of Valsalva.[5] Direct blunt trauma to the chest may can also cause traumatic leaflet rupture.[5,6]

In chronic severe AR, the left ventricle (LV) has dilated and become compliant to accommodate excess LV end-systolic volume and can maintain a forward stroke volume (SV). However, in acute severe AR, the LV has not adapted to the large regurgitant volume and cannot generate an appropriate forward SV.[7,8] Ultimately, LV end-diastolic pressure exceeds that of the left atrium (LA), resulting in premature closure of the mitral valve during diastole and decreased LV filling, further decreasing SV and increasing left atrial pressure, leading to increased pulmonary pressures, low cardiac output, hypotension, and increased systemic vascular resistance.[7,8]

Clinical Presentation

Severe acute AR is often dramatic in its presentation. Owing to the acute decrease in cardiac output and high LV filling pressures, cardiovascular collapse and acute pulmonary edema are often seen. Preceding fever may be present in patients with endocarditis. Examination findings of profound cardiogenic shock—hypotension, pallor, cool extremities, altered mentation, and pulmonary edema—are common. In contrast, examination findings in chronic severe AR reflect, LV adaptation to the high regurgitant volume and increased SV causes various classic signs of higher output with widened pulse pressure (Duroziez's murmur, waterhammer pulse, widened pulse pressure). In acute severe AR, the pulse pressure will either be normal or decreased owing to rapid equalization of the aortic and LV pressures.[7] Premature mitral valve closure and LV volume overload may cause a soft S1 and S3, respectively. A low-pitched early diastolic murmur can be heard, but the presence of the murmur is not a reliable marker of severity owing to rapid equalization of pressures between the LV and aorta, causing a minimal gradient in diastole and thus minimal murmur. ECG findings usually reflect the underlying disorder, such as acute ST-elevation myocardial infarction (STEMI) from retrograde dissection of a coronary artery, as may occur in acute aortic dissection, or are nonspecific. Chest radiography may show pulmonary edema and pulmonary vascular prominence, along with cardiomegaly or widened mediastinum in patients with aortic dissection.

Diagnosis

Echocardiography is the diagnostic modality of choice in severe AR. The markers of severity include (1) a vena contracta (narrowest neck of the AR jet) width of greater than 6 mm, (2) more than 65% of the LVOT width occupied by the color jet, and (3) holodiastolic flow reversal within the abdominal aorta (indicating flow back through the aortic valve during diastole).[9] Doppler imaging may reveal a dense continuous wave signal with steep diastolic slope of AR velocity indicating rapid equalization of pressures. Premature closure of the mitral valve, suggested with a very short mitral valve deceleration time (<150 ms) may indirectly indicate the severity of the AR jet[9] (**Fig. 1**). Additionally, echocardiography can evaluate LV dysfunction and reveal valvular vegetations. Transesophageal echocardiography (TEE) or computed tomography angiography will reveal the presence of aortic dissection.[9]

Treatment

Acute AR with hemodynamic collapse is a surgical emergency. Stabilization of the patient should be focused on afterload reduction and supporting the volume overloaded ventricle in preparation for surgery. Intravenous vasodilators, such as nitroprusside in normotensive or hypertensive patients and inotropic agents in hypotensive patients to augment SV, are first-line therapies.[10] In general, although beta-blockers are the first line in treatment of patients with acute aortic dissection, they should be avoided in acute AR, because it can decrease SV.[10]

Mechanical circulatory support, including intra-aortic balloon counterpulsation, venoarterial extracorporeal membranous oxygen, or percutaneous ventricular assist devices such as the Impella (AbioMed, Danvers, MA), are contraindicated.[8,9,11]