Valvular Shock in the Cardiac Intensive Care Unit: Perioperative Care for Transcatheter Valve Interventions

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KEYWORDS

• Cardiogenic shock • Valvular heart disease • Transcatheter intervention • Aortic stenosis
• Aortic regurgitation • Mitral regurgitation • Mitral stenosis

KEY POINTS

- Valvular heart disease is a less common but increasingly recognized cause of cardiogenic shock and is associated with high mortality yet remains underrepresented in randomized trials and guidelines.
- Diagnosis is challenging in critically ill patients; early comprehensive echocardiography and invasive hemodynamic assessment are essential to identify the responsible lesion and its acuity.
- Management of valvular cardiogenic shock is highly lesion-specific, and standard shock therapies may be ineffective or harmful depending on valve pathology.
- Mechanical circulatory support can be lifesaving but must be carefully selected based on valve lesion, ventricular loading conditions, and procedural feasibility.
- Definitive treatment requires timely surgical or transcatheter valve intervention, with percutaneous strategies preferred when possible, due to prohibitive surgical risk.

INTRODUCTION

Cardiogenic shock (CS) is a state of circulatory failure where ineffective cardiac output due to cardiac dysfunction leads to tissue hypoperfusion, typically associated with hypotension. CS is phenotypically heterogenous with associated high in-hospital mortality. While the majority of CS is from ventricular dysfunction from acute myocardial infarction and decompensated cardiomyopathy, the incidence of valvular heart disease (VHD) associated CS is growing[1] and is independently associated with increased in-hospital mortality.[2] Randomized clinical trials in patients with CS have focused predominantly on the myocardial infarction population and have excluded patients with severe VHD.[3–7] Patients with valvular CS have been inadequately studied resulting in a lack of high-quality evidence guiding the management of patients with valvular CS. Accordingly, societal guidelines have been unable to adequately address treatment options for these critically ill patients.[8,9] With the development of transcatheter valve therapies and mechanical circulatory support (MCS), significant potential exists to alter the natural history in patients with valvular CS.

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Abbreviations

AR	aortic regurgitation
AS	aortic stenosis
BAV	balloon aortic valvuloplasty
CS	cardiogenic shock
CT	computed tomography
ECMO	extracorporeal membrane oxygenation
IABP	intra-aortic balloon pump
LAVA	left-atrial veno-arterial
LV	left ventricle
LVOTO	left ventricular outflow tract obstruction
MCS	mechanical circulatory support
MR	mitral regurgitation
MS	mitral stenosis
PBMV	percutaneous balloon mitral valvuloplasty
PPV	positive pressure ventilation
RV	right ventricle
TAVR	transcatheter aortic valve replacement
TEE	transesophageal echocardiography
TEER	transcatheter edge-to-edge repair
TMVR	transcatheter mitral valve replacement
TTE	transthoracic echocardiogram
VHD	valvular heart disease

In this article, we will discuss the etiology and management of valvular CS, the initial medical stabilization, MCS considerations, and percutaneous interventions. Additionally, we will review potential complications of common percutaneous transcatheter interventions that are relevant for the critical care practitioner.

MECHANISMS OF CARDIOGENIC SHOCK FROM VALVULAR HEART DISEASE

Epidemiology

According to the Global Burden of Valvular Heart Disease in 2021, over 6 million American adults live with VHD (1117 per 100,000 people).[10] Moderate or severe VHD has been detected in 6.4% of asymptomatic people aged over 65 years.[11] Significant VHD is present in 14% to 39% of patients admitted to contemporary cardiac intensive care units with CS and the direct cause of CS in 4% to 8%.[12–14] Importantly, the presence of significant VHD in patients with CS is associated with increased 30 day all-cause mortality.[13] Valvular CS is predominantly due to left-sided valve disease and can occur because of a de novo acute valvular lesion, from the progressive worsening of a chronic valvular lesion or (most commonly) an intercurrent medical illness (eg, arrhythmias or infection) that decompensates a chronic valvular lesion. Native valve dysfunction has been causative in 71% of valvular CS cases and valvular CS is due to aortic valve disease in 64% of cases.[14] Right-sided valve lesions, even when acute, result in volume overload (as opposed to pressure overload) of the right ventricle (RV) which is better tolerated hemodynamically and generally presents with right-sided heart failure rather than overt CS.

Etiologies of Acute Valvular Lesions

Acute valvular dysfunction on an unconditioned ventricle often results in a dramatic presentation of acute CS due to limited physiologic reserve. CS from acute native valve dysfunction occurs predominantly due to a regurgitant lesion, while CS from prosthetic valve dysfunction can be due to either a regurgitant or stenotic lesions. In patients with acute prosthetic valve dysfunction, endocarditis should always be considered especially in the setting of early valvular degeneration or paravalvular dehiscence. While structural deterioration of bioprosthetic valves is often a slow process, in certain populations, unexpected and rapid structural valve deterioration has been noted.[14,15] Lastly, patients with CS who have a mechanical valve should always be considered to have acute valve thrombosis. Although patients with highly thrombotic Starr-Edwards, Lillehei-Cutter, or Bjork-Shiley are rarely encountered, subtherapeutic anticoagulation from medication noncompliance is frequently the cause for modern bileaflet tilting disc valve thrombosis.[16]

Evaluation and Diagnosis

Patient's history and physical examination are often limited in critical care settings. Patients in extremis, those with cerebral hypoperfusion, or those receiving positive pressure ventilation (PPV) may be unable to provide a history. Physical examination may miss quiet murmurs as a result of low cardiac output, systemic hypotension, and rapid pressure equalization when regurgitation is severe.[17] Therefore, objective measures to identify the presence and etiology of valvular pathology are required.

Chest radiographs can rapidly identify the presence and location of prosthetic valves and presence of sternotomy wires also suggest prior cardiac surgery. While point-of-care ultrasound often provides a preliminary survey, a comprehensive transthoracic echocardiogram (TTE) is often needed to accurately diagnose the presence and severity VHD.[17] When TTE is inadequate to quantify valvular dysfunction (especially in the case of prosthetic valves), transesophageal echocardiography (TEE) should be considered. Echocardiography can also assist in determining the chronicity of a valvular lesion based on presence or absence of compensatory ventricular remodeling.

Invasive hemodynamic assessment with a pulmonary artery catheter can be invaluable to guide effective management.[18,19] The pulmonary artery catheter can help to identify the etiology, such as in mitral regurgitation (MR) where giant V waves are seen on pulmonary capillary wedge pressure waveforms while in mitral stenosis (MS) giant A-waves are seen in sinus rhythm. Early coronary angiography may also be warranted in the initial evaluation for CS to rule out acute coronary artery pathology.

One critically important clinical decision is to determine whether the valvular lesion is the primary driver of CS or rather an incidental or complicating factor. This takes careful consideration of other triggers (such as a superimposed systemic illness or arrhythmia) which should be promptly treated to resolve the patient's CS. Atrial fibrillation is a common cause of decompensation of a previously stable valve lesion that often dramatically worsens hemodynamics via loss of atrioventricular coupling, prompting a rhythm control strategy.[20] The clinician should always aim to identify and treat these potential triggers. However, if no triggers are identified, or resolution of all potential triggers have not reversed the CS, then correction of the valvular lesion should be targeted. Greater lesion severity and signs of acute worsening of the lesion tend to indicate that the valve lesion is the primary problem.

MANAGEMENT OF VALVULAR CARDIOGENIC SHOCK

General Principles

The standard medical therapies for CS consisting of diuretics, inotropes, and vasopressors may be harmful to some patients with a valvular CS (**Fig. 1**). Specifically, the tachycardia from inotropes or increased afterload from vasopressors can paradoxically worsen hemodynamics in specific valvular pathologies. Medical therapy is often ineffective and primarily serves as a bridge to definitive valvular intervention. In patients who do not rapidly respond to initial medical therapy, escalation to MCS is often needed, but not all valvular lesions respond favorably to MCS devices. Like medical therapy, the goal of MCS is to preserve end-organ function allowing for definitive treatment of the lesion.

While controversial in myocardial infarction-related CS,[4] the intra-aortic balloon pump (IABP) can be highly effective in select valvular lesions.[21] Similarly, microaxial flow pumps such as the Impella (Abiomed, Danvers, MA) and extracorporeal membrane oxygenation (ECMO) have a role in valvular CS but are also lesion specific. Given the complex decision-making and need for both surgical and percutaneous intervention expertise, referral to a comprehensive valve center with the capability of performing advanced MCS should be considered early.[22] Emergency cardiac surgery in patients with CS is associated with a high risk of morbidity and mortality, and so whenever feasible, a percutaneous strategy is preferred.[23]

PPV via noninvasive ventilation or endotracheal intubation is often unavoidable in these critically ill patients who may have severe pulmonary edema. PPV reduces left ventricular preload and afterload, which may be either beneficial or detrimental depending on the valvular lesion, presence of associated conditions such as pulmonary hypertension and the baseline ventricular loading conditions.[24]

Prosthetic valve dysfunction from valve thrombosis is commonly encountered in patients with mechanical valves with subtherapeutic anticoagulation. In hemodynamically stable patients, a trial of aggressive antithrombotic therapy with high-intensity heparin is preferable and often effective.[25] For patients with hemodynamic instability or CS, especially secondary to mechanical valve thrombosis, low-dose systemic thrombolytic therapy, transcatheter leaflet release, or surgical valve replacement can be considered.[9] If surgery is indicated, replacement with a bioprosthetic or mechanical valve should be carefully considered based on patient factors.

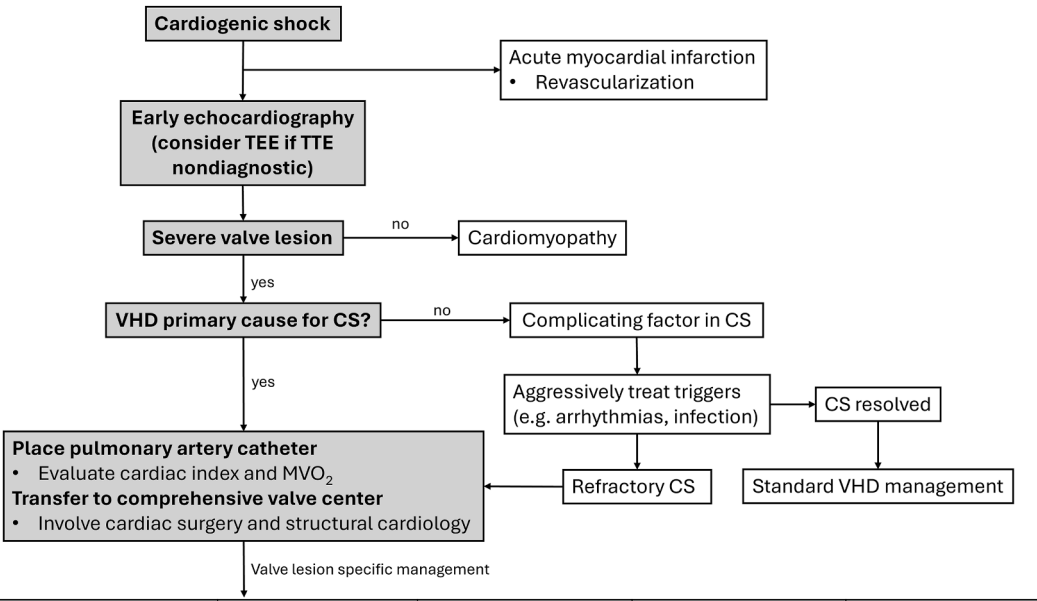
In all forms of valvular CS, the goal of the critical care cardiologist is to provide stabilization of hemodynamics, preserve end-organ function, and to facilitate the necessary investigations that can lead to a surgical or transcatheter fix of the primary valve problem.

Aortic Stenosis

Aortic stenosis (AS) is a common progressive VHD with an increasing prevalence with age (**Fig. 2**).[26,27] It develops commonly from degeneration of a trileaflet or congenitally bicuspid aortic valve but may also be seen secondary to rheumatic heart disease, prosthetic valve degeneration, or prosthetic valve thrombosis.[28] Most patients with CS from AS have impaired left ventricle (LV) function and often experienced an acute trigger for decompensation such as atrial fibrillation.

Diagnosis

Echocardiography is the gold standard in identifying severe AS.[29] Making the diagnosis of severe AS in a decompensated patient in a low-flow state can be difficult, as standard Doppler criteria require adequate stroke volume to generate



	Aortic Stenosis	Aortic Regurgitation	Mitral Regurgitation	Mitral Stenosis
Ideal heart rate	Normal	High	Normal/high	Low
(+) or (–) inotrope	(+) Inotrope (dobutamine or milrinone)	(+) Inotrope (dobutamine or milrinone)	(+) Inotrope (dobutamine or milrinone)	(-) Inotrope (β blocker, CCB, digoxin)
Preferred vasopressor	Norepinephrine	Epinephrine or dopamine	Norepinephrine	Phenylephrine or vasopressin
Vasodilators	Caution	Beneficial	Beneficial	Harmful
Positive pressure ventilation	Effective	Effective	Effective	Caution
IABP	Effective	Contraindicated	Effective	Ineffective
Impella	Effective	Ineffective	Effective (caution if flail or endocarditis)	Ineffective
ECMO	Effective	Contraindicated	Caution (may worsen pulmonary edema)	Effective (LAVA-ECMO or TandemHeart preferred)
Definitive Treatment	• TAVR • SAVR	• TAVR • SAVR • Surgical AV repair	• M-TEER • TMVR • Surgical MV repair or replacement	• PMBV • TMVR • Surgical MV repair or replacement

Fig. 1. General approach to cardiogenic shock patients with valvular heart disease.

elevated gradients.[30] Dobutamine stress echocardiography is rarely possible in critically ill patients to confirm severe AS, as severely decompensated ventricles may not generate adequate contractile reserve.[31]

Management

Severe AS results in fixed outlet obstruction that causes a marked increase in LV afterload, with resultant diastolic dysfunction from LV hypertrophy necessitating elevated filling pressures to maintain cardiac output. In hypotensive patients without evidence of pulmonary congestion, gentle fluid administration may be necessary to increase preload. In normotensive or hypertensive patients with AS and congestion, cautious use of vasodilators and diuretics can safely improve congestion. Slow titration and judicious use of short acting diuretics and vasodilators are preferred to avoid rapid or irreversible fluctuations in preload and afterload. Our choice is a short-acting balanced vasodilator like nitroprusside, which can reduce LV filling pressures and increase cardiac output in CS.[32] Norepinephrine is our preferred vasopressor of choice because its mild inotropic effects can help support left ventricular function while phenylephrine can be helpful to oppose the vasodilatory effects of anesthesia during intubation.[33]

In patients with AS refractory to medical interventions, IABP therapy can help reduce systemic vascular resistance and increase coronary perfusion pressure.[34] However, it provides only marginal augmentation of cardiac output and may

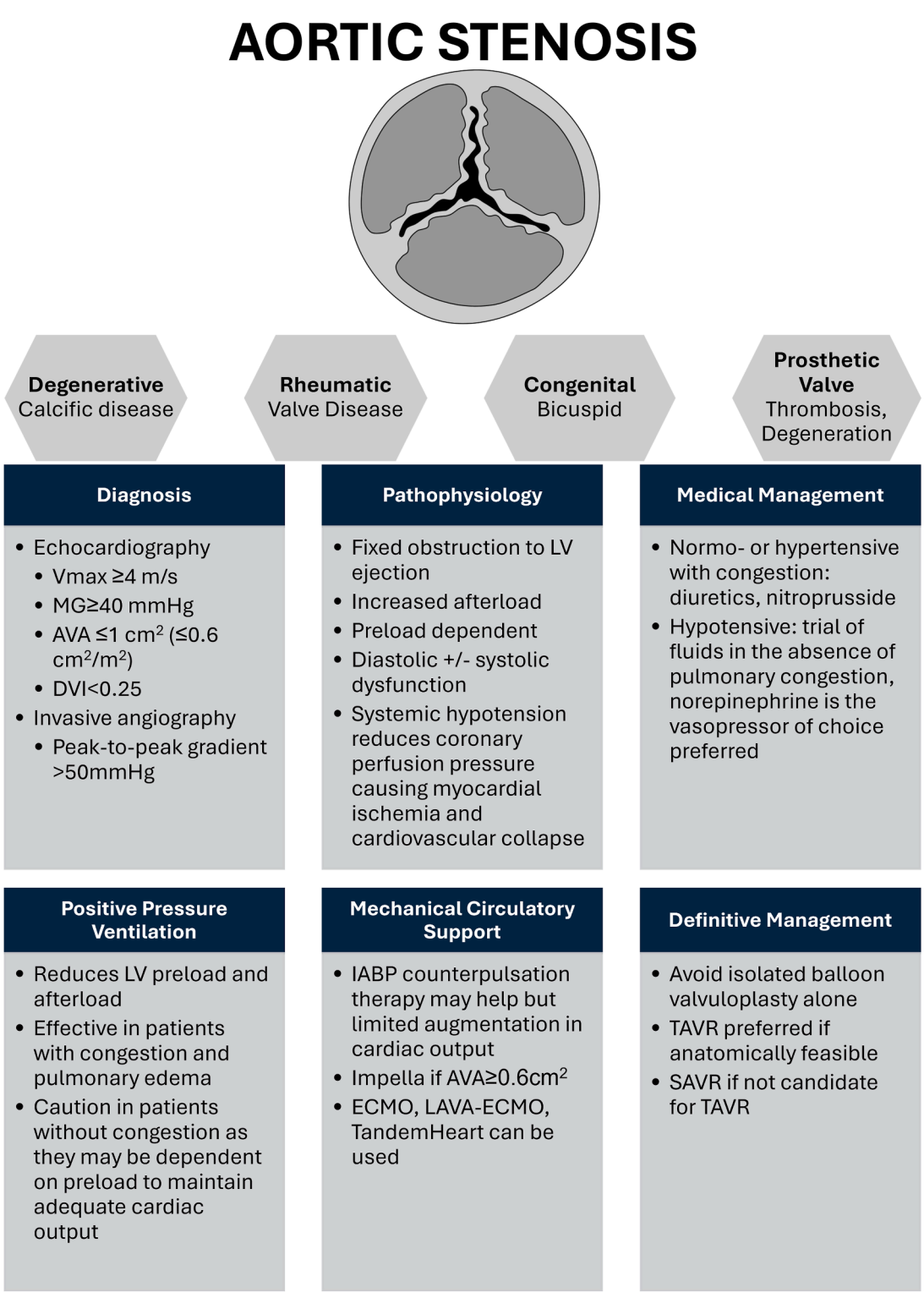


Fig. 2. Key points in the management of aortic stenosis related-cardiogenic shock.

not adequately support a critically ill patient with tenuous hemodynamics. While Impella can provide greater augmentation in cardiac placement can be challenging as it requires crossing the stenotic AV and generally is not recommended if the aortic valve area is below 0.6 cm^2.[35] Other therapies such as TandemHeart (Pittsburgh, PA), left-atrial veno-arterial (LAVA) ECMO or peripheral ECMO can be used as a bridge to definitive therapy.[36,37]

After hemodynamic stabilization with medical therapy or MCS, the definitive management involves aortic valve replacement. Surgery in this population is often associated with prohibitive operative risk and poor short-term outcomes.[38,39] Transcatheter aortic valve replacement (TAVR) is now the preferred strategy for intervention but still carries a high 30 day mortality rate of 33.3% in patients with severe AS complicated by CS.[38,40] Balloon aortic valvuloplasty (BAV) can produce modest improvements in the hemodynamic impact of AS, but in patients who do not go on to have a definitive aortic valve replacement, 90 day mortality is approximately 50%, without a clear advantage in terms of complication rate compared with TAVR.[41,42] When compared to BAV, TAVR is associated with improved hemodynamics, lower 30 day mortality, and decreased need for subsequent procedures without an increased risk of adverse events.[43]

Aortic Regurgitation

Aortic regurgitation (AR) is a less common cause for CS (**Fig. 3**).[14] Patients with chronic AR develop LV dilation, which preserves stroke volume without increasing LV end diastolic pressure but eventually the LV ejection fraction drops and LV end diastolic pressure increases resulting in heart failure.[44] In these patients, CS may occur from bradyarrhythmias or initiation of negative chronotropic agents such as beta blockers, which prolong diastolic filling time causing the LV end diastolic pressure to further increase. More commonly, CS from AR is due to high-risk acute etiology such as endocarditis, aortic dissection, or prosthetic valve failure with an unprepared ventricle causing a rapid increase in LVEDP and decreased forward flow.[44]

Diagnosis

The typical peripheral manifestations of severe AR are often absent in patients with CS. The classic murmur, wide pulse pressure, and exaggerated arterial pulsations seen in chronic AR are often replaced by a narrow pulse pressure, pulmonary edema, and a faint early diastolic murmur. TEE is often required to quantify the severity of AR and determine a causative etiology.[45]

Management

Medical therapy in CS from AR is to treat pulmonary congestion and reduce LV afterload to augment cardiac output. Higher heart rate and lower blood pressure are beneficial, typically targeting the lowest mean arterial pressure that provides adequate organ perfusion. In patients without hypotension, vasodilators such as nitroprusside, or calcium channel blockers help reduce LV afterload to improve forward flow. If despite vasodilator therapy forward flow remains impaired, inodilators such as milrinone or dobutamine are useful.[46] In hypotensive patients requiring vasopressors, pure vasoconstrictors such as phenylephrine or vasopressin should be avoided, and instead drugs with strong chronotropic and inotropic effects such as epinephrine or dopamine should be considered. Patients with AR who remain bradycardic or have relative chronotropic incompetence despite beta-agonist therapy, overdrive pacing shortens diastolic filling time which in turn reduces regurgitation to increase forward flow.[47] In the absence of atrioventricular nodal disease, atrial pacing is preferred to maintain atrial-ventricular synchrony. The effects of PPV are generally well tolerated in patients with severe AR due to providing a reduction in both LV preload and LV afterload.[48]

Due to the incompetent aortic valve, intra-arterial MCS devices such as the IABP, TandemHeart, and peripheral ECMO all result in increased AR causing further elevations in the LVEDP, and Impella results in recirculation resulting in ineffective support.[49] Trans-Aortic Balloon to Ease Regurgitation Applying Counter-Pulsation, which inflates a transaortic balloon during diastole to prevent regurgitation, has been proposed, but it has not yet been applied outside of the research setting.[50]

Surgical aortic valve repair or replacement is the current gold standard and in patients with acute pathologies such as aortic dissection, infective endocarditis, or mechanical prosthetic valve failure, it is the only definitive option. Previously, in nonoperative candidates, balloon expandable and self-expanding TAVR valves have been used for native valve AR without calcification but generally carries an increased risk of adverse events, especially valve embolization, which occurs in approximately 15% of cases.[51] Now that a dedicated transcatheter valve is available for those without anchoring calcification, this may be a preferable option for these patients.[52] In patients with intraprosthetic valve failure, valve-in-valve TAVR can be effective with similar results compared with SAVR.[53] Periprosthetic AR can respond to the placement of a percutaneous vascular plug, if the prosthesis has no signs of infection or severe dehiscence.[53,54]

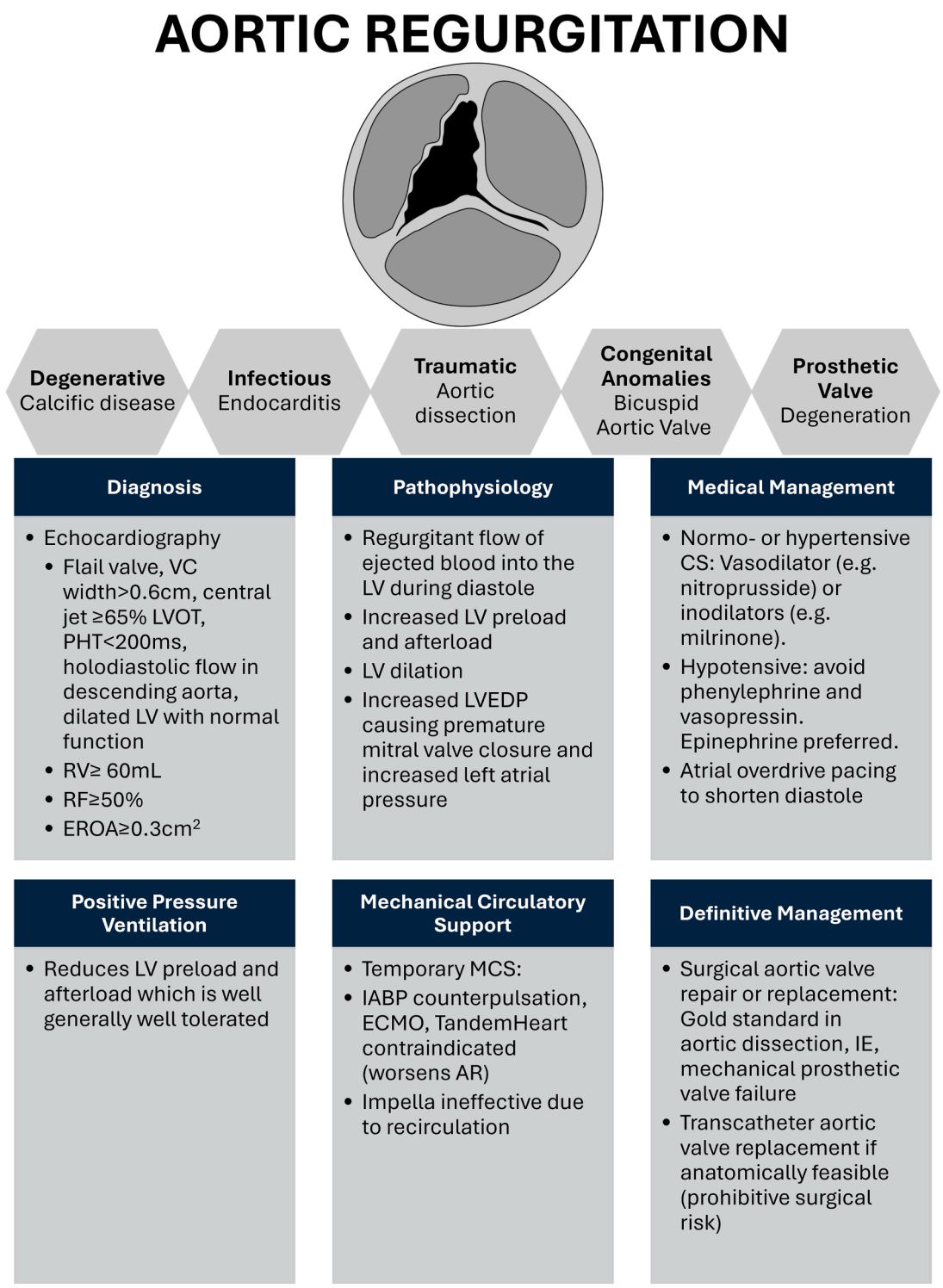


Fig. 3. Key points in the management of aortic regurgitation-related cardiogenic shock.

Mitral Regurgitation

MR is the most common valvular lesion in adults with numerous underlying etiologies (Fig. 4).[54] Patients with cardiomyopathy and underlying chronic MR display a greater propensity to develop CS due to the dynamic nature of MR that worsens under volume and pressure loading.

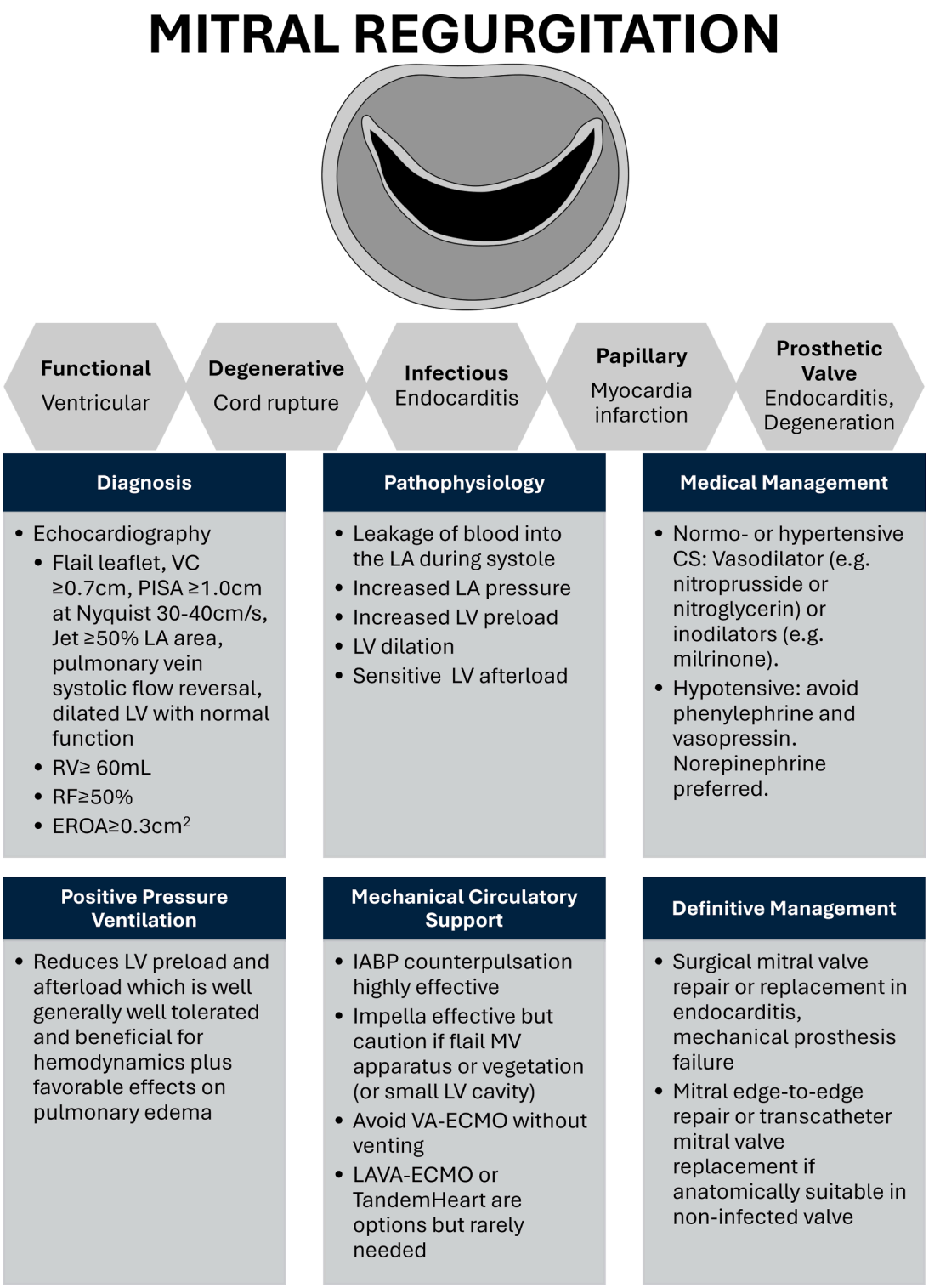


Fig. 4. Key points in the management of mitral regurgitation-related cardiogenic shock.

Chronic MR can be classified as being either primary (due to a structural abnormality of the mitral valve) or secondary (due to the impaired ability of structurally normal mitral valve leaflet coaptation).[55,56] Acute primary MR is often because of infective endocarditis, chordal rupture with a flail segment or leaflet, or papillary muscle rupture after acute myocardial infarction (AMI).

The incidence of ischemic papillary muscle rupture has decreased dramatically in the reperfusion era but carries a 30 day mortality that approaches 80% without surgery and 39% even with surgical correction.[57–59] The dynamic nature of secondary functional MR can make severity assessment challenging in critically ill patients, making it necessary to re-evaluate MR severity as volume overload and systemic hypertension are treated. MR results in chronically elevated left atrial pressure and a volume-loaded left ventricle that leads to progressive left ventricular dilation and dysfunction. Eventually, the left ventricular systolic function becomes impaired. Therefore, like AR, a dilated ventricle with systolic dysfunction is suggestive of a chronic MR or a concomitant cardiomyopathy. Anything less than hypercontractile left ventricular function in the setting of severe MR suggests that underlying ventricular dysfunction is present.

Diagnosis

Rapid pressure equalization from acute severe MR may render murmurs quiet or absent. Eccentric jets can result in misleading findings such as unilateral or asymmetric pulmonary edema misdiagnosed as pneumonia and can easily be missed or underestimated with point-of-care ultrasound. Left ventricular angiography at the time of coronary angiography can identify significant MR but is preferable to avoid to spare the additional contrast load.[56] Pulmonary artery catheterization showing elevated pulmonary capillary wedge pressure with giant V waves should prompt consideration of significant MR warranting further investigations. Comprehensive TTE or TEE is critical to determine the severity and etiology of the MR.[45,60]

Management

Patients with MR often present with pulmonary edema or CS in the setting of hypertensive emergency due to their afterload sensitive state. Initiation of arteriolar vasodilators is an important initial approach in normotensive or hypertensive patients targeting the lowest pressure that preserves end-organ perfusion. In patients with CS, we recommend short-acting vasodilators such as nitroprusside or nitroglycerin when congestion is present, as these are easily titratable.[61] When LV systolic function is impaired, inodilators such as dobutamine or milrinone can be beneficial. In patients with hypotension, we recommend avoiding pure vasoconstrictors such as phenylephrine or vasopressin and instead favor norepinephrine titrated to the lowest effective blood pressure that maintains end-organ perfusion. PPV is generally beneficial for pulmonary venous congestion and improves MR severity by reducing both LV preload and afterload.[48] If patients require MCS, IABP counterpulsation therapy is extremely effective because it lowers afterload without lowering blood pressure, and we consider IABP placement early in preference to vasopressors.[61] If initial medical therapy and IABP support are inadequate, escalating to an Impella is effective in reducing afterload and MR, but caution should be used if there are flail mitral valve segments that could interact with the Impella inflow.[62] Although suboptimal, VA ECMO can be considered for refractory shock, but this increases LV distension worsening worsen MR and pulmonary edema, which requires LV venting using an IABP or Impella. A TandemHeart or LAVA-ECMO might be better MCS strategies to avoid VA ECMO-induced pulmonary edema.

While patients with secondary functional MR often improve with medical therapy alone, those with primary MR require mitral valve intervention for definitive management and are more likely to fail initial medical management alone. Mitral valve repair is the ideal approach for eligible patients with chronic severe primary MR and for selected patients with decompensated acute MR(60). Stabilization with medical therapy and LV unloading is paramount to reduce the procedural risk regardless of the modality of intervention. In patients with infective endocarditis, mitral valve replacement is generally the preferred strategy. In noninfected patients with CS due to MR percutaneous edge-to-edge repair is becoming an established therapy both in primary and secondary MR.[63–65] Among patients with moderate-severe functional MR and LV dysfunction, percutaneous edge-to-edge repair can improve long-term outcomes. For noninfected patients with degeneration of a bioprosthetic mitral valve, annuloplasty ring, or severe MAC, transcatheter mitral valve replacement (TMVR) using a balloon expandable TAVR valve in the mitral position has been performed.[66] For patients with severe periprosthetic MR, percutaneous vascular plugs can be effective after ensuring the etiology is not infectious or overt valve dehiscence.[67] In patients with severe functional MR and advanced cardiomyopathy who are not candidates or fail percutaneous repair, left ventricular assist devices or cardiac transplant should be considered.[67,68]

Mitral Stenosis

MS from annular calcification and prosthetic valve stenosis represents most cases seen in North American practice while more rarely valve thrombosis and bulky endocarditis vegetations may

cause valve obstruction acutely (Fig. 5).[10,69] Rheumatic MS may be encountered among immigrants or travelers from endemic areas of the world.[10] MS causes elevated left atrial pressure and compromises left ventricular filling and commonly diastolic filling time is reduced from tachycardia (especially with loss of atrial contraction in atrial fibrillation), or from volume overload during surgery or in the third trimester of pregnancy.[70]

Diagnosis

The auscultatory findings of MS can be challenging even outside of the critical care setting, and are often inaudible in patients with CS. The murmur is often soft, requires augmentation maneuvers, and is better heard when heart rate is low. Comprehensive transthoracic echocardiography to identify a mitral valve area by direct planimetry less than 1.5 cm^2 or a calculated pressure half time of 150 milliseconds or greater is generally indicative of severe MS with Doppler gradients being less reliable in the state of decompensation where tachycardiac and low cardiac output confound this measurement.[29,30] Once a diagnosis of MS is made, TEE is often required to confirm the etiology and determine the feasibility of percutaneous balloon mitral valvuloplasty (PBMV).[71]

Management

Medical therapy for CS in patients with MS is focused on improving LV diastolic filling. Commonly, tachyarrhythmias (especially atrial fibrillation/flutter) are the primary cause for decompensation with rapid heart rates and loss of atrial contraction resulting in inadequate diastolic filling time for sufficient left ventricular preload.[72] Aggressive rate control should be pursued with beta blockers, calcium channel blockers, amiodarone, and digoxin to achieve a target heart rate of 60 bpm. Rhythm control with electrical or chemical cardioversion should be considered in patients who are anticoagulated, but in those without adequate anticoagulation, the risk of embolization of a left atrial thrombus is high, necessitating systemic anticoagulation and intracardiac imaging to rule out a left atrial thrombus before cardioversion.[72,73] Diuretics can effectively treat pulmonary edema, but therapies including vasodilators and inotropes are ineffective in improving left ventricular filling to provide adequate preload and can even be counterproductive. In hypotensive patients, vasopressors that do not elicit tachycardia such as phenylephrine or vasopressin are preferred. Extreme caution should be used when considering PPV as it may precipitate worsening hypotension due to a further reduction in left ventricular preload and afterload and may also worsen commonly pre-existing pulmonary hypertension and RV failure.[48,74] A key challenge is the patient with low-output RV failure and severe MS, in whom the same therapies that may benefit the RV (inotropes, pulmonary vasodilators) can aggravate pulmonary edema; MCS may be needed in these cases.

If escalation to MCS is required, devices such as the IABP and Impella are generally ineffective. Peripheral ECMO, TandemHeart, and LAVA-ECMO can be considered with the latter 2 being generally preferable due to direct left atrial unloading.[74]

For patients with rheumatic MS who are anatomically feasible, PBMV is the preferred approach.[75] However, many patients have contraindications including heavy calcification, concomitant moderate or more MR, or left atrial thrombus.[76] As a salvage therapy even when echocardiography is unfavorable, PMBV in the unstable CS patient that is unsuitable for immediate surgical intervention may still be considered as a bridge to eventual surgical mitral valve replacement.[71] Transcatheter mitral valves replacement remains limited to stenotic bioprosthetic mitral valves, those with severe mitral annular calcification, and after surgical annuloplasty. However, these interventions require careful preprocedural planning with electrocardiographic-gated cardiac computed tomography (CT) angiography and are more challenging to perform in a shock state. The 30 day mortality in stable patients undergoing mitral valve-in-valve, valve-in-ring, and valve-in-MAC were 3.3%, 6.7%, and 16.7%, respectively, in highly experienced centers.[77] Surgical intervention is often the only option for many of these patients. As new transcatheter mitral valve interventions such as the Sapien M3, Intrepid, and Tendyne devices become approved we may see more options for these patients in the future.[78–80]

POSTPROCEDURAL CONSIDERATIONS AFTER VALVULAR PROCEDURES

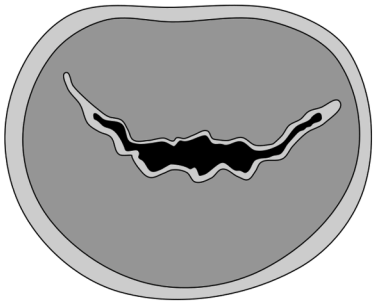
Common Complications

All transcatheter valve interventions share a host of common complications related to vascular access and cardiac intervention. The initial approach to a hypotensive patient after transcatheter valve intervention should focus on rapidly identifying any hemorrhagic complications, cardiac tamponade, arrhythmias, vascular injury, and lastly device-specific complications such as embolization or thrombosis (Fig. 6).

General Principles of Care

In addition to acute resuscitation, we perform the following initial investigations to identify the

MITRAL STENOSIS



Degenerative
Calcific disease

Rheumatic
Valve Disease

Prosthetic Valve
Thrombosis, Degeneration

Diagnosis

- Echocardiography
  - MVA ≤1.5 cm^2 (especially 1.0 cm2)
  - Diastolic PHT ≥150 ms (especially ≥220 ms)

Pathophysiology

- Fixed obstruction to LV filling
- Increased LA pressure
- Decreased LV preload
- Requires prolonged diastolic filling time to maintain cardiac output
- Concomitant pulmonary hypertension and RV dysfunction frequently present

Medical management

- Aggressive management of tachyarrhythmias (beta blockers, CCBs, digoxin, amiodarone). Rhythm control if adequate anticoagulation.
- Diuretics for pulmonary edema

Positive Pressure Ventilation

- Further decreases LV preload and increases RV afterload
- Extreme caution due as it may precipitate hypotension due to LV underfilling

Mechanical Circulatory Support

- IABP counterpulsation and Impella ineffective
- LAVA-ECMO or TandemHeart preferrable due to direct left atrial unloading

Definitive Management

- Percutaneous mitral balloon valvuloplasty if anatomically feasible in rheumatic disease
- TMVR if anatomically feasible
- Surgical mitral valve replacement if not a candidate for transcatheter intervention

Fig. 5. Key points in the management of mitral stenosis-related cardiogenic shock.

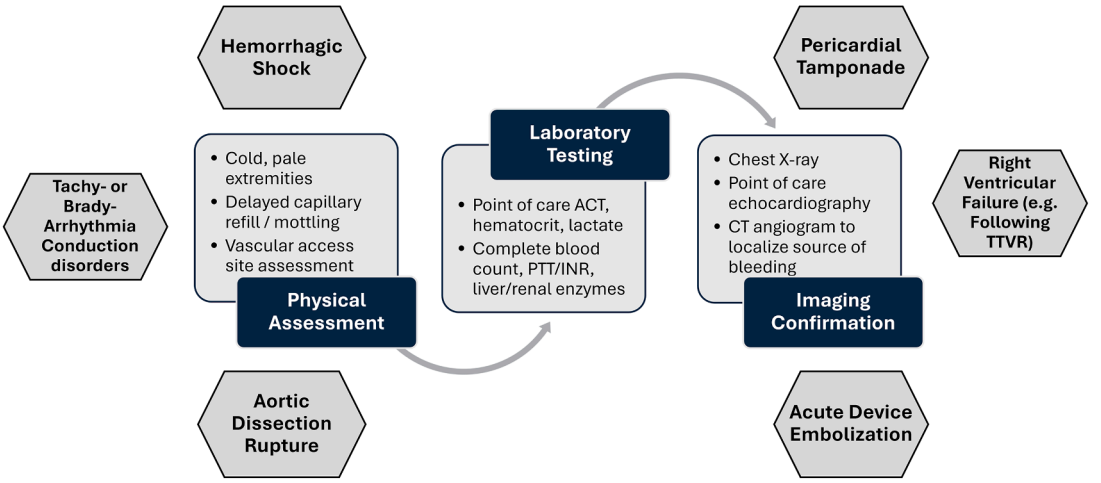


Fig. 6. Considerations and approach to hypotension in the posttranscatheter intervention patient.

etiology of hypotension after an intervention (see Fig. 6).

- Identify and examine all arterial and venous access sites to assess pulses and examine for signs of hemorrhage. Patients who are awake may complain of back, flank, abdominal, or hip pain but otherwise may have no appreciable clinical signs apart from hypotension.
- Point-of-care ultrasound to assess for the presence of a new pericardial effusion and can be useful to evaluate fluid collections (pseudoaneurysm/hematoma) surrounding vascular sites.[81] Retroperitoneal bleeding typically cannot be identified using point-of-care ultrasound, but intraperitoneal bleeding may be seen (eg, by focused assessment with sonography in trauma [FAST] examination).
- Chest radiograph or lung ultrasound to quickly identify a tension pneumothorax related to inadvertent puncture from venous or arterial access in the neck or subclavian.
- Rhythm monitoring and electrocardiography to assess for tachyarrhythmias or bradyarrhythmia (especially high-grade atrioventricular block that would necessitate transcutaneous or transvenous pacing).
- Laboratory testing, including complete blood count, chemistries, lactate, coagulation parameters, and blood gas.

Many patients receive heparin reversal with protamine at the end of the procedure, which can result in a severe anaphylactoid response consisting of systemic hypotension, pulmonary vasoconstriction, allergic symptoms, pulmonary hypertension, bronchoconstriction, and bradycardia which can be delayed.[82] Reactions are generally short-lived and supported with vasopressors and intravenous fluids with rare cases of catastrophic reactions.[83] Complications such as esophageal hemorrhage or perforation can occur form iatrogenic injury to the esophagus from TEE used to guide the procedure especially in patients with pre-existing esophagus or stomach pathologies such as varices.[84]

Transcatheter aortic valve replacement

TAVR is the most common transcatheter valve procedure, typically performed via femoral artery access.[85] Complications causing hypotension include bradyarrhythmia, aortic annulus rupture, aortic dissection or aortic rupture, and left ventricular perforation. Aortic annulus rupture is a rare but often catastrophic complication occurring in 0.33% of cases with increased incidence in patients with a bicuspid valve, asymmetric or small annulus, and when left ventricular outflow tract (LVOT) and valvular calcification is severe.[86] The 30 day mortality of annular rupture is approximately 50%.[87] Ruptures into the pericardium often have immediately hemodynamic decompensation due to hemorrhagic pericardial tamponade unless the leak is small, which may not become evident until the patient returns to the recovery area. Right heart chamber perforation from the pacing wire is often presented in a delayed manner due to lower pressure bleeding. Treatment of acute hemopericardium with cardiac tamponade from cardiac perforations generally requires percutaneous drainage via pericardiocentesis and emergency cardiac surgery if the patient is a candidate; initially, we often defer reversal of heparin to avoid pericardial clotting until drainage is established. If bleeding is significant and ongoing, it can be

beneficial to return the patient's blood with the use of a Cell Saver. Iatrogenic aortic dissection from TAVR is a rare but serious with a 66% 30 day mortality.[88] Typically, these patients require urgent CT angiography for diagnosis with management following standard practices.[89] Device embolization usually occurs at the time of implant, but late embolization causing severe AR has been reported, especially in patients with minimal calcification to anchor the valve.[90,91]

Mitral Valve Transcatheter Edge-to-edge Repair

Mitral valve transcatheter edge-to-edge repair (TEER) is a lower risk transcatheter procedure primarily performed via femoral vein access with rare periprocedural complications.[92] Procedure-specific complications resulting in hypotension can include a hemorrhagic pericardial effusion from a transseptal puncture that is too posterior landing within the epicardial interatrial fold known as Waterson's groove or direct injury to cardiac structures such as the aorta or atrial wall.[93] Although uncommon, CS can occur after mitral TEER due to an abrupt increase in effective ventricular afterload on a severely impaired ventricle.[94,95] Postprocedural single leaflet detachment is rare, but late leaflet tears could abruptly increase the degree of MR with reports of developing progressive shock.[92,96,97] Chest radiograph is a rapid tool to assess for unexpected orientation of mitral TEER devices, but definitive identification often requires echocardiography. Lastly, persistent hypoxemia can occur from right to left shunt through an iatrogenic atrial septal defect.

Transcatheter Mitral Valve Replacement

TMVR is a newer intervention with greater complexity in the devices and anatomic considerations. Short-term postprocedural complications from TMVR can include ventricular perforation causing tamponade, mitral annular disruption, circumflex artery occlusion, and left ventricular outflow tract obstruction (LVOTO). Fixed LVOTO can occur after TMVR due to anatomically crowding of the left ventricular outflow tract especially in patients with small, hypertrophic ventricles.[98] Fixed LVOTO does not typically respond to medical therapy, but a trial of fluid loading, beta-blockers (esmolol), vasoconstrictors (phenylephrine/vasopressin), and RV pacing can be attempted similarly to dynamic LVOTO. Otherwise, options include emergency TAVR with a ventricular implantation to open the LVOTO, anterior mitral valve leaflet laceration, or alcohol septal ablation. In patients who are hemodynamically unstable, temporary MCS with Impella (difficult to use as LVOTO tends to occur in small ventricles) or preferably ECMO are options to bridge patients to emergency cardiac surgery. Procedural risk planning with cardiac CT is key to identify patients who have prohibitive risk of LVOTO and identify those who require pre-emptive therapy to enhance the size of the left ventricular outflow tract. Options can include pre-emptive alcohol septal ablation, Laceration of the Anterior Mitral Leaflet to Prevent Outflow Obstruction, or Balloon-Assisted Translocation of the Mitral Anterior Leaflet. Lastly, persistent hypoxemia is more common due to the larger transseptal hole created to deliver the TMVR devices.[99]

Transcatheter Tricuspid Valve Replacement

Transcatheter tricuspid valve replacement is a relatively new technology associated with a 25% rates of pacemaker implantation and 10% risk of severe bleeding including cardiac tamponade.[100] Acute RV failure after a sudden reduction in TR that rapidly drops RV preload and increases effective RV afterload has been reported in up to 10% of cases.[101] This is more prevalent in patients with significant RV dysfunction or pulmonary hypertension. Inotropic support with inodilators such as dobutamine or milrinone or afterload reduction with inhaled or systemic pulmonary vasodilators may be considered among patients with RV-pulmonary artery uncoupling for temporary postprocedural hemodynamic support.

SUMMARY

VHD represents a minority of cases of CS but is associated with substantial morbidity and mortality. While some forms of valvular CS respond to standard medical therapies including vasopressors, inotropes, and MCS, many types of VHD do not respond favorably to these stabilizing measures. Randomized trial data are lacking to guide care in most cases; therefore, pathophysiological principles and diligent hemodynamic monitoring are essential. Definitive therapy includes surgical or (when feasible) percutaneous valve repair or replacement, but mortality risk remains high even with aggressive care. Early recognition that CS could be due to severe VHD is crucial to avoid a delayed diagnosis, which might preclude a favorable outcome. We advocate for excluding patients with CS from outcomes reporting for percutaneous VHD interventions, which may promote more aggressive use of these potentially life-saving therapies in a population with limited therapeutic alternatives.

CLINICS CARE POINTS

- Valvular causes of cardiogenic shock should be considered early in patients with cardiogenic shock in the absence of an obvious cause such as myocardial infarction or cardiomyopathy.
- Rapid comprehensive echocardiography is needed to accurately diagnose severe valvular lesions contributing to cardiogenic shock.
- The efficacy of standard medical therapy and percutaneous mechanical circulatory support for cardiogenic shock varies by valvular lesion, and may be ineffective or harmful in some pathologies such as mitral stenosis or aortic regurgitation.
- Due to the high risk associated with cardiac surgery in unstable patients with cardiogenic shock, urgent percutaneous valvular interventions should be considered to salvage patients with valvular shock.

DISCLOSURE

The authors have nothing to disclose.

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