Open Access | Original Research
Journal of Heart Valve Disease, 29(1), 2024 | PP: 60– 70
https://doi.org/10.36923/jhvd.v29i1.241

Check for updates

Management of Cardiogenic Shock in Mitral Valve Diseases: A Review Article

Ashraf Ahmed[1], Mohamed Badheeb[2], Kayla Boyea[3], Rasha Kaddoura[4], Fabricio Webber[5], Todd Lane[6], Gilead Lancaster[7]

Article History:

Received: 25-07-2024
Accepted: 26-10-2024
Publication: 10-11-2024

Cite this article as:

Ahmed, A., Badheeb, M., Boyea, K., Kaddoura, R., Webber, F., Lane, T., & Lancaster, G. (2024). Management of Cardiogenic Shock in Mitral Valve Diseases: A Review Article: CS management in MVDs. *Journal of Heart Valve Disease,* 29(1). 60-70. doi.org/10.36923 /jhvd.v29i1.239

Corresponding Author(s):

Ashraf Ahmed
Department of Internal Medicine, Bridgeport Hospital, Yale New Haven Health, CT, USA. Email: asrafo2017@hotmail.com

Abstract: Despite advances in the field of cardiology, cardiogenic shock (CS) remains a management dilemma with a high mortality rate. While most cases of CS are secondary to acute coronary syndrome (ACS), approximately one-fifth can be attributed to delayed mechanical complications of ACS, such as arrhythmia, cardiac tamponade, or valvular heart diseases. The coexistence of CS and mitral valve diseases (MVDs) further complicates clinical presentation, diagnosis, and management strategies. Surgical interventions are considered the gold standard for managing MVDs in the context of CS. However, these patients are often at extreme surgical risk and may not achieve favorable outcomes. Catheter-based interventions have garnered increasing interest, but despite their promising results, most trials have excluded patients in unstable or critical conditions. Pharmacological and mechanical circulatory support provide a bridge to definitive transcatheter or surgical intervention.

Keywords: Mitral Valve diseases, Cardiogenic Shock, percutaneous mitral valve repair, Mechanical Circulatory Support, Inotropic Agents

Key messages:

- Mitral valve diseases are an uncommon cause of cardiogenic shock. The concomitant presence of both conditions creates a management dilemma and underscores the need for patient-centred care with a multi-disciplinary team approach.
- Surgical intervention is the first-line treatment option for mitral valve diseases in cardiogenic shock in patients with non-prohibitive surgical risk.
- Mitral transcatheter edge-to-edge repair is a minimally invasive approach that may be a feasible and safe option.
- Pharmacological and mechanical circulatory support can be a bridge to definitive transcatheter or surgical intervention.

1. Introduction

Acute coronary syndrome (ACS) is acknowledged as the predominant cause of cardiogenic shock (CS) [1]. Nonetheless, valvular heart disease (VHD) also emerges as a critical etiology of cardiogenic shock, necessitating prompt management and intervention [2]. VHD often presents with severe heart failure symptoms that are challenging to manage medically [2]. This, combined with its relatively lower incidence compared to ACS, contributes to its frequent underdiagnosis and lack of recognition as a cause of CS [3]. While acute valvular lesions are rare, the majority of VHD-related CS cases stem from acute decompensation or exacerbation of pre-existing chronic VHD [4]. Among the various forms of VHD, aortic valve diseases followed by mitral valve diseases (MVDs)—particularly acute mitral valve regurgitation (MR)—are common etiologies of CS secondary to valvular abnormalities [2]. Diagnosing and treating MVDs presenting with CS is further complicated by the challenge of determining whether VHD is the primary cause of shock or merely coincidental, especially in patients without a known history of VHD [5]. Furthermore, recognizing and diagnosing acute or severe valvopathies can be difficult even for experienced physicians, as such evaluations require multimodal imaging to assess the severity of CS, left ventricular ejection fraction (LVEF), filling pressures, and the presence of valvular abnormalities, which can be influenced by the prevalence of VHD and ACS as causes of CS [6] [7] [8]. Evidence for the management of CS is often derived from studies involving stable patients with an underlying condition complicated by CS, such as acute myocardial infarction. While this approach is not ideal, it is generally reasonable in the absence of proven alternatives. This is partly due to the difficulty of conducting trials in CS, given the variability of etiologies, severities, and consequent outcomes [9]. Managing CS secondary to VHD is challenging and requires a comprehensive, multidisciplinary approach, as the mortality rate associated with such conditions remains high despite significant advancements in the field [5] [7]. This review aims to provide

[1,2,3] Department of Internal Medicine, Bridgeport Hospital, Yale New Haven Health, CT, USA.
[4] Department of Pharmacy, Heart Hospital, Hamad Medical Corporation, Doha, Qatar
[5] Department of Cardiovascular Diseases, University of Miami, FL, USA
[6] Department of Academic Affairs, Yale New Haven Health, Bridgeport Hospital, CT, USA.
[7] Department of Cardiovascular Diseases, Bridgeport Hospital, Yale New Haven Health, CT, USA

a comprehensive overview of the approach and management of CS secondary to mitral valvopathies, specifically mitral stenosis and mitral regurgitation, highlighting the complexities, challenges, and recent updates related to this complex clinical syndrome.

2. Cardiogenic Shock

2.1. Definition and classification of CS

Cardiogenic shock is a critical clinical syndrome characterized by decreased myocardial contractility and cardiac output, resulting in global hypoperfusion and end-organ failure [10]. CS is defined by a set of hemodynamic parameters: systolic blood pressure (SBP) below 90 mmHg for 30 minutes or more, or the need for medical circulatory support to maintain an SBP of 90 mmHg or higher, and a cardiac index of less than 2.2 L/min/m^2 [10]. While this definition provides a reasonable estimate of poor hemodynamics, it does not account for the acuity of these parameters, as seen in patients with chronic myocardial disease who maintain proper tissue perfusion with an SBP below 90 mmHg [10]. Additionally, many patients with CS may have an SBP above 90 mmHg due to initial vasoconstriction in the early stages of CS [10]. Classification systems for CS, such as those by Killip and Forrester, do not address CS severity, hemodynamic-guided management, or the timely use of circulatory support devices [9]. To address these gaps, the Society for Cardiovascular Angiography and Interventions (SCAI) proposed a classification system to facilitate communication about patient status and enable rapid assessment [11]. The SCAI classification comprises a five-stage scheme: stage A (at risk), stage B (beginning), stage C (classic), stage D (deteriorating), and stage E (extremis). Each stage is assessed based on three domains: physical examination, biochemical markers, and hemodynamic parameters [11] [12]. Comprehensive evaluation should include assessing hemodynamic instability, peripheral hypoperfusion (evidenced clinically by altered sensorium, cold mottled extremities, decreased urine output, and elevated lactate levels), and signs of pulmonary congestion [10].

2.2. General management of CS

Early hemodynamic support is critical for patients in shock, regardless of the underlying cause. Pharmacological or mechanical circulatory support should be initiated promptly to counteract hypotension and maintain tissue perfusion. Intravenous inotropes and vasopressors (i.e., vasoactive agents) are the initial circulatory support for CS patients unresponsive to fluid resuscitation. Vasoactive agents help maintain end-organ perfusion by reducing filling pressures and enhancing myocardial contractility and cardiac output. Several randomized controlled trials have investigated vasoactive agents in CS [13] [14] [15] [16] [17] [18] [19]. Overall, vasoactive agents improved hemodynamic parameters but did not affect mortality, except for dopamine, which increased mortality [13]. Although add-on levosimendan showed short-term survival benefits compared to enoximone, the study included only 32 patients with severe refractory CS [19]. A meta-analysis revealed no mortality benefit when using vasoactive agents in CS complicating acute myocardial infarction [20]. Additionally, evidence did not demonstrate the superiority of one vasoactive agent over another. Consequently, international guidelines do not have unified recommendations for a first-line vasoactive agent for treating CS [21]. Except for levosimendan, vasoactive agents increase myocardial oxygen consumption and the risk of arrhythmias due to increased intracellular calcium [9]. Therefore, pharmacological circulatory support should not be used for long durations, and if there is a need for ongoing dose escalation, temporary mechanical circulatory support (MCS) devices should be considered without delay (Table 1). The intra-aortic balloon pump (IABP) is often the initial choice, but pharmacological support may still be required alongside it. More robust support can be achieved with Impella® LP 2.5 or CP. In cases of patient deterioration, devices like TandemHeart® (left atrium-to-aorta assist devices), veno-arterial extracorporeal membrane oxygenation (VA-ECMO), or Impella® LP 5.0 can be considered. MCS devices help unload the ventricles, reducing filling pressure and volume [9]. Randomized controlled trials did not demonstrate mortality benefits with IABP [22], VA-ECMO [23] [24], or Impella® LP 2.5 [25] and Impella® CP [26]. However, a recent trial indicated that Impella® CP use was associated with a lower mortality risk compared to standard care, albeit with a higher incidence of adverse events [27]. Other devices are also available for right ventricular support. Finally, durable MCS (e.g., left ventricular assist devices) or heart transplantation may be considered in cases of refractory CS when revascularization, vasoactive agents, and temporary MCS have failed [9].

Table 1: Temporary mechanical circulatory support devices

MCS device	Description	Hemodynamic effects
LV support		
IABP	• Device: counter pulsation pump placed in descending aorta • CO support/flow: 0.3-0.5 L/min • Implantation: percutaneous	• LV unloading (modest) • ↓ Afterload • ↑ Cardiac power • ↑ MAP • ↓ LVEDP • ↑ Coronary perfusion

Impella® LP 2.5	• Device: micro-axial pump that decompresses LV and pumps blood into ascending aorta • CO support/flow: 1.0-2.5 L/min • Implantation: percutaneous • Max implant duration: 7-10 day	• LV unloading • ↑↑ Cardiac power • ↓ Afterload • ↑↑ MAP • ↓↓ LVEDP • ↓↓ LV preload • ↑ Coronary perfusion
Impella® CP	• Device: micro-axial pump that decompresses LV and pumps blood into ascending aorta • CO support/flow: 3.7-4.0 L/min • Implantation: percutaneous • Max implant duration: 7-10 day	• As above
Impella® LP 5.0	• CO support/flow: 5.0 L/min • Implantation: surgical cutdown of artery prior to insertion of sheath • Max implant duration: 2-3 week	• As above
Impella® 5.5	• CO support/flow: 5.5-6.0 L/min • Insertion: femoral or axillary artery	• LV unloading • ↑↑ Cardiac power • ↓↓ Afterload • ↑ Coronary perfusion
TandemHeart® LV-FA	• Device: centrifugal pump with inflow cannula placed in LA and outflow cannula in one or both femoral arteries across interatrial septum • Pumps blood from LA to iliofemoral arterial system • CO support/flow: 2.5-5.0 L/min • Implantation: transeptal puncture • Max implant duration: 2-3 week	• LV unloading • ↑↑ Cardiac power • ↑ Afterload • ↑↑ MAP • ↓↓ LVEDP • ↓↓ LV preload
RV support		
Impella® RP	• Device: axial catheter-based pump or RV assist device that pumps blood from RA to PA • CO support/flow: 4.0-5.0 L/min	• RV unloading
CentriMag®	• Device: centrifugal pump with magnetically levitated propeller • CO support/flow: up to 9.9 L/min • Insertion: femoral-to-femoral bypass	• RV unloading
TandemHeart® RA-PA	• CO support/flow: 4.0. L/min	• RV unloading
LV and RV support		
VA-ECMO	• Device: heart-lung bypass machine • V-V for oxygenation only or V-A for oxygenation and circulatory support • CO support/flow: 7.0 L/min • Implantation: percutaneous or surgical cutdown • Max implant duration: 3-4 week	• RV unloading • ↑↑↑ Cardiac power • ↑↑↑ Afterload • ↑↑ MAP • ↓ LV preload

Abbreviations: CO; cardiac output, ECMO; extracorporeal membrane oxygenation, FA; femoral artery, IABP; intra-aortic balloon pump, LV; left ventricle/ventricular, LVEDP; left ventricle end-diastolic pressure, MAP; mean arterial blood pressure, Max; maximum, PA; pulmonary artery, RA; right atrium/arterial, RV; right ventricle, V-A; veno-arterial, V-V; veno-venus.

3. Mitral Valve Diseases

3.1. Types of MVDs

The prevalence of MVDs is approximately 15% of all VHDs, and it is higher in older adults and in developing countries [28]. Mitral valve prolapse is the most common and benign type of MVD, accounting for around 3% of the total population [29]. However, it is associated with MR, infective endocarditis, and potentially CS only when it progresses into MR [30]. MR affects about 2% of the total population [29] and is the second most common VHD in Europe [29]. MR can be classified as either primary (degenerative), where the valve anatomy is damaged—affecting the leaflets, chordae, or papillary muscles, causing failure of leaflet coaptation—or secondary (functional), due to a dilated left ventricle that leads to mitral annular dilation and papillary muscle displacement [4], causing posterior leaflet tethering [29] [31]. MR can present chronically, where patients experience progressive heart failure symptoms, or acutely, with CS and acute decompensated heart failure [32]. Chronic MR can also lead to CS if hemodynamics suddenly change, for example, due to tachyarrhythmias or sepsis [33]. Primary MR is most frequent in Western countries [31], with mitral valve prolapse as the leading cause [34], whereas in low-income countries, rheumatic etiology is more common. Secondary MR is frequently diagnosed in patients with ischemic or dilated cardiomyopathies [31]. Mitral valve stenosis (MS) is most often caused by rheumatic heart disease [32] [35], and it is more prevalent in low- and middle-income countries, predominantly affecting females and flaring

up during pregnancy [35]. Nonrheumatic or degenerative mitral stenosis, often due to mitral annular calcification, has a different pathology, with its prevalence increasing with age [31] [34]. Calcific MS results from calcification of the mitral annulus that extends into the leaflet bases, leading to annular narrowing and leaflet rigidity [34]. MS does not develop acutely, as it is inherently a chronic condition, with affected patients typically presenting with heart failure symptoms. However, it is not an uncommon cause of CS [32].

3.2. General management of MVDs

In primary MR, no drug class, including vasodilators, has been proven to reduce MR severity [34]. Surgery is indicated for patients with symptomatic severe primary MR and acceptable surgical risk. The presence of specific echocardiographic measurements can warrant intervention, regardless of symptoms. In asymptomatic patients with severe primary MR lacking these specific criteria, watchful waiting is a reasonable approach. Mitral valve repair is the preferred option if deemed potentially durable by the Heart Team [31] [34]. If repair is not feasible, mitral valve replacement is recommended. Urgent surgery is necessary in cases of acute severe MR, such as papillary muscle rupture, which requires valve replacement. Transcatheter mitral valve implantation for severe primary MR is feasible and safe for inoperable patients or those with high surgical risk [31]. In secondary MR, guideline-directed medical therapy for heart failure with reduced ejection fraction and severe secondary MR should be optimized as the first-line management, including the use of cardiac devices as indicated. If symptoms persist after optimal medical therapy, mitral valve intervention should be considered. Mitral transcatheter edge-to-edge repair (TEER), a minimally invasive procedure, is recommended for selected patients [31] [34], supported by evidence from randomized trials [36] [37]. Surgery involving mitral valve replacement is preferred over repair. Patients with end-stage left ventricular and/or right ventricular failure may be candidates for cardiac transplantation or left ventricular assist device implantation. In cases of rheumatic MS, medical therapy depends on the presence of valvular atrial fibrillation or tachycardia with normal sinus rhythm. Optimal management involves percutaneous mitral balloon commissurotomy (PMBC) or surgery (open or closed commissurotomy) [31] [34] for patients with moderate-to-severe rheumatic MS [31]. Mitral valve replacement should be considered for patients with severe symptoms when no other treatment options are available [34]. In patients with nonrheumatic calcific MS, both surgical and transcatheter interventions are considered high-risk due to the lack of randomized trial evidence [31]. However, interventions should be performed in patients with severe symptoms [34].

4. Acute Mitral Valve Regurgitation and Cardiogenic Shock Management

Acute MR is a medical emergency that leads to severe acute decompensated heart failure due to the sudden increase in pressure and volume load on the left atrium and a sharp decline in cardiac output [38]. Figure 1 illustrates the hemodynamic changes associated with MVDs complicated by CS. Acute MR occurs due to disruptions in various parts of the mitral valve apparatus. Infective endocarditis can cause chordal rupture or leaflet perforation. Myxomatous mitral valve disease is a cause of spontaneous chordal rupture. Acute ST-segment myocardial infarction, typically involving an inferior infarction, is a common cause of papillary muscle rupture [34]. Patients with MR and CS are typically over 65 years of age, predominantly male (>70%), and often have multiple comorbidities, high-risk surgical scores, functional MR, ischemic etiology of cardiomyopathy (if present), and severe MR at baseline. Early diagnosis of acute MR and identification of the underlying etiology are key to effective management [38].

4.1. Circulatory Support for CS in Acute MR

Pharmacological or mechanical circulatory support serves as a bridge to recovery, especially when surgery is unsuitable for critically ill patients [38]. The goal of circulatory support is to improve cardiac output and tissue perfusion. The optimal vasoactive drug is not clearly defined and is usually tailored based on hemodynamic parameters and physiological changes [39]. In MR, cardiac output decreases as much of the blood flow is pumped backwards, a situation exacerbated by increased afterload. Therefore, vasopressors and inotropes might not be the best options in such cases [40]. Milrinone and dobutamine may be preferred over vasopressors [6]. However, norepinephrine, which has weak β-adrenergic properties, can be a suitable agent to augment cardiac output, mean arterial pressure (MAP), and systemic vascular resistance (SVR), with only a modest increase in myocardial oxygen demand. Additionally, it has a short half-life and does not require dosage adjustment in cases of renal impairment [41]. The use of vasodilators such as nitrates or sodium nitroprusside reduces preload and improves cardiac contractility [42]. However, the mortality benefit of vasodilators in hemodynamic instability remains controversial [43]. Overall, vasodilators enhance hemodynamic compensation in acute MR, but their use is limited by hypotension, which is exacerbated by reduced peripheral resistance [34].

Mechanical circulatory support (MCS) devices are often used as a bridge to definitive MR management [44]. The choice of an MCS device in MR should depend on availability and, more importantly, the clinical team's familiarity and experience with the device [45]. In MR, an intra-aortic balloon pump (IABP) can be a good initial choice as it decreases afterload and modestly increases cardiac output, thereby improving MR [46]. The non-pulsatile IMPELLA® device is more favorable as it directly unloads the left ventricle, augmenting cardiac output more effectively than the IABP without exacerbating MR [47]. Vandenbriele et al. were the first to report the feasibility of an

upfront Impella® strategy to facilitate bridging to transcatheter intervention with MitraClip® [48]. The VA-ECMO device increases vascular resistance, so it is not typically used alone but in combination with the Impella® device (i.e., ECPella configuration). Venoarterial (VA)-ECMO and left atrial-veno-arterial (LAVA)-ECMO may be considered as first-line options rather than ECMO alone [49]. Figure 2 summarizes the management of MVDs complicated by CS.

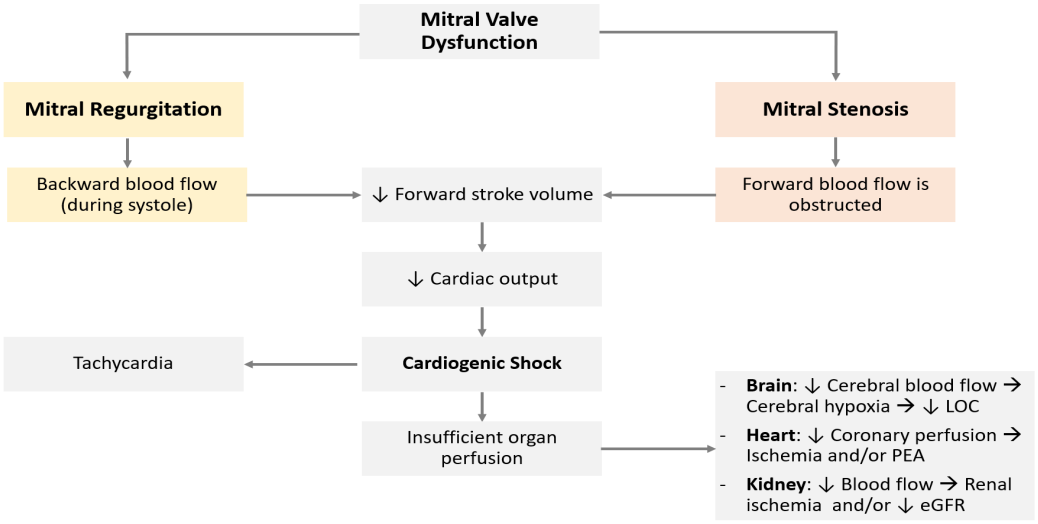


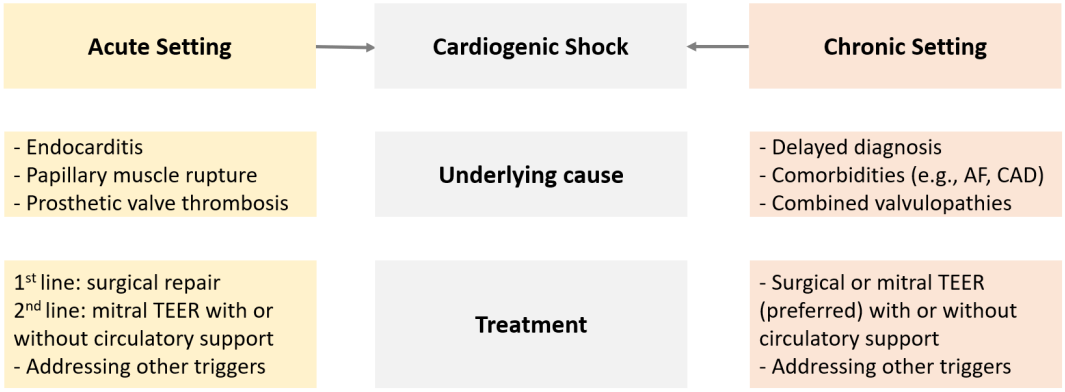
Figure 1: Mitral valve diseases are complicated with cardiogenic shock.

Abbreviations: eGFR; estimated glomerular filtration rate, LOC; level of consciousness, PEA; pulseless electrical activity.

4.1. Catheter-based management of MR complicated with CS

The gold standard for managing acute MR is surgical intervention [34]. However, up to 53% of patients with severe MR remain untreated and are managed medically. These patients typically have low LVEF (around 27%) and high surgical risk, and many decline surgery during acute critical conditions [50]. A less invasive approach using transcatheter mitral valve repair or mitral transcatheter edge-to-edge repair (TEER) has been explored to improve outcomes in patients with MR, although those with acute MR and CS were excluded from pivotal trials [50] [51]. Kovach et al., in a single-center study, evaluated mitral TEER in 20 critically ill patients—half with primary MR and half with secondary MR, including eight with CS. The success rate was 85%, with a 30-day mortality rate of 21%, comparable to the benchmark for surgical mortality [52]. Aldrugh and colleagues reported data from the National Readmission Database for patients with CS and MR treated medically, with TEER, or with surgical valve replacement or repair. The in-hospital mortality rates were 31% for medically treated patients, 26% for TEER, 14% for surgical replacement, and 17% for repair (P<0.001) [53]. Lee et al. compared emergent mitral TEER in CS with elective TEER and found no significant difference in success rates (87.5% vs. 95.2%, p=0.514), but long-term survival was poorer with the emergent procedure (p=0.008) [54]. In the International Registry of MitraClip in Acute Mitral Infarction (IREMMI), 30-day mortality rates for CS versus non-CS patients were 10% and 2.3%, respectively (P=0.212). There were no significant differences in combined mortality and rehospitalization at the seven-month follow-up (28% vs. 25.6%, p=0.793), immediate procedural success (90% vs. 93%, p=0.739), or readmission due to heart failure at three months (13% vs. 23%, p=0.253) [55]. These results support the safety of mitral TEER in critical conditions such as CS. Additionally, analysis of IREMMI registry patients with an LVEF below 35% indicated that TEER is feasible and safe even in those with severe left ventricular dysfunction [56]. Flint et al. identified 12 patients (8.9%) among 135 MR patients who underwent TEER and concluded that TEER is a reasonable option for critically ill patients. Surviving CS patients had a shorter time from MR diagnosis to TEER procedure compared to those who did not survive (35±68 vs. 374±111 days, p=0.0001) [57]. Findings from a large national database (n=38,166) showed that the use of MitraClip® in CS increased significantly from 2014 to 2019. Patients who received MitraClip® had lower in-hospital (odds ratio 0.60, 95% CI: 0.47–0.77, p<0.001) and one-year (hazard ratio 0.76, 95% CI: 0.65–0.88, p<0.001) mortality rates compared to those who did not [58]. Other smaller studies have confirmed the feasibility and safety of TEER in CS [59] [60] [61] [62] [63] [64]. Despite the promising results, these studies were observational, which may introduce bias and confounding. The ongoing Transcatheter Mitral Valve Repair for Inotropes Dependent Cardiogenic Shock (CAPITAL MINOS) open-label, multicenter randomized trial (NCT05298124) is enrolling 144 patients with SCAI class C or D CS and MR of at least grade 3+ to receive TEER or medical treatment. This trial aims to determine whether TEER improves clinical outcomes and can be an optimal treatment for high-risk patients [65]. The European Association of Percutaneous Cardiovascular Interventions (EAPCI) recently updated its guidance on managing VHD in critical conditions, justifying the use of mitral TEER in MR and CS if surgery poses prohibitive risk [66]. The MitraClip® system, initially approved in Europe and the United States in 2008 and 2018, respectively, has undergone various techno-

logical advancements, resulting in the second (NT), third (NTR and XTR), and fourth (NTW and XTW) generations [67]. Current evidence supports its efficacy with infrequent adverse events [68] [69]. However, no studies have yet evaluated its use in acute or critical settings



Valvulopathy	Drugs	MCS	Catheter-based management
MR	Inotropes, nitroprusside, (NE)	IABP, ECMO, LVAD	Mitral TEER
MS	NE, inotropes (for severe RV dysfunction)	ECMO	PMBC, TMVI (in severe calcification)

Figure 2: Management of mitral valve diseases complicated with cardiogenic shock.

Abbreviations: AF; atrial fibrillation, CAD; coronary artery disease; ECMO; extracorporeal membrane oxygenation, IABP; intra-aortic balloon pump, LVED; left ventricular assist device, MCS; mechanical circulatory support, MR; mitral valve regurgitation, MS; mitral valve stenosis, NE, norepinephrine, PMBC; percutaneous mitral balloon commissurotomy, TEER; transcatheter edge-to-edge repair, TMVI; transcatheter mitral valve implantation.

5. Acute Mitral Valve Stenosis and Cardiogenic Shock Management

The evidence for the medical management of patients with MS and CS is limited. Surgical intervention poses significant challenges due to the critical hemodynamic changes that occur during cardiopulmonary bypass [6].

5.1. Circulatory support for CS in MS

There is no consensus on the most effective vasoactive medications for CS in MS [70]. However, understanding the pathophysiology and hemodynamics of patients with MS can aid in management. Cardiac contractility is generally not affected by the presence of MS, but reflex vasoconstriction, increased afterload, decreased left ventricular filling, and left atrial dilation can lead to pulmonary congestion and hypoxia, which are the driving factors of CS in MS [71]. Inotropes may be used to enhance right ventricular contractility, ideally guided by invasive hemodynamic measurements [6]. The key hemodynamic feature in MS is the high left atrial/left ventricular gradient, which affects the pulmonary circulation. Therefore, the most suitable MCS device would be the TandemHeart® or LAVA-ECMO, as these directly unload the left atrium into the systemic arterial circulation, compensating for poor flow and output due to MS [49]. While IABP and Impella® can be used, their effectiveness is limited because left atrial emptying into the left ventricle is often inadequate in patients with MS [34] [50]. Both pharmacological and mechanical circulatory support should act as a bridge to definitive valve repair, such as emergent catheter-based or surgical intervention [72].

5.2. Catheter-based management of MS complicated with CS

Although mitral valve narrowing does not occur acutely, coexisting stressors such as arrhythmias, ischemia, and sepsis can impact the hemodynamics of patients with MS, leading to a drop in cardiac output and CS [5] [6]. Consequently, management becomes challenging, requiring the prompt initiation of circulatory and respiratory support, treatment of the underlying cause, and addressing triggering factors [31]. PMBC, the first catheter-based treatment for VHD since 1982, remains the primary intervention for severe MS, particularly in the presence of calcification [6]. PMBC has been proven to alleviate the symptoms of severe MS and increase the mitral valve area [34]. However, data on its role in CS are scarce. In cases of severe calcified MS, although data on transcatheter mitral valve intervention as an alternative for high-risk surgical patients are limited, urgent PMBC appears to be safe and feasible in critically ill patients [6]. Pillai et al. reported outcomes of PMBC in pregnant women with severe MS and decompensated heart failure, defined by the New York Heart Association (NYHA) functional classification as class IV, acute pulmonary edema, and CS [35]. Among the 96 patients studied, 17% were in CS, and 33.33% required mechanical ventilation. The procedure success rate was 80.2%, with 11 deaths reported: six in the successful procedure group and five in the failed procedure group [35]. Worku et al. reported a case in which a transapical mitral valve-in-valve implant successfully managed mitral valve prosthetic stenosis and combined regurgitation lesions [73]. Despite limited data, the EAPCI recently discussed the complex management of CS in

the context of VHD and supported a minimally invasive, transcatheter approach for patients with high surgical risk [66]. Our systematic literature search identified six cases in which PMBC successfully rescued CS secondary to MS, with favorable outcomes [74] [75] [76] [77] [78] [49]. The average patient age was 35.1±12.3 years, with an equal male-to-female ratio. The survival rate was 83.3%, with one in-hospital death (Table 2).

Table 2: Summary of the cases reported to use PMBC in the management of CS

Parameters	Case 1 [74]	Case 2 [75]	Case 3 [78]	Case 4 [76]	Case 5 [77]	Case 6 [49]
Age	18	59	35	32	30	37
Sex	Male	Female	Female	Male	Female	Male
History of RHD	Yes	Yes	NR	Yes	NR	No
Admitting diagnoses	Sepsis	CS	CS	HF	CS	CS
Intubation	Yes	NR	Yes	Yes	Yes	Yes
Pre MVG (mmHg)	12.6	25	12	NR	33	9
Post MVG (mmHg)	4	16	3	NR	13	2
Pre LAP (mmHg)	28	35	NR	22	36	17
Post LAP (mmHg)	18	26	NR	15	28	9
Pre MVA (cm^2)	0.7	0.5	NR	0.4	0.8	0.7
Post MVA (cm^2)	1.9	1.1	NR	NR	1.2	1.9
Pre CO (L/min)	3.4	1.8	NR	NR	3.2	NR
Post CO (L/min)	4.2	2.5	NR	NR	4.7	NR
Pre CI (L/min/m^2)	1.9	NR	NR	NR	2.5	NR
Post CI (L/min/m^2)	2.5	NR	NR	NR	2.9	NR
Outcome	Survived	Survived	Survived	Deceased	Survived	Survived

Abbreviations: CI, Cardiac Index; CO, cardiac output; CS, cardiogenic shock; LAP, Left Atrial Pressure; MVA, Mitral valve area; MVG, Mitral valve gradient; NR, not reported; PMBC, percutaneous mitral balloon commissurotomy; RHD, Rheumatic Heart Disease.

6. Conclusion

Managing cardiogenic shock complicating MVD is challenging and requires a multidisciplinary approach. Strong evidence supporting any specific intervention is limited. Pharmacological and mechanical circulatory support play a crucial role as a bridge to definitive management. Although surgical intervention for the mitral valve remains the gold standard, high or very high surgical risk can impact outcomes. Minimally invasive catheter-based interventions such as mitral TEER and PMBC are currently considered safe and feasible. Further randomized studies are necessary to address the management uncertainty and validate short- and long-term outcomes.

Author Contributions

Author 1 is responsible for the literature review and manuscript writing. Authors 2, 4, and 5 are contributing to the manuscript writing. Author 3 has contributed to the tables and figures, manuscript writing, and critical revision. Authors 6 and 7 have assisted with the systematic literature search of the case reports, manuscript proofreading, and data interpretation.

References

[1]. D. Kolte *et al.*, "Trends in Incidence, Management, and Outcomes of Cardiogenic Shock Complicating ST-Elevation Myocardial Infarction in the United States," *Journal of the American Heart Association*, vol. 3, no. 1, p. e000590, doi: 10.1161/JAHA.113.000590.

[2]. P. Lurz and C. Besler, "Mitral Regurgitation in Cardiogenic Shock," *JACC: Cardiovascular Interventions*, vol. 14, no. 1, pp. 12–14, Jan. 2021, doi: 10.1016/j.jcin.2020.09.030.

[3]. E. Lüsebrink, S. Massberg, and M. Orban, "Ten things ICU specialists need to know about new valvular procedures in interventional cardiology," *Intensive Care Med*, vol. 46, no. 1, pp. 102–106, Jan. 2020, doi: 10.1007/s00134-019-05824-6.

[4]. M. Enriquez-Sarano, C. W. Akins, and A. Vahanian, "Mitral regurgitation," *Lancet*, vol. 373, no. 9672, pp. 1382–1394, Apr. 2009, doi: 10.1016/S0140-6736(09)60692-9.

[5]. S. Bernard, S. Deferm, and P. B. Bertrand, "Acute valvular emergencies," *European Heart Journal. Acute Cardiovascular Care*, vol. 11, no. 8, pp. 653–665, Aug. 2022, doi: 10.1093/ehjacc/zuac086.

[6]. M. Akodad, G. Schurtz, J. Adda, F. Leclercq, and F. Roubille, "Management of valvulopathies with acute severe heart failure and cardiogenic shock," *Archives of Cardiovascular Diseases*, vol. 112, no. 12, pp. 773–780, Dec. 2019, doi: 10.1016/j.acvd.2019.06.009.

[7]. P. Ponikowski *et al.*, "2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the special contribution of the Heart Failure Association (HFA) of the ESC," *Eur Heart J*, vol. 37, no. 27, pp. 2129–2200, Jul. 2016, doi: 10.1093/eurheartj/ehw128.

[8]. P. Shah and J. A. Cowger, "Cardiogenic shock," *Crit Care Clin*, vol. 30, no. 3, pp. 391–412, Jul. 2014, doi: 10.1016/j.ccc.2014.03.001.

[9]. R. Kaddoura and S. Elbdri, "Current evidence in the diagnosis and management of cardiogenic shock complicating acute coronary syndrome," *RCM*, vol. 22, no. 3, Art. no. 3, Sep. 2021, doi: 10.31083/j.rcm2203078.

[10]. C. Vahdatpour, D. Collins, and S. Goldberg, "Cardiogenic Shock," *Journal of the American Heart Association*, vol. 8, no. 8, p. e011991, Apr. 2019, doi: 10.1161/JAHA.119.011991.

[11]. "SCAI clinical expert consensus statement on the classification of cardiogenic shock - Baran - 2019 - Catheterization and Cardiovascular Interventions - Wiley Online Library." Accessed: Oct. 20, 2024. [Online]. Available: https://onlinelibrary-wiley-com.qulib.idm.oclc.org/doi/full/10.1002/ccd.28329

[12]. S. S. Naidu *et al.*, "SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies: This statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC) Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in December 2021.," *Journal of the Society for Cardiovascular Angiography & Interventions*, vol. 1, no. 1, Jan. 2022, doi: 10.1016/j.jscai.2021.100008.

[13]. D. D. Backer *et al.*, "Comparison of Dopamine and Norepinephrine in the Treatment of Shock," *New England Journal of Medicine*, vol. 362, no. 9, pp. 779–789, Mar. 2010, doi: 10.1056/NEJMoa0907118.

[14]. B. Levy, P. Perez, J. Perny, C. Thivilier, and A. Gerard, "Comparison of norepinephrine-dobutamine to epinephrine for hemodynamics, lactate metabolism, and organ function variables in cardiogenic shock. A prospective, randomized pilot study," *Crit Care Med*, vol. 39, no. 3, pp. 450–455, Mar. 2011, doi: 10.1097/CCM.0b013e3181ffe0eb.

[15]. B. Levy *et al.*, "Epinephrine Versus Norepinephrine for Cardiogenic Shock After Acute Myocardial Infarction," *J Am Coll Cardiol*, vol. 72, no. 2, pp. 173–182, Jul. 2018, doi: 10.1016/j.jacc.2018.04.051.

[16]. M. J. García-González, A. Domínguez-Rodríguez, J. J. Ferrer-Hita, P. Abreu-González, and M. B. Muñoz, "Cardiogenic shock after primary percutaneous coronary intervention: Effects of levosimendan compared with dobutamine on haemodynamics," *Eur J Heart Fail*, vol. 8, no. 7, pp. 723–728, Nov. 2006, doi: 10.1016/j.ejheart.2006.01.007.

[17]. J. T. Fuhrmann *et al.*, "Levosimendan is superior to enoximone in refractory cardiogenic shock complicating acute myocardial infarction," *Crit Care Med*, vol. 36, no. 8, pp. 2257–2266, Aug. 2008, doi: 10.1097/CCM.0b013e3181809846.

[18]. T. Husebye *et al.*, "Levosimendan in acute heart failure following primary percutaneous coronary intervention-treated acute ST-elevation myocardial infarction. Results from the LEAF trial: a randomized, placebo-controlled study," *Eur J Heart Fail*, vol. 15, no. 5, pp. 565–572, May 2013, doi: 10.1093/eurjhf/hfs215.

[19]. "Milrinone as Compared with Dobutamine in the Treatment of Cardiogenic Shock | New England Journal of Medicine." Accessed: Oct. 20, 2024. [Online]. Available: https://www-nejm-org.qulib.idm.oclc.org/doi/full/10.1056/NEJMoa2026845

[20]. "Vasopressors and Inotropes in Acute Myocardial Infarction Related Cardiogenic Shock: A Systematic Review and Meta-Analysis - PubMed." Accessed: Oct. 20, 2024. [Online]. Available: https://pubmed-ncbi-nlm-nih-gov.qulib.idm.oclc.org/32629772/

[21]. R. Kaddoura *et al.*, "Vasoactive pharmacologic therapy in cardiogenic shock: a critical review," *J Drug Assess*, vol. 10, no. 1, pp. 68–85, 2021, doi: 10.1080/21556660.2021.1930548.

[22]. H. Thiele *et al.*, "Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock," *New England Journal of Medicine*, vol. 367, no. 14, pp. 1287–1296, Oct. 2012, doi: 10.1056/NEJMoa1208410.

[23]. "Venoarterial extracorporeal membrane oxygenation or standard care in patients with cardiogenic shock complicating acute myocardial infarction: the multicentre, randomised EURO SHOCK trial - PubMed." Accessed: Oct. 20, 2024. [Online]. Available: https://pubmed-ncbi-nlm-nih-gov.qulib.idm.oclc.org/37334659/

[24]. H. Thiele *et al.*, "Extracorporeal Life Support in Infarct-Related Cardiogenic Shock," *New England Journal of Medicine*, vol. 389, no. 14, pp. 1286–1297, Oct. 2023, doi: 10.1056/NEJMoa2307227.

[25]. M. Seyfarth *et al.*, "A randomized clinical trial to evaluate the safety and efficacy of a percutaneous left ventricular assist device versus intra-aortic balloon pumping for treatment of cardiogenic shock caused by myocardial infarction," *J Am Coll Cardiol*, vol. 52, no. 19, pp. 1584–1588, Nov. 2008, doi: 10.1016/j.jacc.2008.05.065.

[26]. D. M. Ouweneel *et al.*, "Percutaneous Mechanical Circulatory Support Versus Intra-Aortic Balloon Pump in Cardiogenic Shock After Acute Myocardial Infarction," *J Am Coll Cardiol*, vol. 69, no. 3, pp. 278–287, Jan. 2017, doi: 10.1016/j.jacc.2016.10.022.

[27]. J. E. Møller *et al.*, "Microaxial Flow Pump or Standard Care in Infarct-Related Cardiogenic Shock," *New England Journal of Medicine*, vol. 390, no. 15, pp. 1382–1393, Apr. 2024, doi: 10.1056/NEJMoa2312572.

[28]. V. T. Nkomo, J. M. Gardin, T. N. Skelton, J. S. Gottdiener, C. G. Scott, and M. Enriquez-Sarano, "Burden of valvular heart diseases: a population-based study," *Lancet*, vol. 368, no. 9540, pp. 1005–1011, Sep. 2006, doi: 10.1016/S0140-6736(06)69208-8.

[29]. S. Douedi and H. Douedi, "Mitral Regurgitation," in *StatPearls*, Treasure Island (FL): StatPearls Publishing, 2023. Accessed: Oct. 29, 2023. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK553135/

[30]. F. N. Delling and R. S. Vasan, "Epidemiology and Pathophysiology of Mitral Valve Prolapse," *Circulation*, vol. 129, no. 21, pp. 2158–2170, May 2014, doi: 10.1161/CIRCULATIONAHA.113.006702.

[31]. "2021 ESC/EACTS Guidelines for the management of valvular heart disease | European Heart Journal | Oxford Academic." Accessed: Oct. 20, 2024. [Online]. Available: https://academic-oup-com.qulib.idm.oclc.org/eurheartj/article/43/7/561/6358470

[32]. S. N. Shah and S. Sharma, "Mitral Stenosis," in *StatPearls*, Treasure Island (FL): StatPearls Publishing, 2023. Accessed: Oct. 29, 2023. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK430742/

[33]. [C. R. Thompson *et al.*, "Cardiogenic shock due to acute severe mitral regurgitation complicating acute myocardial infarction: a report from the SHOCK Trial Registry," *Journal of the American College of Cardiology*, vol. 36, no. 3, Supplement 1, pp. 1104–1109, Sep. 2000, doi: 10.1016/S0735-1097(00)00846-9.

[34]. C. M. Otto *et al.*, "2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines," *Circulation*, vol. 143, no. 5, pp. e72–e227, Feb. 2021, doi: 10.1161/CIR.0000000000000923.

[35]. Ananthakrishna Pillai, C. Ramasamy, S. G. V, and H. Kottyath, "Outcomes following balloon mitral valvuloplasty in pregnant females with mitral stenosis and significant sub valve disease with severe decompensated heart failure," *J Interv Cardiol*, vol. 31, no. 4, pp. 525–531, Aug. 2018, doi: 10.1111/joic.12507.

[36]. "Percutaneous Repair or Medical Treatment for Secondary Mitral Regurgitation | New England Journal of Medicine." Accessed: Oct. 20, 2024. [Online]. Available: https://www-nejm-org.qulib.idm.oclc.org/doi/full/10.1056/NEJMoa1805374

[37]. G. W. Stone *et al.*, "Transcatheter Mitral-Valve Repair in Patients with Heart Failure," *New England Journal of Medicine*, vol. 379, no. 24, pp. 2307–2318, Dec. 2018, doi: 10.1056/NEJMoa1806640.

[38]. R. Kaddoura and M. Al-Hijji, "Transcatheter Mitral Valve Repair in Acute and Critical Cardiac Conditions," *Heart Views : The Official Journal of the Gulf Heart Association*, vol. 24, no. 1, p. 29, Feb. 2023, doi: 10.4103/heartviews.heartviews_73_22.

[39]. Kosaraju, V. S. Pendela, and O. Hai, "Cardiogenic Shock," in *StatPearls*, Treasure Island (FL): StatPearls Publishing, 2023. Accessed: Oct. 29, 2023. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK482255/

[40]. Y. Topilsky, "Mitral Regurgitation: Anatomy, Physiology, and Pathophysiology—Lessons Learned From Surgery and Cardiac Imaging," *Front Cardiovasc Med*, vol. 7, p. 84, May 2020, doi: 10.3389/fcvm.2020.00084.

[41]. Shankar *et al.*, "A Clinical Update on Vasoactive Medication in the Management of Cardiogenic Shock," *Clin Med Insights Cardiol*, vol. 16, p. 11795468221075064, Feb. 2022, doi: 10.1177/11795468221075064.

[42]. Singh, S. Laribi, J. R. Teerlink, and A. Mebazaa, "Agents with vasodilator properties in acute heart failure," *Eur Heart J*, vol. 38, no. 5, pp. 317–325, Feb. 2017, doi: 10.1093/eurheartj/ehv755.

[43]. K. Uhlig *et al.*, "Inotropic agents and vasodilator strategies for the treatment of cardiogenic shock or low cardiac output syndrome," *Cochrane Database Syst Rev*, vol. 11, no. 11, p. CD009669, Nov. 2020, doi: 10.1002/14651858.CD009669.pub4.

[44]. A. den Uil *et al.*, "Short-term mechanical circulatory support as a bridge to durable left ventricular assist device implantation in refractory cardiogenic shock: a systematic review and meta-analysis," *Eur J Cardiothorac Surg*, vol. 52, no. 1, pp. 14–25, Jul. 2017, doi: 10.1093/ejcts/ezx088.

[45]. J. L. Peura *et al.*, "Recommendations for the use of mechanical circulatory support: Device strategies and patient selection: A scientific statement from the American heart association," *Circulation*, vol. 126, no. 22, pp. 2648–2667, 2012, doi: 10.1161/CIR.0b013e3182769a54.

[46]. J. R. Kimman *et al.*, "Mechanical Support in Early Cardiogenic Shock: What Is the Role of Intra-aortic Balloon Counterpulsation?," *Curr Heart Fail Rep*, vol. 17, no. 5, pp. 247–260, Oct. 2020, doi: 10.1007/s11897-020-00480-0.

[47]. S. Imaoka *et al.*, "Impella Support as a Bridge to Surgery for Severe Mitral Regurgitation With Cardiogenic Shock," *Circ Rep*, vol. 3, no. 3, pp. 178–181, doi: 10.1253/circrep.CR-21-0016.

[48]. "Left Impella®-device as bridge from cardiogenic shock with acute, severe mitral regurgitation to MitraClip®-procedure: a new option for critically ill patients - PubMed." Accessed: Oct. 20, 2024. [Online]. Available: https://pubmed-ncbi-nlm-nih-gov.qulib.idm.oclc.org/33620436/

[49]. P. Villablanca *et al.*, "Mechanical Circulatory Support in Cardiogenic Shock due to Structural Heart Disease," *Interv Cardiol Clin*, vol. 10, no. 2, pp. 221–234, Apr. 2021, doi: 10.1016/j.iccl.2020.12.007.

[50]. S. S. Goel *et al.*, "Prevalence and outcomes of unoperated patients with severe symptomatic mitral regurgitation and heart failure: comprehensive analysis to determine the potential role of MitraClip for this unmet need," *J Am Coll Cardiol*, vol. 63, no. 2, pp. 185–186, Jan. 2014, doi: 10.1016/j.jacc.2013.08.723.

[51]. S. V. Arnold *et al.*, "Health Status After Transcatheter Mitral-Valve Repair in Heart Failure and Secondary Mitral Regurgitation: COAPT Trial," *Journal of the American College of Cardiology*, vol. 73, no. 17, pp. 2123–2132, May 2019, doi: 10.1016/j.jacc.2019.02.010.

[52]. P. Kovach, S. Bell, A. Kataruka, M. Reisman, and C. Don, "Outcomes of urgent/emergent transcatheter mitral valve repair (MitraClip): A single center experience," *Catheter Cardiovasc Interv*, vol. 97, no. 3, pp. E402–E410, Feb. 2021, doi: 10.1002/ccd.29084.

[53]. S. Aldrugh, N. Kakouros, and W. Qureshi, "National prevalence and outcomes of different mitral valve interventions for mitral regurgitation among patients with cardiogenic shock: an analysis of the national readmission database 2010–2018," *European Heart Journal*, vol. 42, no. Supplement_1, p. ehab724.2212, Oct. 2021, doi: 10.1093/eurheartj/ehab724.2212.

[54]. C.-W. Lee *et al.*, "Feasibility of the transcatheter mitral valve repair for patients with severe mitral regurgitation and endangered heart failure," *Journal of the Formosan Medical Association*, vol. 120, no. 1, Part 2, pp. 452–459, Jan. 2021, doi: 10.1016/j.jfma.2020.04.035.

[55]. R. Estévez-Loureiro *et al.*, "Use of MitraClip for mitral valve repair in patients with acute mitral regurgitation following acute myocardial infarction: Effect of cardiogenic shock on outcomes (IREMMI Registry)," *Catheter Cardiovasc Interv*, vol. 97, no. 6, pp. 1259–1267, May 2021, doi: 10.1002/ccd.29552.

[56]. Haberman *et al.*, "Safety and Feasibility of MitraClip Implantation in Patients with Acute Mitral Regurgitation after Recent Myocardial Infarction and Severe Left Ventricle Dysfunction," *J Clin Med*, vol. 10, no. 9, p. 1819, Apr. 2021, doi: 10.3390/jcm10091819.

[57]. K. Flint, A. Brieke, D. Wiktor, and J. Carroll, "Percutaneous edge-to-edge mitral valve repair may rescue select patients in cardiogenic shock: Findings from a single center case series," *Catheter Cardiovasc Interv*, vol. 94, no. 2, pp. E82–E87, Aug. 2019, doi: 10.1002/ccd.28089.

[58]. G. H. L. Tang, R. Estevez-Loureiro, Y. Yu, J. B. Prillinger, S. Zaid, and M. A. Psotka, "Survival Following Edge-to-Edge Transcatheter Mitral Valve Repair in Patients With Cardiogenic Shock: A Nationwide Analysis," *J Am Heart Assoc*, vol. 10, no. 8, p. e019882, Apr. 2021, doi: 10.1161/JAHA.120.019882.

[59]. C.-Y. So *et al.*, "Transcatheter Edge-to-Edge Repair for Acute Mitral Regurgitation With Cardiogenic Shock Secondary to Mechanical Complication," *Cardiovasc Revasc Med*, vol. 45, pp. 44–50, Dec. 2022, doi: 10.1016/j.carrev.2022.07.003.

[60]. G. Falasconi *et al.*, "Use of edge-to-edge percutaneous mitral valve repair for severe mitral regurgitation in cardiogenic shock: A multicenter observational experience (MITRA-SHOCK study)," *Catheter Cardiovasc Interv*, vol. 98, no. 1, pp. E163–E170, Jul. 2021, doi: 10.1002/ccd.29683.

[61]. "Percutaneous Mitral Repair as Salvage Therapy in Patients With Mitral Regurgitation and Refractory Cardiogenic Shock - PubMed." Accessed: Oct. 20, 2024. [Online]. Available: https://pubmed-ncbi-nlm-nih-gov.qulib.idm.oclc.org/31694413/

[62]. R. Cheng *et al.*, "Percutaneous Mitral Repair for Patients in Cardiogenic Shock Requiring Inotropes and Temporary Mechanical Circulatory Support," *JACC Cardiovasc Interv*, vol. 12, no. 23, pp. 2440–2441, Dec. 2019, doi: 10.1016/j.jcin.2019.05.042.

[63]. S. Garcia *et al.*, "Percutaneous Mitral Valve Repair With MitraClip in Inoperable Patients With Severe Mitral Regurgitation Complicated by Cardiogenic Shock," *J Invasive Cardiol*, vol. 32, no. 6, pp. 228–231, Jun. 2020.

[64]. R. G. Jung *et al.*, "Transcatheter Mitral Valve Repair in Cardiogenic Shock and Mitral Regurgitation: A Patient-Level, Multicenter Analysis," *JACC Cardiovasc Interv*, vol. 14, no. 1, pp. 1–11, Jan. 2021, doi: 10.1016/j.jcin.2020.08.037.

[65]. S. Parlow *et al.*, "Transcatheter mitral valve repair for inotrope dependent cardiogenic shock - Design and rationale of the CAPITAL MINOS trial," *Am Heart J*, vol. 254, pp. 81–87, Dec. 2022, doi: 10.1016/j.ahj.2022.08.008.

[66]. C. Fraccaro *et al.*, "Transcatheter interventions for left-sided valvular heart disease complicated by cardiogenic shock: a consensus statement from the European Association of Percutaneous Cardiovascular Interventions (EAPCI) in collaboration with the Association for Acute Cardiovascular Care (ACVC) and the ESC Working Group on Cardiovascular Surgery," *EuroIntervention*, vol. 19, no. 8, pp. 634–651, Oct. 2023, doi: 10.4244/EIJ-D-23-00473.

[67]. S. A. Al-Asmi *et al.*, "Transcatheter Mitral Valve Repair with MitraClip®: A Nationwide Experience," *Heart Views*, vol. 24, no. 4, pp. 179–187, 2023, doi: 10.4103/heartviews.heartviews_90_23.

[68]. M. Orban *et al.*, "Transcatheter edge-to-edge repair for secondary mitral regurgitation with third-generation devices in heart failure patients – results from the Global EXPAND Post-Market study," *European Journal of Heart Failure*, vol. 25, no. 3, pp. 411–421, 2023, doi: 10.1002/ejhf.2770.

[69]. R. S. von Bardeleben *et al.*, "Real-World Outcomes of Fourth-Generation Mitral Transcatheter Repair: 30-Day Results From EXPAND G4," *JACC Cardiovasc Interv*, vol. 16, no. 12, pp. 1463–1473, Jun. 2023, doi: 10.1016/j.jcin.2023.05.013.

[70]. B. Levy, J. Buzon, and A. Kimmoun, "Inotropes and vasopressors use in cardiogenic shock: when, which and how much?," *Current Opinion in Critical Care*, vol. 25, no. 4, p. 384, Aug. 2019, doi: 10.1097/MCC.0000000000000632.

[71]. J. E. Bloom, W. Chan, D. M. Kaye, and D. Stub, "State of Shock: Contemporary Vasopressor and Inotrope Use in Cardiogenic Shock," *Journal of the American Heart Association*, vol. 12, no. 15, p. e029787, Aug. 2023, doi: 10.1161/JAHA.123.029787.

[72]. V. Atti *et al.*, "A Comprehensive Review of Mechanical Circulatory Support Devices," *Heart Int*, vol. 16, no. 1, pp. 37–48, Mar. 2022, doi: 10.17925/HI.2022.16.1.37.

[73]. B. Worku, A. R. de Biasi, I. Gulkarov, S.-C. Wong, and A. Salemi, "Transapical Mitral Valve-In-Valve Implantation for Patients in Cardiogenic Shock," *The Annals of Thoracic Surgery*, vol. 99, no. 5, pp. e103–e105, May 2015, doi: 10.1016/j.athoracsur.2015.01.046.

[74]. M. Litmanovitch, G. M. Joynt, J. Skoularigis, and J. Lipman, "Emergency percutaneous balloon mitral valvotomy in a patient with septic shock," *Chest*, vol. 108, no. 2, pp. 570–572, Aug. 1995, doi: 10.1378/chest.108.2.570.

[75]. W. H. Chow and T. C. Chow, "Percutaneous balloon mitral valvotomy as a bridge to elective mitral valve replacement," *Cathet Cardiovasc Diagn*, vol. 45, no. 1, p. 102, Sep. 1998, doi: 10.1002/(sici)1097-0304(199809)45:1<102::aid-ccd26>3.0.co;2-o.

[76]. J. S. Dugal, V. Jetley, J. S. Sabharwal, S. Sofat, and C. Singh, "Life-saving PTMC for critical calcific mitral stenosis in cardiogenic shock with balloon impasse," *Int J Cardiovasc Intervent*, vol. 5, no. 3, pp. 172–174, 2003.

[77]. S. Strick *et al.*, "[Emergent percutaneous mitral valve repair with Inoue balloon-catheter in severe mitral stenosis and cardiogenic shock]," *Dtsch Med Wochenschr*, vol. 119, no. 33, pp. 1110–1114, Aug. 1994, doi: 10.1055/s-2008-1058810.

[78]. M. Notrica *et al.*, "Life-saving Percutaneous Mitral Valvuloplasty on a Pregnant Woman with Refractory Cardiogenic Shock," *Heart, Lung and Circulation*, vol. 18, no. 4, pp. 301–304, Aug. 2009, doi: 10.1016/j.hlc.2007.12.008.

[79]. J. Endrys, A. G. Habashy, and N. Hayat, "Life-saving balloon mitral valvuloplasty in patient with cardiogenic shock after cardiac arrest," *J Invasive Cardiol*, vol. 13, no. 11, pp. 752–754, Nov. 2001.

About the Author (s)

Dr. Ahmed completed his medical degree at Omdurman Islamic University (OIU) Faculty of Medicine in Sudan, followed by four years of residency training at Hamad Corporation, a Cornell-affiliated institution in Qatar. Currently, he is an Internal Medicine resident at BPTHOSP, with a keen interest in Cardiology. In addition to his residency, Dr. Ahmed contributes as a teaching assistant at the Harvard T.H. Chan School of Public Health. His dedication to research and excellence in the field was recognized with the Paul Dudley White International Award at the 2021 AHA scientific sessions.