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Cardiogenic Shock: Integrating Pathophysiology, Phenotypes, and Advanced Therapeutic Approaches

Mubariz Ahmed Hassan[1*], Ikram Ahmed Rana[2]

[1]Howard University Hospital, Washington, DC, USA

[2]Tahir Heart Institute, Punjab, Pakistan

***Corresponding author:** Mubariz Ahmed Hassan, Howard University Hospital, Washington, DC, USA

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Abstract

Cardiogenic shock (CS) is a critical and hemodynamically intricate condition characterized by a wide range of causes and clinical manifestations. Despite advancements in treatment, CS continues to exhibit high morbidity and mortality rates, typically between 35% and 50%. Recent observational studies have focused on improving early detection and management of CS through standardized team-based protocols, comprehensive hemodynamic profiling, and the strategic use of temporary mechanical circulatory support (tMCS) devices. This narrative review explores the pathophysiology of CS, identifies emerging phenotypes, examines evolving definitions and staging systems, and evaluates current pharmacologic and device-based treatments. Additionally, it highlights the importance of standardized team-based management protocols and regionalized systems-of-care in enhancing patient outcomes in CS.

Introduction

Cardiogenic shock (CS) is a complex, multifactorial syndrome marked by severe and unrelenting circulatory failure due to compromised myocardial contractility. This results in systemic hypoperfusion, metabolic acidosis, and multiorgan dysfunction. Despite over twenty years of advancements in interventional techniques, the introduction of quickly deployable temporary mechanical circulatory support (tMCS) devices, and comprehensive systems-of-care strategies for acute myocardial infarction (AMI), outcomes for CS patients remain poor, with 30-day mortality rates ranging from 30% to 50% and significant multiorgan complications [1]. The groundbreaking Should We Emergently Revascularize Occluded Arteries in Cardiogenic Shock (SHOCK) trial in 1999 established that early revascularization can enhance survival rates. Although randomized controlled trials (RCTs) have not definitively proven the benefits of pharmacologic or device-based interventions, emerging observational data from North American CS registries emphasize the value of standardized, team-based care [2]. These findings suggest that implementing a multidisciplinary "Shock team," utilizing early pulmonary artery catheter (PAC) hemodynamic monitoring, and applying selective, tailored tMCS within a comprehensive care framework at an American Heart Association (AHA) Level 1 cardiac intensive care unit (CICU) can lower in-hospital mortality [2]. The diversity in clinical presentations, hemodynamic disturbances, treatment strategies, and outcomes underscores the inadequacy of a uniform management approach for CS. This narrative review explores the pathophysiology of CS, its phenotypes, evolving definitions for risk stratification, current pharmacologic and device-based therapies, and potential treatment protocols and systems-of-care strategies to improve outcomes. Finally, it identifies opportunities for further research to address existing knowledge gaps in this condition.

Discussion

Pathophysiology of Cardiogenic Shock

Cardiogenic shock (CS) is a complex and vicious cycle that often culminates in multiorgan failure and death. It is initiated by a progressive impairment in ventricular contractility, leading to a critical reduction in mean arterial pressure (MAP) and cardiac output (CO). This reduction results in systemic hypoperfusion and decreased coronary perfusion pressure. In response, baroreceptors

and chemoreceptors are activated to maintain hemodynamics and perfusion [3].

Epidemiology of Cardiogenic Shock

CS is the most common type of shock in patients admitted to cardiac intensive care units (CICU). Historically seen as primarily caused by left ventricular (LV) dysfunction from acute myocardial infarction (AMI), recent years have recognized acute decompensation of chronic HF as the most common underlying etiology, contributing to over 50% of CS admissions [3]. Increased use of preventive therapies, early revascularization strategies, and improved AMI survival have led to more survivors with chronic HF due to LV dysfunction. The number of hospitalizations for CS has increased more than threefold from 2004 to 2018, with a 25% reduction in in-hospital mortality from 49% to 37% [4].

Hemodynamic Changes

The activation of baroreceptors and chemoreceptors causes arterial and veno- constriction, leading to increased vascular resistance. Blood is redistributed away from the splanchnic circulation, elevating pulmonary venous and central venous pressure (CVP).

These mechanisms result in multiorgan congestion, often exacerbating preexisting volume overload seen in patients with heart failure (HF). This further compromises end-organ perfusion, represented by worsening lactic acidemia [5].

In response to tissue ischemia, a state of systemic inflammatory response syndrome (SIRS) ensues. SIRS leads to systemic vasodilation and inflammation in an already dysfunctional myocardium, propagating the progressive maladaptive spiral of CS [5].

Etiologies of Cardiogenic Shock

The two most commonly recognized etiologies of CS are acute myocardial infarction- related cardiogenic shock (AMI-CS) and heart failure-related cardiogenic shock (HF-CS) [6].

AMI-CS

Typically associated with injury to more than 40% of the left ventricular (LV) myocardium but can also be precipitated by mechanical complications such as ventricular septal defect and free wall or papillary muscle rupture.

Analysis of pressure-volume loop curves in AMI-CS shows a rightward and downward shift of the end-systolic pressure volume relationship, suggesting a sudden reduction in LV contractility. This results in reduced stroke volume (SV), CO, and MAP, and increases in pulmonary capillary wedge pressure (PCWP) and CVP [7].

The hemodynamic changes reflect the canonical clinical course for patients with AMI-CS, often beginning with hypotension from the acute ischemic insult leading to hypoperfusion and culminating with congestion [8].

HF-CS

It often follows a more indolent clinical course compared to AMI-CS. It usually presents with congestion in acute on chronic HF-CS phenotypes, leading to hypoperfusion and ending with systemic hypotension [9].

Interplay of Pulmonary Artery Pulsatility Index and Cardiac Power Output

The Pulmonary Artery Pulsatility Index (PAPi) and Cardiac Power Output (CPO) are crucial parameters in understanding the hemodynamic profile and severity of CS [9].

Pulmonary Artery Pulsatility Index (PAPi)

PAPi is calculated as (Pulmonary Artery Systolic Pressure - Pulmonary Artery Diastolic Pressure) / Right Atrial Pressure.

A lower PAPi indicates poor right ventricular function and is often associated with worse outcomes in CS. It helps in differentiating between left ventricular (LV) and right ventricular (RV) failure [10]. PAPi can guide the selection and escalation of mechanical circulatory support (MCS) devices, particularly in the context of RV dysfunction.

Cardiac Power Output (CPO)

CPO is calculated as (MAP × CO) / 451

CPO is a direct measure of cardiac function and is considered a strong predictor of mortality in CS. A lower CPO reflects severe cardiac dysfunction and correlates with higher mortality [10]. CPO can be used to assess the effectiveness of therapeutic interventions and the need for escalation to advanced therapies.

Integrating PAPi and CPO in CS Management

Understanding the interplay between PAPi and CPO is essential in the management of CS. Patients with low CPO and low PAPi are at a particularly high risk and may benefit from early and aggressive intervention. These parameters can help tailor the use of vasopressors, inotropes, and MCS devices to optimize hemodynamics and improve outcomes [11].

Early Recognition and Intervention

PAPi and RV Dysfunction: Low PAPi should prompt consideration of therapies targeting RV support, such as inotropic support or RV-assist devices.

CPO and Global Cardiac Function: Low CPO indicates the need for interventions to improve overall cardiac output, such as the use of inotropes or LV-assist devices [12].

Therapeutic Strategies

The choice of MCS devices, such as Impella, Intra-Aortic Balloon Pump (IABP), or Extracorporeal Membrane Oxygenation (ECMO), can be guided by PAPi and CPO values [12]. Continuous monitoring of PAPi and CPO allows for dynamic adjustment of therapies to respond to the evolving clinical condition of the patient.

Prognostic Value

Both PAPi and CPO are valuable for prognostication in CS. Low values are associated with higher mortality and can help identify patients who may need advanced therapies or palliative care. Incorporating these hemodynamic parameters into the management of CS provides a more nuanced and individualized approach, potentially improving outcomes in this critically ill population [13].

Novel Phenotypes and Evolving Definitions

CS has been traditionally categorized by LV dysfunction, but recent studies have identified HF-CS as a distinct etiology. The Society for Cardiovascular Angiography and Interventions (SCAI) proposed a five-stage classification system (A to E) in 2019, encompassing the full spectrum of the syndrome based on physical examination findings, laboratory markers, and invasive hemodynamics [14]. The SCAI classification system has undergone retrospective and prospective validation, emphasizing the presence of cardiac arrest with coma as an adverse effect modifier, dynamic baseline and maximum SCAI staging, and serial re-staging to stratify risk.

SCAI Stage A: "At Risk"

This stage includes patients who are not currently in shock but are at high risk of developing CS due to underlying cardiac conditions. Patients may have stable hemodynamics and are typically asymptomatic but have conditions that predispose them to CS, such as acute myocardial infarction (AMI), heart failure (HF), or post-cardiac surgery status [10].

SCAI Stage B: "Beginning"

Patients in this stage exhibit early signs of hemodynamic instability but are not yet in overt shock. This stage is marked by hypotension (systolic blood pressure < 90 mmHg or mean arterial pressure < 65 mmHg) or mild hypoperfusion, which may include signs such as cool extremities, decreased urine output, and altered mental status. These patients are often normotensive due to compensatory mechanisms [10].

SCAI Stage C: "Classic"

This stage represents classic CS, with evident hemodynamic instability and hypoperfusion. Patients typically present with hypotension that requires pharmacologic support (e.g., vasopressors or inotropes) to maintain adequate perfusion. Clinical signs include cold, clammy skin, oliguria, and altered mental status. Hemodynamic monitoring often reveals low cardiac output and elevated filling pressures [15].

SCAI Stage D: "Deteriorating"

Patients in this stage are experiencing worsening shock despite initial therapeutic interventions. There is persistent hypotension and hypoperfusion despite the use of vasopressors and inotropes, and/or mechanical circulatory support (MCS) [15]. This stage is marked by increasing lactate levels, worsening acidosis, and signs of multiorgan failure.

SCAI Stage E: "Extremis"

This stage represents the most severe form of CS, where patients are in refractory shock and at imminent risk of death. These patients exhibit profound hemodynamic collapse and hypoperfusion, often requiring maximal pharmacologic and mechanical support. They may have severe lactic acidosis, anuria, and altered consciousness or coma [16]. The prognosis is extremely poor without immediate and aggressive intervention.

Acute Myocardial Infarction-Related Cardiogenic Shock (AMI-CS)

AMI-CS is associated with injury to over 40% of the LV myocardium but can also be caused by mechanical complications such as ventricular septal defect and free wall or papillary muscle rupture. The SHOCK Trial in 1999 demonstrated a 13% absolute reduction in all- cause mortality at one year in patients undergoing revascularization [15]. The Feedback Intervention and Treatment Times in ST-Elevation Myocardial Infarction (FITT-STEMI) trial reinforced the importance of timely invasive reperfusion, showing that every 10-minute delay in treatment was associated with three additional deaths per 100 patients with AMI- CS undergoing percutaneous coronary intervention (PCI) [17].

Emergency Department Care

Prompt recognition of CS by emergency medical services (EMS) personnel and emergency department providers is crucial. Steps to expedite care include early 12-lead electrocardiogram acquisition, administration of vasopressors to achieve MAP >65 mm Hg, mechanical ventilation, point-of-care echocardiography to assess for mechanical complications, and immediate transfer to a primary PCI-capable facility [18]. SCAI Stage C or D patients may require adjunctive stabilization measures while mitigating significant

delays to invasive reperfusion.

Best Practices for Vascular Access

Transradial access is the default approach for coronary angiography and PCI due to reductions in major bleeding and vascular complications compared to the femoral approach. However, AMI-CS is a predictor of transradial access failure due to systemic constriction and vasoactive therapies. If radial access is challenging or tMCS is required, safe femoral access using multimodality imaging techniques should be employed [18]. Core elements of "vascular safety bundles" include ultrasound and fluoroscopic guided micropuncture access, pre-and post-procedure run-off angiography, and validated hemostatic protocols.

Antithrombotic Therapy

Antithrombotic therapy in CS is challenging due to impaired absorption of oral P2Y12 inhibitors, platelet dysfunction, impaired clopidogrel activation, and bleeding and vascular complications. The AHA Position Statement and European guidelines recommend unfractionated heparin as the anticoagulant of choice due to its rapid offset and reversibility. Dual antiplatelet therapy with aspirin and oral P2Y12 inhibitors, such as prasugrel and ticagrelor, is the mainstay of contemporary antiplatelet therapy in AMI-CS. In cases with limited oral bioavailability, intravenous P2Y12 inhibitor cangrelor may be used [17].

Revascularization Strategy

Up to 80% of patients with AMI-CS have multivessel coronary artery disease (CAD). The Culprit Lesion Only PCI vs. Multivessel PCI in Cardiogenic Shock (CULPRIT-SHOCK) trial demonstrated a 17% absolute reduction in 30-day death or renal replacement therapy with culprit-vessel PCI [15]. However, the applicability of these findings to real-world practice is challenged due to variations in the use of tMCS and other factors. Current US and European guidelines recommend against routine multivessel PCI in AMI-CS based on the CULPRIT-SHOCK findings [15].

Heart Failure-Related Cardiogenic Shock (HF-CS)

HF-CS differs from AMI-CS in pathophysiology, clinical presentation, and management. Chronic HF progresses to HF-CS through worsening hypoperfusion, acute on chronic hepatic and renal injury, lactic acidosis, and systemic inflammatory response. Elevated filling pressures are a strong predictor of adverse outcomes [19]. For patients failing initial therapeutic interventions, a selective and tailored approach to tMCS device selection is recommended.

CICU Management of Cardiogenic Shock

Team-based interventions have shown improvements in outcomes for high-mortality conditions such as trauma, cardiac arrest, sepsis, and stroke. The implementation of "Shock teams" in CS management has been associated with improved short-term outcomes. The Critical Care Cardiology Trials Network (CCCTN) demonstrated favorable outcomes with standardized team-based approaches to CS [20]. The 2022 AHA/ACC/HFSA HF guidelines also recommend the use of multidisciplinary shock teams in CS management [17].

Role of Vasopressors and Inotropes

The acute management of cardiogenic shock (CS) often requires intravenous vasopressors and inotropes to enhance cardiac output. Norepinephrine is typically the first-line agent, showing superiority over dopamine in reducing deaths within CS subgroups. Epinephrine, however, has been linked to higher incidences of refractory AMI-CS [10]. Studies comparing milrinone to placebo and dobutamine found no significant differences in key outcomes, although both drugs can increase myocardial oxygen demand and risk of arrhythmias. Observational data indicate that using multiple vasoactive agents is associated with worse mortality, emphasizing the need to minimize their number and duration [9]. Clinical guidelines recommend vasopressors and/or inotropes for CS management. Selection should consider effects on right and left ventricular profiles to optimize treatment and improve survival, aiming to balance myocardial support with minimizing adverse effects.

Invasive Hemodynamic Monitoring

Invasive hemodynamic monitoring, particularly using pulmonary artery catheters (PACs), is increasingly recommended in managing heart failure-related cardiogenic shock (HF-CS). Despite early trials like ESCAPE showing no benefit, recent studies suggest PACs help identify CS phenotypes and tailor therapies, improving outcomes [5]. Data from the National Inpatient Sample and contemporary studies demonstrate regional variations in PAC use, but consistent findings show its association with reduced mortality, especially when used early. Guidelines now support PACs for selected HF patients with worsening symptoms and signs of end-organ perfusion. PAC monitoring allows for timely diagnosis, optimal use of mechanical circulatory support (MCS), and informed decision-making on therapy escalation or de-escalation [5]. Current and future trials aim to confirm PAC benefits in CS, particularly in early and routine use, to better define its role in improving survival and guiding therapy in complex cases.

Temporary Mechanical Circulatory Support Devices

Temporary mechanical circulatory support (tMCS) devices are widely used in managing cardiogenic shock (CS), with increasing utilization seen globally. Despite mixed results from randomized controlled trials (RCTs), observational studies suggest that tMCS, when used selectively and based on time-sensitive protocols,

can improve outcomes. Devices like intra-aortic balloon pumps (IABP), Impella, TandemHeart, and extracorporeal membrane oxygenation (ECMO) each offer unique benefits and risks [12]. IABP provides counterpulsation support, reducing left ventricular afterload, while Impella offers significant hemodynamic support but is associated with higher bleeding risks.

TandemHeart supports both left and right sides via a transseptal approach, and ECMO provides gas exchange and hemodynamic support [18]. Ongoing trials aim to clarify the optimal timing and use of these devices in CS. Clinical guidelines emphasize an algorithmic approach to tMCS, incorporating interdisciplinary collaboration and serial hemodynamic assessments to tailor therapy and improve patient outcomes.

Use of Intra-Aortic Balloon Pump (IABP)

The intra-aortic balloon pump (IABP) supports cardiac function using counterpulsation. During diastole, the balloon inflates, enhancing aortic root diastolic pressure and coronary perfusion, while deflation during systole reduces left ventricular (LV) afterload, lowering cardiac work and oxygen consumption. Though the IABP-SHOCK II trial found no survival benefit in AMI-CS patients, IABPs show promise in heart failure-related CS (HF-CS), aiding in bridging to durable LVAD or heart transplantation [21]. Non-randomized studies suggest IABP benefits, particularly in patients with non-ischemic cardiomyopathy and higher pulmonary artery pulsatility index (PAPi) scores. Predictors of positive IABP response include high systemic vascular resistance (SVR) and low cardiac index at baseline. The ongoing AltShock-2 trial aims to compare IABP with vasoactive therapy in HF-CS patients to address existing knowledge gaps and optimize treatment strategies [15].

Percutaneous Ventricular Assist Device (pVAD)

Impella percutaneous ventricular assist devices (pVADs) from Abiomed Inc. are catheter- based devices providing up to 5.5 liters of cardiac output. These devices displace blood from the left ventricle to the aorta, reducing LV preload and oxygen consumption while enhancing mean arterial pressure and tissue perfusion. Despite FDA approval for temporary support, randomized data on their efficacy in cardiogenic shock (CS) remain limited. Studies like ISAR-SHOCK and IMPRESS-in-Severe-SHOCK show improvements in cardiac index but not 30-day mortality reduction compared to intra-aortic balloon pumps (IABP) [17]. Meta-analyses indicate no significant mortality difference between Impella and IABP but highlight higher bleeding risks with pVADs [15]. The RECOVER IV trial explores early Impella support in STEMI patients with CS. While Impella devices offer substantial hemodynamic support, more clinical data is needed to define their role in optimizing outcomes and informing guidelines for appropriate patient selection and device utilization.

Extracorporeal Membrane Oxygenation (ECMO)

Extracorporeal membrane oxygenation (ECMO) supports patients with severe cardiac and respiratory failure. Configurations include veno-venous (VV-ECMO) for gas exchange and veno-arterial (VA-ECMO) for combined gas exchange and hemodynamic support. VA- ECMO, inserted percutaneously, returns oxygenated blood to a central artery, requiring systemic anticoagulation due to large cannula size [16]. Despite high complication rates, VA-ECMO is used as a first-line strategy in cardiogenic shock (CS). Trials like ECMO-CS and EURO SHOCK have not demonstrated significant mortality benefits, showing high rates of serious adverse events [21]. Optimal VA-ECMO use in acute myocardial infarction-related CS (AMI-CS) remains unclear. VA-ECMO increases left ventricular (LV) afterload, leading to LV distension and complications, requiring strategies like reduced ECMO flow or left ventricular venting [12]. Further research is needed to determine VA-ECMO's role and improve patient outcomes in CS management.

Left Ventricular Venting During ECMO

Left ventricular (LV) venting is crucial during veno-arterial extracorporeal membrane oxygenation (VA-ECMO) to address increased LV afterload. Retrograde flow from ECMO raises LV end-diastolic pressure, leading to distension, higher oxygen demand, and reduced LV function. This can cause pulmonary edema and systemic hypoxia, complicating recovery, especially in acute myocardial infarction-related cardiogenic shock (AMI-CS) [7]. Various strategies, including using intra-aortic balloon pumps (IABP) for passive venting or Impella devices for active unloading, mitigate these effects. Surgical options like CentriMag pumps provide full univentricular or biventricular support, venting the LV through the left atrium or apex. Effective LV venting reduces afterload, improves coronary perfusion, and enhances patient outcomes during ECMO support [15]. Tailoring these strategies to individual patient needs is essential for optimal management in CS.

Role of Anticoagulation and Anti-Thrombotic Therapy

Anticoagulation is essential in managing temporary mechanical circulatory support (tMCS) devices to prevent thrombosis and embolization due to shear stress and foreign materials. Devices like VA-ECMO, IABP, and Impella require systemic anticoagulation, typically with unfractionated heparin (UFH) [17]. Monitoring challenges arise due to comorbidities and multi-organ dysfunction, making parallel anti-Xa and aPTT assessments preferable. Direct thrombin inhibitors like bivalirudin or argatroban are alternatives for patients with heparin- induced thrombocytopenia. Studies highlight higher mortality and bleeding risks with microaxial

flow pumps, underscoring the need for optimal anticoagulation strategies. Dual antiplatelet therapy (DAPT) in acute coronary syndrome post-PCI and acquired von Willebrand syndrome increases bleeding risks. Given the presence of comorbidities and multi-organ dysfunction in critically ill patients, activated partial thromboplastin time (aPTT) alone may not accurately assess anticoagulant effect. Thus, monitoring both anti-Xa levels and aPTT in parallel is deemed superior [3]. This dual approach mitigates the influence of confounding factors that affect aPTT readings. Supporting studies have demonstrated that mortality rates increase when aPTT and anti-Xa values diverge, underscoring the importance of concurrent monitoring to ensure precise and effective anticoagulation management in these patients [15].

Long-Term Outcomes

Long-term outcomes for cardiogenic shock (CS) survivors are increasingly studied, revealing significant challenges but also potential for meaningful recovery. Despite high in- hospital mortality, those discharged show variable intermediate and long-term survival rates. Studies report 16% readmission within a year, with infections and acute decompensated heart failure being common causes. The SHOCK trial indicated 87% of one-year survivors had favorable functional status [7]. Research emphasizes the need for structured follow-up, addressing functional status, quality of life, and readmission prevention. Trials like HALO-Shock aim to explore remote hemodynamic monitoring to improve post-discharge outcomes, highlighting the importance of continuous care and early intervention for CS survivors [9].

Palliative and Hospice Care Integration

Palliative care is crucial in managing cardiogenic shock (CS), focusing on symptom control and quality of life, complementing curative treatments. Despite high mortality rates, palliative care utilization in CS remains low, with only 9% of patients receiving such services [15]. Studies show palliative care is associated with lower readmission rates and hospitalization costs, benefiting both patients and healthcare systems. It provides emotional, social, and spiritual support, improving patient and family experiences.

Hospice care, typically for end-of-life stages, is less frequently employed but necessary for terminal CS cases. Increasing palliative and hospice care integration into CS management can enhance patient outcomes, ensuring holistic, patient-centered care even when curative options are limited [20].

Conclusion

Cardiogenic shock (CS) remains a complex syndrome with high mortality, necessitating a comprehensive, multidisciplinary approach for effective management. Advances in pathophysiology understanding, phenotyping, and novel staging systems like the SCAI classification have improved risk stratification and treatment strategies. Early invasive hemodynamic monitoring, tailored mechanical circulatory support (MCS), and regionalized care systems have shown promise in improving outcomes. Understanding and enhancing long-term outcomes after critical illness is crucial. Efforts should be focused on promoting convalescence and full recovery, particularly during the vulnerable transition from ICU to post-discharge follow-up. This phase is critical and demands greater attention to ensure patients receive the necessary support and care for a successful recovery. Long-term follow-up and support for CS survivors are essential, emphasizing functional recovery and quality of life. Incorporating palliative care ensures holistic, patient-centered care. Despite ongoing challenges, structured, evidence-based protocols and evolving multidisciplinary approaches offer hope for better management and prognosis of CS in the future.

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