Cardiogenic shock in acute coronary syndrome - A review

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Abstract

Background: Cardiogenic shock (CS) is a life-threatening complication of acute coronary syndrome (ACS), characterized by end-organ hypoperfusion and tissue hypoxia secondary to severe cardiac dysfunction. It occurs in approximately 3%-13% of ACS cases and carries a high mortality rate, reaching up to 50% at one year despite advancements in management. Patients with ST-elevation myocardial infarction (STEMI) complicated by cardiogenic shock or cardiac arrest experience higher incidences of multiorgan failure, acute kidney injury, and in-hospital mortality.

Objective: To review and summarize recent literature on the incidence, pathophysiology, and management of cardiogenic shock in ACS, with emphasis on diagnostic challenges and therapeutic strategies aimed at improving survival outcomes.

Methods: A literature review was conducted using the PubMed database for studies published between 2010 and 2023. The search terms included "acute coronary syndrome," "ACS," and "cardiogenic shock." Relevant studies focusing on adult patients with ACS complicated by cardiogenic shock were analyzed, and data were synthesized to outline current evidence and recommendations.

Results: Cardiogenic shock complicates nearly 10% of all acute myocardial infarctions and remains the leading cause of in-hospital mortality following ACS. Early recognition and intervention are essential to prevent irreversible end-organ injury. Emergent coronary angiography and revascularization remain the only evidence-based therapies proven to improve survival. Optimal management requires a multidisciplinary, time-sensitive approach integrating pharmacologic support with vasoactive agents and mechanical circulatory support (MCS) before, during, and after revascularization.

Conclusion: Despite major therapeutic advancements, cardiogenic shock continues to pose significant clinical and prognostic challenges. Prompt diagnosis, coordinated multidisciplinary management, and early revascularization are critical to improving outcomes. Further research is warranted to refine hemodynamic monitoring, optimize MCS utilization, and enhance survival in this high-risk population.

Keywords: Cardiogenic shock, Acute coronary syndrome, ST-elevation myocardial infarction, Revascularization, Mechanical circulatory support, Mortality

Introduction

Cardiogenic shock represents a range of clinical presentations marked by end-organ hypoperfusion and tissue hypoxia resulting from cardiac dysfunction, which can lead to multiorgan failure and death. It occurs in 3% to 13% of patients with acute coronary syndromes (ACS) and is associated with mortality rates of about 40% at 30 days and 50% at one year[1,2]. In patients with ST-elevation myocardial infarction (STEMI) cardiogenic shock, cardiac arrest, and both conditions together are reported in 5.8%, 6.2%, and 2.7% of cases, respectively. These patients also experience higher rates of multi-organ failure, acute renal failure, and in-hospital mortality. Identifying cardiogenic shock is particularly challenging due to its varied presentations, which can range from normal blood pressure with peripheral vasoconstriction {High Systemic Vascular Resistance (SVR)} affecting organ perfusion to significantly reduced systemic blood pressure[3,4].

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Early recognition of cardiogenic shock in ACS is crucial for preventing end-organ hypoperfusion and associated complications. Accurate assessment requires versatile clinical and diagnostic skills. Despite advances in reperfusion therapy and mechanical circulatory support, morbidity and mortality rates among patients with cardiogenic shock remain high[5,6,7].

Definition of cardiogenic shock[1,2,6]

Various guidelines and studies have established different criteria for defining cardiogenic shock. The most commonly accepted classification divides it into clinical and hemodynamic criteria. (Table-1)

Table 1: Clinical and hemodynamic criteria for cardiogenic shock.

Clinical	Haemodynamic
Systolic blood pressure <90 mmHg with adequate volume Clinical signs of hypoperfusion which include • Cold peripheries • Oliguria • Impaired mentation • Narrow pulse pressure	Persistent hypotension • Systolic blood pressure < 90 mmHg, • MAP 30 mmHg below baseline
Laboratory signs of hypoperfusion • Metabolic acidemia • Increased serum lactate • Increased serum creatinine	Cardiac Index • <1.8 L/min/ m2 unsupported, or <2.0 - 2.2 L /min/m2 with support) • Adequate/elevated filling pressures LVEDP >18 mmHg or RVEDP >10-15 mmHg

Aetiology and pathophysiology of cardiogenic shock in ACS

Acute left ventricular dysfunction occuring due to myocardial infarction is a common cause for cardiogenic shock, studies have shown forty-two percent of patients had lesions in the left anterior descending (LAD) artery, while the left circumflex (LCX) artery accounted for 21%, the left mainstem (LMS) artery for 8%, and the right coronary artery (RCA) for 28% of the remaining lesions who presented with cardiogenic shock; Few patients had combinations of lesions and triple vessel disease.

Although acute myocardial infarction is the most common cause of cardiogenic shock, other conditions affecting the myocardium, valves, conduction system, or pericardium can also lead to this condition. (Table 2) Historically, it was believed that compensatory systemic vasoconstriction in response to reduced cardiac function was the primary mechanism behind cardiogenic shock. However, it is now understood that cardiogenic shock arises from acute to subacute dysfunctions across the entire circulatory system. Loss of ventricular function, including both systolic and diastolic dysfunction, is the major contributor, but other factors within the circulatory system also play a role, either through inadequate compensation or additional defects[1,2,6].

Table 2: Myocardial complications after acute myocardial infarction leading to Cardiogenic shock[8,9,10]

Parameter	Ventricular septal rupture	Ventricular free-wall rupture	Papillary muscle rupture
Incidence	Up to 4 % in cardiogenic shock Up to 3.0% without reperfusion therapy 0.3% after thrombolysis	Up to 6% PCI seems to reduce risk Thrombolysis increases risk	Up to 1% Predominately affects the posteromedial papillary muscle
Time duration	24 hour with thrombolysis 3-7 days without reperfusion therapy	2.7 days with thrombolysis 1-7 days without reperfusion therapy	24-36 h (range 1-14 days)

The hallmark of cardiogenic shock is hypoperfusion of the extremities and vital organs. While compensatory vasoconstriction may temporarily improve coronary and peripheral perfusion, it increases afterload. Prolonged shock can lead to systemic inflammation, which often results in pathological vasodilation. Endothelial and inducible nitric oxide (NO) synthase contribute to this process by producing high levels of NO and peroxynitrite, which can be cardiotoxic and negatively affect myocardial contractility. Other inflammatory markers, such as interleukins (ILs) and tumor necrosis factor (TNF), also play a role. (FIGURE-1)

The expanded shock spiral concept encompasses various treatment options, such as reperfusion through PCI or Coronary Artery Bypass Grafting (CABG), mechanical support with Intraaortic Ballon Pump (IABP) or Left Ventricular assist devices (LVAD), and the use of inotropes or vasopressors. These treatments, shown in red on the left-hand side, aim to counteract the shock spiral but also have potential drawbacks, including bleeding complications and effects on systemic inflammation[10,11,12].

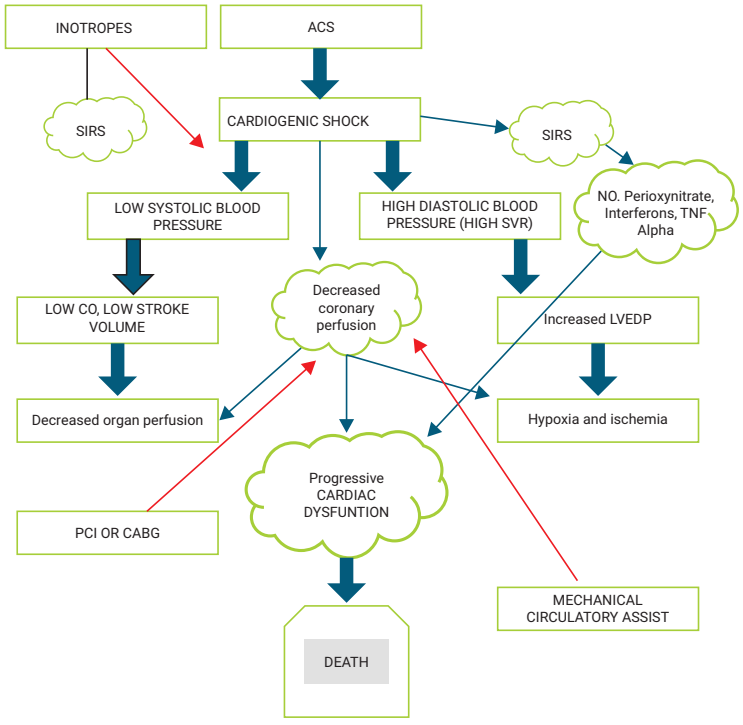

Figure 1: Schematic representation of the mechanisms of cardiogenic shock and interventions that may improve patient outcomes.

Abbreviations - ACS-Acute coronary syndrome, SIRS-Systemic inflammatory response syndrome, SVR- Systemic Vascular resistance, NO-Nitric oxide, TNF-Tumour necrosis factor, CO-Cardiac Output, PCI-Percutaneous Intervention, CABG- Coronary artery Bypass Grafting, LVEDP- Left Ventricular end diastolic pressure

Assessment and Diagnosis[5,6,13,14,15]

Patients presenting with acute coronary syndromes should be vigilantly monitored to detect signs of shock. Delay in diagnosis can lead to irreversible organ dysfunction, resulting in significant morbidity and mortality. Bedside clinical evaluation for symptoms of cardiogenic shock is crucial; if such symptoms are present, further assessment with echocardiography and metabolic parameters is necessary. Early identification and diagnosis of the underlying cause of cardiogenic shock, followed by prompt medical or surgical management, can significantly improve outcomes. (Table-3)[5,6,13,15]

Table 3: Clinical features for diagnosis[5,6,13,15]

Features Suggestive of Right heart failure	Features Suggestive of Right or Left heart failure	Features Suggestive of Left heart failure
Pedal edema	Cold peripheries	Crepitations in Lung fields
Sacral edema	Cyanosis	Respiratory wheeze
Hepatomegaly	Orthopnea	
Increased JVP	Delayed capillary refill	

Hemodynamic Monitoring[2,7,8]

Pulmonary artery catheters can provide valued hemodynamic data to confirm the presence and severity of cardiogenic shock, assess right ventricular involvement, and offer significant short-term prognostic insights, particularly through cardiac power and stroke work index. However, their use has declined, as evidenced by changes in criteria for including patients in cardiogenic shock trials and controversy regarding their benefits, as highlighted in a meta-analysis. Although non-invasive devices are available, their reliability in this context has not been thoroughly studied. The European Society of Cardiology still recommends individualized use of pulmonary artery catheters (IIb B recommendation) for monitoring hemodynamic variables or guiding treatment in patients with severe heart failure who are not responding to standard therapy.

Management

Medical management focuses on optimization of revascularization strategies, fluid balance and resuscitation, and inotropic support. Additional therapies include mechanical circulatory support devices and surgical interventions for myocardial reperfusion and management of mechanical complications following myocardial infarction.

Establishing selection criteria for suitable candidates and determining the optimal timing for mechanical circulatory support (MCS) are crucial. Currently, about 40-50% of cardiogenic shock (CS) patients with acute coronary syndromes (ACS) survive with standard medical therapy. However, 25-35% may not respond effectively to MCS. On the other hand, 15-35% of these patients exhibit significant benefits from MCS and ultimately survive with VA-ECMO (Veno Arterial - Extra corporeal membrane Oxygenation) support.

Revascularisation

Early revascularization remains the most critical treatment approach for cardiogenic shock resulting from acute myocardial infarction and is the only strategy supported by trial data to reduce mortality in these patients. (SHOCK TRIAL). Although the SHOCK trial did not achieve its primary endpoint demonstrating a clear benefit of early revascularization over medical therapy for 30-day mortality (46.7% vs. 56.0%) - it did show a significant reduction in mortality at longer follow-up intervals of 6 months, 1 year, and 6 years. Notably, while 64% of patients in the trial were treated with percutaneous coronary intervention, 36% received early coronary artery bypass grafting, a detail that is often overlooked. The number needed to treat with early revascularization to save one life compared to initial medical stabilization is fewer than eight. Culprit shock Trial evaluated outcome in patients of Acute MI complicated by cardiogenic and evidence of multi vessel disease on early Coronary angiogram, showed single vessels Culprit lesion only PCI showed reduction in 30 days risk of composite death or RRT in comparison with Multi vessel PCI. Thus, culprit-vessel-only PCI is a reasonable intervention in myocardial infarction patients presenting with cardiogenic shock[2,5,16,17].

Fluid Resuscitation[1,7]

Fluid resuscitation in the early management of cardiogenic shock poses a clinical challenge due to the variability and difficulty in accurately assessing fluid needs. It is crucial to evaluate the patient's optimal fluid status whenever shock develops following a myocardial infarction, as hypovolemia can be one of the differential diagnosis. With advancements in dynamic fluid status assessment and non-invasive cardiac output monitoring, it is recommended to use bedside echocardiography and non-invasive cardiac output measurements to diagnose shock and determine the need for fluid resuscitation. Utilizing dynamic indices of fluid responsiveness should guide fluid resuscitation efforts.

In cases of right-sided heart failure, right atrial pressures and pulmonary artery wedge pressures are unreliable indicators of fluid responsiveness. Echocardiography can help evaluate right-sided heart volume status and detect pericardial fluid collections. The most definitive method for assessing volume status and the effectiveness of resuscitation is right heart catheterization, which should be performed alongside coronary angiography. If hypovolemia is identified, conservative administration of crystalloids (250 mL) is appropriate while the patient is being prepared for cardiac catheterization.

Airway and breathing[18,19]

Patients with cardiogenic shock often require oxygen support due to pulmonary edema or other related complications. Identifying and categorizing right and left ventricular dysfunction in cardiogenic shock is crucial for selecting the appropriate oxygen delivery devices. Positive pressure ventilation can help reduce afterload and pulmonary edema in left ventricular (LV) failure but may worsen right ventricular (RV) failure by increasing afterload. Conversely, spontaneous ventilation can benefit RV dysfunction but may exacerbate LV dysfunction. When selecting sedative medications, it is important to choose those with minimal effects on cardiac function to prevent further deterioration of heart failure.

Inotropes[1,2,7]

The treatment of cardiogenic shock typically begins with initial stabilization through volume expansion to achieve optimal filling pressures, if fluid status is appropriate, the use of vasopressors and inotropes is recommended. Despite the frequent use of pharmacological support in cardiogenic shock, there is limited clinical outcome data and few randomized studies to guide treatment selection.

The SOAP II trial conducted in 2010 compared noradrenaline and dopamine as first-line therapies for cardiogenic shock. It was found that dopamine was associated with a higher rate of arrhythmias and increased mortality in patients with cardiogenic shock. Subsequent studies supported these findings, leading to the current recommendation against the use of dopamine. Noradrenaline is now recommended as the first-line therapy to achieve a systolic blood pressure (SBP) greater than 90 mmHg. For second-line therapy, vasopressin or adrenaline may be used.

In cases of cardiogenic shock where hypotension is not present (SBP >90 mmHg), inotropes such as dobutamine, levosimendan, or milrinone can be used based on organ dysfunction, therapy tolerance, depending on predominance of right or left heart failure, and the presence of organ dysfunction.

A brief summary of inotropes, including their effects on hemodynamics, dosing, indications, and considerations, is provided in Tables 4 and 5. Additionally, therapies such as pulmonary vasodilators, including inhaled nitric oxide and prostacyclins, are recommended for managing pulmonary artery hypertension with right ventricular dysfunction.

Table 4: Hemodynamic effects, dosing, indications, and key considerations of commonly used inotropes

Inotropes And Vasopressors	Mechanism Of Action	Effects On Hemodynamics	Indications	Considerations	
Norepinephrine Dose- 1 -12 mcg/min	α < β agonist	Inotropy, chronotropy, dromotropy, and vasoconstriction	Most common first line agent in shock	Most benefits demonstrated in septic shock	
Epinephrine Dose- 0.05-0.2 mcg/kg/minute	α and β agonist	Inotropy, chronotropy, dromotropy, and vasoconstriction	Commonly used as second line agent or first line in anaphylactic shock	Surviving Sepsis Guidelines has most data for epinephrine as second line agent	
Phenylephrine Dose-10 to 200mcg/min	α 1 agonist	Vasoconstriction	Various forms of shock Increased after load	Caution in cardiac dysfunction as it increases afterload May decrease stroke volume and Cardiac output	
Dopamine Beta1 effects: 2-10 mcg/kg/min Alpha effects: >10 mcg/kg/min Dopaminergic effects: 0.5-2 mcg/kg/min	Dose dependent α, β, and D agonism	Inotropy, dromotropy, chronotropy, and vasoconstriction (at highest doses)	Not recommended	SOAP II trial demonstrated more incidence of tachy-arrythmias and increased mortality in CS patients when dopamine was used as first line	
Vasopressin Dose-0.01 to 0.03 units/minute	V1 agonist	Vasoconstriction	Second line agent in most forms of shock, less effect on pulmonary vasoconstriction(Better in RV dysfuntion with high PAH)	On or Off dosing, can cause hyponatremia May reduce stroke Volume and Cardiac Output in myocardial dysfuntion or May precipitate ischaemia in CAD	
Levosimendan Dose- Loading 6 to 12mcg/kg over 10mins, maintanance 0.05-0.2 µg/kg/min for 24hrs	Myofilament Ca2+ sensitizer and K+ channel modifier	Inodilator	Used in acutely decompensated chronic heart failure	Minimal effect on myocardial oxygen consumption	
Dobutamine	B agonist	Intravenous	Hypotension, tachyarrhythmia, headache, thrombocytopenia	0.5-1 mcg/kg/min IV continuous infusion initially, then 2-20 mcg/kg/min; not to exceed 40 mcg/kg/min	RV and LV dysfunction with SBP>90

Table 5: Overview of hemodynamic effects, dosing, indications, and important considerations of infrequently used inotropes.

Agent	Mechanism of Action	Route	Adverse effects	Dose	Consideration
Nitric Oxide	↑ cGMP	Inhaled	Blurred vision, confusion, sweating, malaise, headache, bleeding	Up to 40ppm	In High PVR with RV dysfunction
Milrinone Dose-0.125 to 0.25mcg/kg/min Max-0.75mcg/kg/min	Phosphodiesterase 3 inhibitor	Intravenous	Bleeding, hypotension, chest pain, tremors, bronchospasm, hypokalemia	50 mcg/kg loading dose by IV push over 10 minutes, then 0.375-0.75 mcg/kg/min IV	High PVR with RV dysfunction and SBP>90 Renal dose modification required
Prostacyclin	↑ cAMP, ↑ K, ↓ ET-1, and ↑ K+	Inhaled or Intravenous	Bleeding, arrhythmias, diarrhea, edema, fevers, chills	--	High PVR

Intra-aortic balloon pump- IABP[5,6,8]

Intra-aortic balloon pumping is still commonly used to mechanically assist patients with cardiogenic shock where pharmacological therapy is not adequate. The device is timed to inflate and deflate in concert with the cardiac cycle, thereby increasing the diastolic blood pressure and reducing the systolic blood pressure. Intra-aortic balloon pumping can augment blood flow in the coronary arteries, unload the left ventricle and therefore enhance myocardial oxygen supply by improving the peak diastolic pressure and reducing the end-systolic pressure by means of diastolic inflation and rapid systolic deflation.

Although a number of factors are physiologically beneficial with the use of intra-aortic balloon pumping, evidence from several meta-analyses, randomized controlled trials and Recent IABP-SHOCK II (Intra-aortic Balloon Pump in Cardiogenic Shock II) trial, intra-aortic balloon pump (IABP) use was not associated with lower 30-day mortality. However, in the study where total of 600 patients (75.9%) were randomly assigned to either intraaortic balloon counterpulsation (IABP group, 301 patients) or no IABP (control group, 299 patients) groups; in the control group, 30 patients (10.0%) later received an IABP, with 26 of these considered protocol violations. In the IABP group, 13 patients (4.3%) did not receive the pump, usually due to death before insertion. As a result, the routine use of an IABP for cardiogenic shock complicating acute myocardial infarction continues to be debated. Currently with increased supportive evidence of Other Mechanical support devices and ECMO the use of IABP among clinicians have reduced due to inconsistent outcome results.

Mechanical circulatory devices[11,12,18,20]

The rationale for initiating mechanical circulatory support (MCS) early in acute myocardial infarction with cardiogenic shock (AMICS) is to reduce ventricular workload, enhance systemic and myocardial perfusion, and provide hemodynamic support during percutaneous coronary intervention (PCI). Persistent clinical signs of hypoperfusion, hypotension, ongoing vasopressor requirements, or cardiac power output less than 0.6 watts despite adequate filling pressures may indicate the need for MCS as an adjunct to stabilization before coronary revascularization.

For patients with predominant left ventricular (LV) failure, MCS options include intra-aortic balloon counter pulsation (IABP), transvalvular axial flow pumps (Impella LP/CP/5.0/5.5), and the Tandem Heart percutaneous LV assist device. In cases of predominant right ventricular (RV) failure, options include the Impella RP pump and Tandem Heart Protek-Duo percutaneous right ventricular assist device. Veno arterial (VA) extracorporeal membrane oxygenation (ECMO) can offer systemic circulatory support but requires close monitoring for LV distension and worsening pulmonary edema. For biventricular failure, patients may be supported with bilateral Impella pumps or VA-ECMO combined with an LV venting mechanism[21].

Randomized trials comparing IABP with other MCS devices have not demonstrated improved survival with any specific MCS device, although these studies were often small and not designed to assess hard endpoints. More prospective research is needed to better understand the clinical utility of IABP in both ischemic and non-ischemic cardiogenic shock. Recent randomized controlled trials, including EURO SHOCK,

ECLS SHOCK, and ECMO-CS, did not show significant mortality benefits from mechanical circulatory devices and ECMO in cardiogenic shock following myocardial infarction. However, the ANCHOR trial suggests that ECMO may be beneficial when early signs of inadequate organ perfusion are present, serving as a critical bridge to recovery or organ transplantation. ECMO can also provide temporary support for patients with severe cardiogenic shock, allowing time for reperfusion therapy and recovery.

A recent trial involving the use of the MCS device (Impella) in patients with STEMI and cardiogenic shock demonstrated improved survival at 180 days. However, the use of the Impella device was also linked to an increased need for renal replacement therapy[21].

Conclusion

Cardiogenic shock complicates approximately 10% of all acute myocardial infarctions (AMI) and is the leading cause of in-hospital mortality following acute coronary syndrome (ACS). The only evidence-based treatment for this condition is emergent angiography and revascularization. Effective management requires the swift and coordinated efforts of a multidisciplinary team of various specialties. Successful outcomes depend on timely, decisive, and patient-centered decision-making. Addressing the failing myocardium, hemodynamic instability, and end-organ dysfunction simultaneously is crucial. This involves the use of vasoactive pharmacological agents and appropriate mechanical circulatory support (MCS) before, during, and after revascularization.

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