Chapter 65
Approach to Cardiogenic Shock in the ICU

65.1 Introduction

Cardiogenic shock (CS) is a life-threatening condition characterized by inadequate tissue perfusion due to severe impairment of cardiac function. It results from a significant reduction in cardiac output, commonly caused by acute myocardial infarction (AMI), severe valvular disease, or mechanical complications. Despite advances in management, CS continues to have a high mortality rate. Early recognition and a multidisciplinary approach in the ICU are essential for improving patient outcomes. This chapter provides a comprehensive approach to managing cardiogenic shock, emphasizing staging, detailed pathophysiology, innovative management strategies, and long-term considerations [1, 2] [Ref: Algorithm 65.1].

65.2 Multidisciplinary Shock Teams

Effective management of cardiogenic shock necessitates the involvement of a multidisciplinary shock team. This team typically includes cardiologists, intensivists, cardiac surgeons, emergency physicians, nurses, and other specialists. Collaborative decision-making enhances patient outcomes by integrating diverse expertise, leading to more comprehensive and timely care plans. The formation of such teams is emphasized in recent consensus statements, highlighting their role in improving survival rates and optimizing resource utilization [3].

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65.3 Pathophysiology

Understanding the complex pathophysiology of cardiogenic shock is crucial for effective management. The body initiates several compensatory mechanisms in response to decreased cardiac output:

- Sympathetic Activation: The sympathetic nervous system responds by increasing heart rate and systemic vasoconstriction to maintain blood pressure and perfusion to vital organs.
- Renin-Angiotensin-Aldosterone System (RAAS): Activation of the RAAS leads to sodium and water retention, increasing preload in an attempt to augment cardiac output.
- Inflammatory Cascades: In severe cases, systemic inflammation can lead to vasodilation and capillary leakage, exacerbating hypotension and tissue hypoperfusion. Nitric oxide, interleukins, and tumor necrosis factor alpha (TNF-α) are the mediators.

These compensatory mechanisms can become maladaptive over time, contributing to worsening cardiac function and progression of shock.

65.4 Comprehensive Classification

65.4.1 SCAI Shock Staging

The Society for Cardiovascular Angiography and Interventions (SCAI) has developed a staging system ranging from A to E to classify the severity of cardiogenic shock:

- Stage A (At risk): Patients are at risk of developing shock but are hemodynamically stable.
- Stage B (Beginning shock): Early signs of shock with mild hemodynamic compromise.
- Stage C (Classic shock): Hypotension with signs of hypoperfusion requiring interventions.
- Stage D (Deteriorating shock): Failure to respond to initial therapies; worsening hemodynamics.
- Stage E (Extremis): Critical condition with severe hypotension and hypoperfusion despite maximal support.

This dynamic staging aids in guiding treatment decisions and monitoring patient progression or improvement.

65.4.2 Phenotypic Classifications

Identifying the phenotype of cardiogenic shock helps tailor therapy:

- Cardiorenal Syndrome: Characterized by renal impairment due to decreased renal perfusion; management focuses on optimizing volume status and renal function.
- Cardiometabolic Shock: Associated with metabolic disturbances like hyperglycemia and acidosis; requires metabolic stabilization alongside hemodynamic support.

Recognizing these phenotypes enables clinicians to address specific underlying mechanisms contributing to shock.

65.5 Etiological Diversity

While AMI is the most common cause, other etiologies should be considered:

- Fulminant Myocarditis: Acute inflammation of the myocardium leading to severe ventricular dysfunction.
- Takotsubo Cardiomyopathy: Stress-induced cardiomyopathy presenting with transient left ventricular apical ballooning.
- Septic Cardiomyopathy: Cardiac dysfunction resulting from severe sepsis and systemic inflammation.

Recognizing these conditions is important, as management may differ from typical ischemic causes.

65.6 Clinical Signs and Symptoms

Patients with cardiogenic shock generally have altered mental status, hypotension, arrhythmia, diminished pulses, breathlessness/dyspnea, peripheral edema, jugular venous distension, and orthopnea. They mostly have cool extremities and signs of pulmonary congestion. This is generally termed as “cold and wet” and is generally associated with reduced cardiac index (CI), increased systemic vascular resistance (SVR), and pulmonary capillary wedge pressure (PCWP), whereas patients with normal PCWP are euvolemic and as termed “cold and dry.” The “wet and warm” types are usually underrecognized and have high mortality. This is an inflammatory response to acute cardiac injury and has low CI, low to normal SVR, and elevated PCWP.

Investigations:

1. Cardiac catheterization is the definitive diagnostic investigation, and it also helps guide the intervention in cardiogenic shock.
2. ECG: Within 10 min of presentation, if shock is suspected to be of cardiac origin.
3. Complete blood counts and metabolic panel (blood gas analysis, electrolytes, renal function tests, and liver function tests): Should be obtained every 12–24 h to gather information about oxygenation, organ failure, and electrolyte status.
4. Echocardiography: Helps detect complications and the site of failure (right or left ventricle) and also gives an idea about the vessel involved (regional wall motion abnormality).

65.7 Stabilization

Maintain airway, breathing, and oxygenation. Target blood saturation levels above 90%. Use supplemental oxygen. Noninvasive ventilation can also be tried. If inadequate, go for invasive ventilation targeting lung-protective ventilation (5–7 ml/kg). Low tidal volume helps in blood flow between pulmonary and parenchymal vasculature.

Fluid resuscitation is challenging in CS as it may lead to congestion. Right heart catheterization helps guide fluid therapy in right ventricular failure if the patient is hypovolemic. Conservative boluses of 250–500 ml may be used.

65.8 Early Intervention Protocols

Implementing protocols for early invasive hemodynamic assessment and intervention is crucial. Standardized protocols ensure prompt recognition of cardiogenic shock and initiation of appropriate therapies, potentially preventing disease progression. These protocols typically include:

- Rapid bedside echocardiography.
- Early placement of arterial and central venous lines.
- Timely initiation of inotropes and vasopressors.
- Immediate consultation with the multidisciplinary shock team.

Vasopressor support (norepinephrine) should be initiated and titrated to a mean arterial pressure (MAP) of 65 mm Hg. Vasopressin may be more beneficial in acute right ventricular failure due to lower pulmonary vasoconstriction. Other agents that can be used are phenylephrine, epinephrine, dopamine, dobutamine, and levosimendan. Nitric oxide, milrinone, prostacyclin, and dobutamine are the vasoactive agents within the pulmonary circuit.

65.9 Early Hemodynamic Monitoring

Continuous blood pressure monitoring must be done using an arterial line. Early insertion of pulmonary artery catheters (PACs) or utilization of advanced noninvasive monitoring techniques is recommended for precise hemodynamic assessment. Real-time data on cardiac output, pulmonary pressures, and systemic vascular resistance guide therapeutic interventions and adjustments. SvO_2 (mixed venous oxygen saturation) must be measured. Low SvO_2 indicates reduced cardiac output, anemia, hypoxemia, or increased oxygen consumption. It can also help assess response to therapy. It should be done every 4 h in the early stages of shock. PACs also offer therapeutic advantages via continuous monitoring of cardiac output during inotrope and pulmonary vasodilator titration.

65.10 Innovative Management Strategies

65.10.1 Temporary Mechanical Circulatory Support (tMCS) Devices

For patients not responding adequately to pharmacologic therapy, temporary mechanical circulatory support devices offer hemodynamic stabilization:

- Intra-aortic Balloon Pump (IABP): Augments diastolic pressure to improve coronary perfusion; suitable for patients with less severe hemodynamic compromise.
- Impella Devices: Microaxial flow pumps providing forward blood flow; ideal for left ventricular support.
- Tandem Heart: Provides left atrial to femoral arterial bypass; used in severe left ventricular failure.
- Extracorporeal Membrane Oxygenation (ECMO): Offers both cardiac and respiratory support; used in profound shock with multi-organ failure.

The choice of device depends on the patient's hemodynamic profile, severity of shock, and specific clinical scenario.

65.11 Management Framework

Acute MI-Associated Cardiogenic Shock (AMI-CS)

1. Urgent Revascularization: Primary percutaneous coronary intervention (PCI) is the gold standard.
2. Pharmacologic Support: Initiate inotropes (e.g., dobutamine) and vasopressors (e.g., norepinephrine) to stabilize hemodynamics.

3. Antiplatelet and Anticoagulant Therapy: Administer as per guidelines to prevent further thrombotic events.
4. Mechanical Support: Consider tMCS devices if there is inadequate response to pharmacologic therapy.

Non-AMI Cardiogenic Shock

1. Identify Underlying Cause: Utilize imaging and laboratory studies to diagnose conditions like valvular disorders, tamponade, or ventricular septal defects.
2. Definitive Management:
   - Valvular Disease: Surgical or transcatheter valve repair/replacement.
   - Tamponade: Urgent pericardiocentesis.
   - Mechanical Complications: Surgical repair of ventricular ruptures or defects.
3. Supportive Care: Optimize preload, afterload, and contractility with appropriate medications.

65.12 Prognostic Insights

Biomarkers
- Lactate Levels: Elevated lactate indicates tissue hypoperfusion; trends in lactate clearance are useful prognostic indicators.
- Other Biomarkers: Elevated troponins, B-type natriuretic peptide (BNP), and inflammatory markers provide additional prognostic information.

Monitoring these biomarkers aids in assessing the severity of shock and the effectiveness of interventions.

Step-by-Step Algorithm

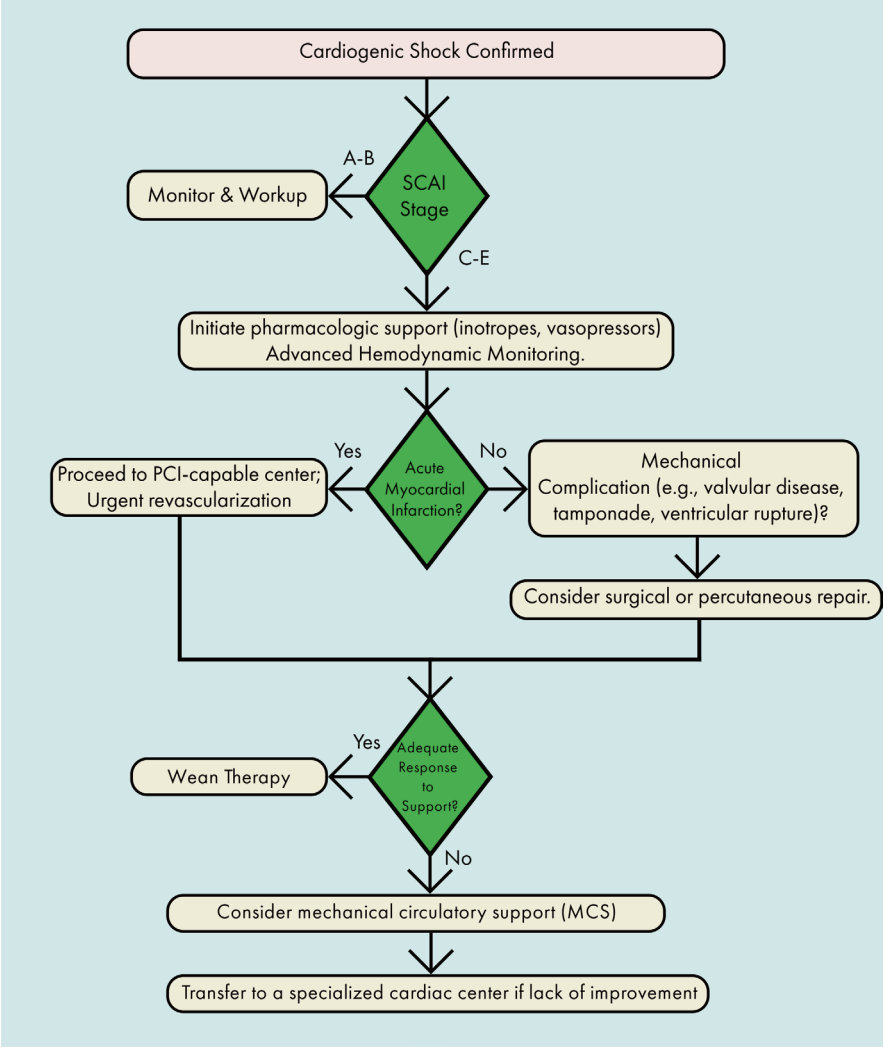
1. Confirmation of Cardiogenic Shock.
   - Clinical Assessment: Hypotension (SBP <90 mm Hg), signs of hypoperfusion (cold extremities, altered mental status).
   - Diagnostic Tools: Bedside echocardiography, ECG, laboratory tests (elevated lactate, troponins, BNP).
2. SCAI Staging Assessment.
   - Determine the shock stage (A to E) to guide urgency and level of intervention.
3. Multidisciplinary Team Activation.
   - Involve the shock team early for collaborative decision-making.
4. Monitoring and Initial Workup.
   - For Stage A-B patients:
   - Close hemodynamic monitoring.
   - Routine workup to identify reversible factors.

5. Initiation of Pharmacologic Support and Hemodynamic Monitoring.
    - For Stage C-E patients:
    - Start inotropes and vasopressors.
    - Initiate advanced hemodynamic monitoring (e.g., pulmonary artery catheter).
6. Assessment for Acute Myocardial Infarction (AMI).
    - Yes:
    - Proceed with urgent revascularization (PCI or CABG).
    - No:
    - Evaluate for mechanical complications or other etiologies.
7. Management of Mechanical Complications.
    - Valvular Disease: Surgical or transcatheter intervention.
    - Tamponade: Pericardiocentesis.
    - Ventricular Rupture: Emergency surgical repair.
8. Evaluating Response to Support.
    - Adequate Response:
    - Gradual weaning from support under close monitoring.
    - Inadequate Response:
    - Escalate to tMCS devices.
9. Consider Mechanical Circulatory Support (tMCS).
    - Select an appropriate device based on hemodynamic needs.
10. Transfer to Specialized Cardiac Center if Lack of Improvement.
    - For advanced therapies or interventions not available at the current facility.

65.13 Conclusion

Management of cardiogenic shock in the ICU requires a structured, multidisciplinary approach that encompasses early recognition, detailed understanding of pathophysiology, comprehensive classification, and innovative management strategies. Incorporating protocols for early intervention, utilizing advanced hemodynamic monitoring, and considering etiological diversity are essential steps. Prognostic insights from biomarkers and implementing regionalized systems of care further enhance patient management. Attention to long-term outcomes, including prevention of recurrent shock and management of chronic heart failure, is crucial for improving survival and quality of life. Familiarity with this comprehensive approach enables clinicians to make informed decisions, ultimately leading to better patient outcomes in this high-stakes environment.

Algorithm 65.1: Approach to cardiogenic shock in the ICU



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