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Narrative Review : Update on Cardiogenic Shock and It's Management

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ABSTRACT

Cardiogenic shock is a condition of tissue hypoperfusion caused by primary abnormalities in the heart where there is a decrease in cardiac output, resulting in circulatory failure which results in hypoperfusion and tissue hypoxia. The most common cause of cardiogenic shock is acute myocardial infarction (AMI), of which 70% show a picture of ST-Elevation infarction (STEMI). The Society for Cardiovascular Angiography and Interventions (SCAI) has established five classifications of cardiogenic shock, namely A (At Risk), B (Beginning), C (Classic), D (Deteriorating), and E (Extreme). The main treatment that can be carried out in patients with cardiogenic shock is stabilizing the patient's oxygenation and circulation, then treating the underlying etiology of cardiogenic shock.

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INTRODUCTION

Cardiogenic shock is a condition of tissue hypoperfusion caused by a primary abnormality in the heart which is characterized by a decrease in cardiac output. This can result in circulatory failure resulting in hypoperfusion and tissue hypoxia. As for risk factors that cannot be modified, such as advanced age, the morbidity and mortality from cardiogenic shock is 30-50%. The most common cause of cardiogenic shock is MI, followed by disorders of the myocardium, valves, conduction system, and disorders of the pericardium.

AMI in the form of STEMI doubles the risk of cardiogenic shock compared to NSTEMI. According to research by Goldberg RJ, the mortality rate for cardiogenic shock in hospitals due to AMI has not changed in the last 10 years, around 40-50%. Considering the high mortality of this disease, fast and appropriate management and management of cardiogenic shock is important to understand. so that the risk of death can be avoided.

Definition

Cardiogenic shock is defined as clinical and biological evidence of tissue hypoperfusion resulting from cardiac dysfunction. Clinical manifestations include hypotension where systolic blood pressure (BP) ≤ 90 mmHg for more than or equal to 30 minutes, or support to maintain systolic BP ≤ 90 mmHg and urine output ≤ 30 mL/hour, or

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cold extremities. Hemodynamic criteria include a depressed cardiac index (≤ 2.2 liters per minute per square meter of body surface area) and an increase in pulmonary capillary pressure > 15 mmHg.

Other clinical manifestations that can be found include signs of hypoperfusion, mental disorders, narrowed pulse pressure, and even oliguria. The biochemical manifestations include increased serum lactate, creatinine, and metabolic acidosis, which conditions reflect tissue hypoxia and changes in cell metabolism that have the potential to cause organ dysfunction..[3]

Etiology

Cardiogenic shock is caused by various cardiac dysfunctions that affect the functioning of the right and/or left ventricle. However, the most common cause is related to left ventricular dysfunction, which if not treated properly will result in AMI. This condition causes disturbances in the myocardium which accelerates the process of regional necrosis and decreases the contractile mass of the heart so that ventricular function decreases. This is associated with decreased cardiac output and systemic hypoperfusion. Apart from that, cardiogenic shock can be caused by other mechanical disorders such as acute mitral regurgitation (papillary muscle rupture), ventricular wall rupture (septal or free wall), cardiac tamponade, cardiac contractility (hypertrophic obstructive cardiomyopathy, aortic stenosis), and left ventricular inflow obstruction. (atrial myxoma). Other causes include the use of cardiotoxic drugs (doxorubicin), drug overdose (beta/calcium channel blockers), metabolic disorders (acidosis), and electrolyte abnormalities (calcium or phosphate).[2,5,6]

Epidemiology

The most common cause of cardiogenic shock is MI accompanied by STEMI which can double the risk of cardiogenic shock compared to NSTEMI. Approximately 70% of patients experiencing acute coronary syndrome (ACS)-related cardiogenic shock present with STEMI, whereas non-ACS-related cardiogenic shock includes a wide range of diseases such as acute decompensation of chronic heart failure, heart valve disorders, myocarditis, and stress-induced cardiomyopathy.

According to research by Goldberg RJ, the death rate from cardiogenic shock in hospitals due to AMI has not changed in the last 10 years at 40-50%. Deaths that occur within 1 year due to cardiogenic shock are 50-60%, and deaths that occur within the first 30-60 days after the onset of shock are 70-80%. An estimated 3.3 deaths per 100 patients treated with percutaneous coronary intervention (PCI) occur due to treatment delays within 10 minutes..[3,7]

Pathophysiology

In general, shock occurs due to blood circulation disorders which cause an imbalance between oxygen demand and consumption. When one component fails, other components will be stimulated to compensate. This mechanism occurs excessively when shock occurs so that the oxygen deficit increases over time. As a result, macro and micro circulation change with a reciprocal interaction.

In the early phase, microcirculation and macrocirculation are connected coherently, but most patients experience incoherence between the two which results in persistent tissue

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hypoperfusion. This is caused by microcirculation heterogeneity, decreased capillary density, decreased local flow, or tissue edema, resulting in permanent damage..[8]

In a state of shock, the symptoms are dominated by a systemic inflammatory response in varying proportions, adding to its complexity. The systemic inflammatory response causes pathological vasodilation, releasing nitric oxide synthase and peroxynitrite, which have cardiotoxic inotropic effects. Interleukins and tumor necrosis factor alpha (TNF-a) are additional mediators that cause vasodilation and contribute to mortality in patients with cardiogenic shock.

Under physiological stress, the stroke volume of the right ventricle and the left ventricle have the same performance. Right ventricular failure (RVF) occurs when ventricular diastolic and/or systolic pressures are not sufficiently compensated by myocardial adaptive processes to produce an appropriate stroke volume. Impaired blood flow in the right ventricle causes end organ perfusion deficits accompanied by increased venous pressure. The right ventricle is less adaptive to afterload pressure and more tolerant of volume overload than the left ventricle, and this explains the inability of the right ventricle to tolerate very high increases in pulmonary artery pressure. When RVF results in right ventricular dilatation, the interventricular septum moves into the left ventricular space, impairing left ventricular diastolic filling and further exacerbating systemic hypoperfusion..[9]

Classification

The Society for Cardiovascular Angiography and Interventions (SCAI) classification defines five stages of the evolution of cardiogenic shock, from A (At Risk) to E (Extrimis), including modifications to cardiac arrest.[3]

a. **Stage A:** "At Risk" or at risk, a condition where patients do not experience signs or symptoms of cardiogenic shock but are at risk of developing it. Patients may appear healthy and have normal laboratory and physical examination results. In general, anterior wall infarctions and wide distribution infarctions carry a higher risk of cardiogenic shock but some patients experience shock with smaller infarcts due to pre-existing left ventricular dysfunction.
b. **Stage B:** “Beginning” or beginning (pre-shock or compensated shock), where patients have clinical evidence of relative hypotension or tachycardia without hypoperfusion. Hypotension was defined as systolic blood pressure (SBP) <90 mmHg or mean arterial blood pressure (MAP) <60 mmHg or >30 mmHg decrease from baseline values. Hypoperfusion is a clinical sign such as cold acral, poor urine output, mental disorders, and the like.
c. **Stage C:** "Classic" or classic stage, where patients with hypoperfusion require a series of initial interventions in addition to volume resuscitation to restore perfusion. These patients usually present with relative hypotension, most exhibiting a classic shock phenotype with hypoperfusion. Laboratory examination revealed impaired kidney function, increased lactate, brain natriuretic peptide, and/or liver enzymes. Invasive hemodynamics (if present) shows depressed cardiac indices associated with cardiogenic shock.

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d. **Stage D:** "Deteriorating," describes a patient who fails to stabilize despite intense initial efforts and further escalation is necessary. This classification requires that the patient has received some level of medical treatment/stabilization or that at least 30 minutes have passed but the patient has not responded with resolution of hypotension or end organ hypoperfusion.
e. **Stage E:** "Extremis" where patients with circulatory collapse, usually experiencing cardiac arrest that is refractory to cardiopulmonary resuscitation (CPR) or after ECMO-facilitated CPR intervention (eCPR).[10]

METHOD

Cardiogenic shock is a severe and life-threatening condition characterized by the heart's inability to pump sufficient blood to meet the body's needs, often resulting from acute myocardial infarction. This literature review aims to provide a comprehensive overview of cardiogenic shock, focusing on its pathophysiology, clinical manifestations, diagnostic criteria, and current therapeutic strategies. The review will examine studies that detail the mechanisms leading to reduced cardiac output and systemic perfusion, including myocardial injury, reduced contractility, and compensatory neurohormonal responses. Additionally, it will explore clinical outcomes associated with various treatment modalities such as pharmacological interventions, mechanical circulatory support, and revascularization techniques. By synthesizing current research findings, this review seeks to highlight advancements in the understanding and management of cardiogenic shock, identifying gaps in knowledge and potential areas for future investigation to improve patient prognosis and survival rates.

RESULTS AND DISCUSSION

Clinical Findings And Further Examination

The classic clinical syndrome in patients with cardiogenic shock is left heart failure characterized by systemic hypotension, SBP <90 mmHg or requiring inotropes to help maintain SBP between 90-100 mmHg, and symptoms or signs of organ hypoperfusion accompanied by pulmonary congestion. On physical examination, distant heart sounds, 3 or 4 heart sounds, or a systolic murmur are found. If a murmur is present, mechanical complications such as mitral regurgitation or ventricular septal rupture should be suspected. In addition, cardiogenic shock that occurs due to right ventricular infarction is characterized by hypotension, inferior wall myocardial infarction can be found on ECG leads II, III, and aVF (augmented vector foot), as well as increased JVP (jugular vein pressure) without pulmonary edema. .

The Forrester classification groups patients with cardiogenic shock based on the presence or absence of adequate perfusion: warm-dry (no congestion and no hypoperfusion); wet-warm (congestion present without hypoperfusion); dry-cold (no congestion but hypoperfusion); and wet-cold (congestion and hypoperfusion occur). This profile is associated with short-term mortality, where mortality is high if there is congestion

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and increases with hypoperfusion. For the clinician, it is important to evaluate signs such as JVP elevation, pulmonary congestion, prolonged CRT, and acral coldness during physical examination to evaluate patients with cardiogenic shock.

A 12-lead ECG needs to be performed in the first 10 minutes to evaluate the cause of cardiogenic shock. ECG findings can include ST segment elevation, ST segment depression, and non-ST deviation. Pathologic "q" waves may reflect low ejection fraction and extensive infarction. In patients with suspected acute coronary syndrome, it is necessary to examine the posterior ECG if there is an inferior wall infarction (ST segment elevation in leads II, III, and aVF), ST segment depression in the septal and anterior precordial leads (V1-V4), R:S ratio >1 on V1-V2, and ST segment elevation on inferior ECG (V7-V9).(12)

A complete blood count and metabolic profile should be performed every 12-24 hours to evaluate oxygenation, electrolyte status, and end organ damage. Blood glucose needs to be evaluated considering that critically ill patients can experience hyperglycemia regardless of whether or not they have a history of diabetes mellitus. Cardiac enzymes can be checked to estimate the extent of infarction damage, but the results should not delay revascularization in cases of ST-segment elevation myocardial infarction.

Thorax radiography can be performed to assess heart enlargement and the presence or absence of pulmonary edema. Echocardiography is important to determine the cause of shock. When cardiogenic shock is caused by infarction, echocardiography can evaluate right and left ventricular function, valve dysfunction, and mechanical complications underlying the shock. Assessments carried out include the inferior vena cava to estimate the volume status and right atrial pressure (RAP). Ventricular ejection fraction is also important to assess and can be categorized as normal, poor, or hyperkinetic. Echocardiography is also useful for evaluating the presence of pericardial effusion or obstructive lesions.

Patients with suspected ischemic right or left heart failure should undergo immediate cardiac catheterization for assessment of coronary anatomy, intracardiac pressure, valvular dysfunction, and structural disorders that often accompany acute coronary syndromes and contribute to cardiogenic shock. In hypotensive patients, it is necessary to install central venous and arterial access. Venous catheterization is used to assess central venous pressure (CVP) and central venous oxygen saturation (ScvO2). Arterial catheterization using a pulmonary artery catheter (PAC) is considered if cardiogenic shock does not respond to initial therapy. PAC can assess pulmonary artery systolic pressure, pulmonary artery diastolic pressure, and Pulmonary Capillary Wedge Pressure (PCWP). The latter is a transpulmonary thermodilution monitor as an alternative to PAC in patients with acute respiratory distress syndrome (ARDS).[14,16]

Management

The key to treating cardiogenic shock is to intervene in the patient as soon as possible, because each stage of shock according to the Society for Cardiovascular Angiography and Interventions (SCAI) is associated with an increase in hospital mortality. Oxygenation and circulation are the main things to stabilize, then treat the underlying etiology while monitoring vital signs. Patients with cardiogenic shock should be monitored to differentiate

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causes of hemodynamic instability, allow monitoring of response to therapeutic interventions, and determine whether mechanical circulatory support is required.

The most effective therapeutic intervention in AMI patients accompanied by cardiogenic shock is coronary reperfusion. When early invasive strategies cannot be implemented in a timely manner, fibrinolysis can be used for cardiogenic shock presenting with STEMI. Indications for fibrinolysis should be individualized depending on the risk of bleeding, the expected benefit of reperfusion, and the expected angiographic delay. Despite the lack of supporting evidence, fibrinolysis is commonly used in the treatment of cardiogenic shock. However, the best way to restore blood flow to the heart is through surgery.

Fluid resuscitation is a challenge in the initial management of cardiogenic shock because it is often difficult to assess and varies over time. The definitive method for assessing volume status and adequacy of resuscitation is through right heart catheterization, performed by coronary angiography. If hypovolemia is present, a conservative crystalloid bolus (250-500 mL) can be given, while the patient is stable for cardiac catheterization.

A pulse oximeter is used to monitor tissue oxygenation due to respiratory disorders. Oxygen targets depend on the patient's comorbidities, where in acute care saturation > 90% is acceptable. When oxygenation and non-invasive ventilation are inadequate, invasive ventilation is required where low tidal volume (5-7 mL/kg ideal body weight) is used in the management of ARDS..(20)

Vasopressors should be titrated until Mean Arterial Pressure (MAP) is >65 mmHg. Vasopressin has less pulmonary vasoconstriction than norepinephrine and is more useful as a first-line vasopressor in patients with cardiogenic shock with acute right ventricular dysfunction. When using this agent, invasive blood pressure monitoring is necessary because it can cause hypotension. The following table shows recommendations for the use of inotropes and/or vasopressors in patients in shock.[9]

Agen Inotropik	Mechanism	Efec	Indikation	Consideration
Phenylephrine	Agonis A1	Vasoconstriction	Various types of shock	In people with heart problems, it can increase afterload.
Norepinephrine	A < B Agonis	Inotropic, chronotopic, dromotropic, and vasoconstrictive	The most common first-line agent used in shock	Shows good effect on septic shock.
Epinephrine	A << B Agonis	Inotropic, chronotopic, dromotropic, and vasoconstrictive	Frequently used second-line or first-line agents in anaphylactic shock	The Surviving Sepsis guidelines show a lot of data regarding epinephrine as a second-line agent.
Dopamine	Dose dependen A, B, dan	Inotropic, chronotopic, dromotropic, and	Second-line agents generally used in shock	The SOAP II trial demonstrated a greater incidence of

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Agen Inotropik	Mechanism	Efec	Indikation	Consideration
	Agonis D	vasoconstrictive (at maximum doses)		tachyarrhythmias and increased mortality in patients with cardiogenic shock when dopamine was used as first line.
Vasopressin	V1 Agonis	Vasoconstriction	Second-line agents generally used in shock	On or off dose, can cause hyponatremia
Dobutamine	B Agonis	Inotropy and mild vasodilation	Generally used in cardiogenic shock	May cause hypotension
Levosimendan	Miofilamen Ca^{2+} dan K^{+} Channel Modifier	Ionotropic and inodilator	Used in decompensated acute chronic renal failure	Minimal effect on the myocardium

Table 1. Summary of Systemic Vasopressors.[9]

Mechanical Circulatory Support Device (MCS) can be classified as temporary or permanent devices. Temporary MCS devices can be placed percutaneously surgically, used as a bridge to recovery, or in patients who have a temporary device implanted and plan to transition to a durable MCS after clinical stabilization. The long-lasting, surgically implanted MCS device can be used as a bridge to recovery or as a final treatment. The use of long-lasting MCS devices is clinically recommended. After implantation of an MCS device, heart transplantation can be performed in suitable patients whose cardiac function is not expected to recover.

Acute renal failure occurs in 13-28% of patients with cardiogenic shock, so patients require Continuous Renal Replacement Therapy (CRRT). This therapy should be considered in stage 2 acute renal failure characterized by elevated serum creatinine (≥29 baseline) and urine output <0.5 mL/kg per hour for ≥12 hours; or when life-threatening changes in fluid, electrolyte, and acid-base balance trigger the need for dialysis.[21]

Prognosis

The prognosis in patients with cardiogenic shock depends on the underlying etiology. A cohort study showed the two most common etiologies causing cardiogenic shock, namely acute decompensation of chronic renal failure and ACS. Cardiogenic shock with acute decompensation of chronic renal failure has a worse prognosis, with a higher mortality rate after 6 months of follow-up. These results are compared with cardiogenic shock that occurs after ACS in the first month where the follow-up period shows a relatively better prognosis.

Actions that can be taken to prolong the survival of patients with cardiogenic shock due to AMI include rapid resuscitation with coronary artery revascularization. However, this action does not rule out the possibility of multiorgan failure so that long-term survival is not guaranteed. Therefore, patients with cardiogenic shock should be treated by an interprofessional team placed in intensive care such as in ICU (*Intensive Care Unit).*[2]

Although cardiogenic shock cannot be completely prevented, doctors must educate patients to reduce risk factors for heart disease such as avoiding smoking and exposure to cigarettes, lowering lipid levels, and ensuring regular blood sugar control. Additionally, following an exercise program can help you lose weight and help achieve better blood pressure control.[2]

REFERENCES

1. Blumer V, Kanwar MK, Barnett CF, Cowger JA, Damluji AA, Farr M, et al. Cardiogenic Shock in Older Adults: A Focus on Age-Associated Risks and Approach to Management: A Scientific Statement From the American Heart Association. Circulation. 2024;
2. Kosaraju A, Pendela V, Hai O. Cardiogenic Shock. Treasure Isl StatPearls. 2023; Available from: https://www.ncbi.nlm.nih.gov/books/NBK482255/
3. Bagaswoto HP, Juzar DA, Habib F, Bramantyo YS, Sanggula PNPP, Widiastuti AZ. Cardiogenic Shock. Indones J Cardiol. 2023;42(3):90–9.
4. Laghlam D, Benghanem S, Ortuno S, Bouabdallaoui N, Manzo-Silberman S, Hamzaoui O, et al. Management of cardiogenic shock: a narrative review. Ann Intensive Care. 2024;14(1). Available from: https://doi.org/10.1186/s13613-024-01260-y
5. Samsky MD, Morrow DA, Proudfoot AG, Hochman JS, Thiele H, Rao S V. Cardiogenic Shock after Acute Myocardial Infarction: A Review. JAMA - J Am Med Assoc. 2021;326(18):1840–50.
6. Bonello L, Laine M, Puymirat E, Ceccaldi V, Gaubert M, Paganelli F, et al. Etiology and Prognosis of Cardiogenic Shock in a Secondary Center without Surgical Back-Up. Cardiol Res Pract. 2019;2019(Lv).
7. Sciaccaluga C, Mandoli GE, Ghionzoli N, Anselmi F, Dini CS, Righini F, et al. Risk Stratification in Cardiogenic Shock: a Focus on the Available Evidence. Heart Fail Rev. 2022;27(4):1105–17. Available from: https://doi.org/10.1007/s10741-021-10140-7
8. Squara P, Hollenberg S, Payen D. Reconsidering Vasopressors for Cardiogenic Shock: Everything Should Be Made as Simple as Possible, but Not Simpler. Chest. 2019;156(2):392–401. Available from: https://doi.org/10.1016/j.chest.2019.03.020
9. Vahdatpour C, Collins D, Goldberg S. Cardiogenic Shock. J Am Heart Assoc. 2019;8(8):1–12.
10. Baran DA, Grines CL, Bailey S, Burkhoff D, Hall SA, Henry TD, et al. SCAI Clinical Expert Consensus Statement on The Classification of Cardiogenic Shock. Catheter Cardiovasc Interv. 2019;94(1):29–37.
11. Reyentovich A, Barghash MH, Hochman JS. Management of Refractory Cardiogenic Shock. Nat Rev Cardiol. 2016;13(8):481–92. Available from: http://dx.doi.org/10.1038/nrcardio.2016.96
12. Levy B, Klein T, Kimmoun A. Vasopressor use in cardiogenic shock. Curr Opin Crit Care. 2020;26(4):411–6.
13. Fuernau G, Beck J, Desch S, Eitel I, Jung C, Erbs S, et al. Mild Hypothermia in Cardiogenic Shock Complicating Myocardial Infarction: Randomized SHOCK-COOL

Trial. Circulation. 2019;139(4):448–57.
14. Mebazaa A, Combes A, van Diepen S, Hollinger A, Katz JN, Landoni G, et al. Management of cardiogenic shock complicating myocardial infarction. Intensive Care Med. 2018;44(6):760–73. Available from: https://doi.org/10.1007/s00134-018-5214-9
15. Tewelde SZ, Liu SS, Winters ME. Cardiogenic Shock. Cardiol Clin. 2018;36(1):53–61. Available from: https://doi.org/10.1016/j.ccl.2017.08.009
16. Saxena A, Garan AR, Kapur NK, O'Neill WW, Lindenfeld J, Pinney SP, et al. Value of Hemodynamic Monitoring in Patients with Cardiogenic Shock Undergoing Mechanical Circulatory Support. Circulation. 2020;141(14):1184–97.
17. Chioncel O, Parissis J, Mebazaa A, Thiele H, Desch S, Bauersachs J, et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock – a position statement from the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2020;22(8):1315–41.
18. Van Diepen S, Katz JN, Albert NM, Henry TD, Jacobs AK, Kapur NK, et al. Contemporary Management of Cardiogenic Shock: A Scientific Statement from the American Heart Association. Vol. 136, Circulation. 2017. 232–268 p.
19. de Asua I, Rosenberg A. On the right side of the heart: Medical and mechanical support of the failing right ventricle. J Intensive Care Soc. 2017;18(2):113–20.
20. L.C. P, S.J. W, S.J. F, P.S. M, S.J. B. Pulmonary vascular and right ventricular dysfunction in adult critical care: Current and emerging options for management: A systematic literature review. Crit Care. 2010;14(5). Available from: http://ccforum.com/content/14/5/R169%5Cnhttp://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=emed9&NEWS=N&AN=2011024738
21. Goyal A, Daneshpajouhnejad P, Hashmi MF, Bashir K. Acute Kidney Injury. NCBI Bookshelf. 2024; Available from: https://www.ncbi.nlm.nih.gov/books/NBK441896/