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The Importance of Hemodynamic Profile in Cardiogenic Shock for Guiding the Appropriate Management

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ABSTRACT

Cardiogenic shock (CS)-a condition where cardiac damage resulting on hypotension and hypoperfusion of end organ-still have high mortality rate and becoming a reason for patients admitted to CICU. Diagnosis of CS can be made using clinical criteria such as unresponsiveness to fluid resuscitation or hypoperfusion of peripheral organ such as cold extremity. But sometimes, it's difficult to distinguish shock caused by hypovolemia or low cardiac output/index (CI) without hemodynamic monitoring. Besides SCAI classification of CS, there is other classification that determined CS into four categories based on its hemodynamic type: dry warm (increasing of CI, low SVRI, low/normal PCWP), wet warm (low CI, low/normal SVRI, elevated PCWP), cold dry (low CI, high SVRI, low/normal PCWP), and cold wet (low CI, high SVRI, elevated PCWP). These approaches are important not only to established the diagnose but also to guiding the appropriate therapy.

Introduction

In this century, the acute myocardial infarction and heart failure management has been progressively advanced and resulting better outcome for the patients. In terms of definite treatment, the percutaneous coronary intervention (Primary PCI for ST segment elevation myocardial infarct (MI); immediate/early for Non ST segment elevation acute coronary syndrome) has shown to reduce mortality and the heart failure incidence for the patients in the future.[1] Nevertheless, following revascularization either mechanical or pharmacology, a condition called cardiogenic shock (CS) might appear before or after reperfusion. Moreover, despite having successful revascularization, patient may suffer CS. In MI patients who suffer CS, the 30-day mortality is about 40-50%. Furthermore, since SHOCK (Should we emergently revascularize Occluded Coronaries for cardiogenic shock) trial published, this incidence has not changed much.[1] In addition, CS is one of the most reasons patients admitted to Cardiac Intensive Care Unit (CICU) beside respiratory failure in United States and Canada. Manifestation of non-cardiac illness like renal failure also shown to prolong CICU length of stay and increase in hospital mortality.[2]

When SHOCK trial was published, the only mechanical support used was intra-aortic balloon pump (IABP). Afterward, other devices (i.e. left atrial to femoral artery bypass, left ventricular assist device, axial left ventricular, right ventricular support and extracorporeal membrane oxygenation (ECMO) have been produced and extensively studied in CS population. To date, there are not any published randomized trials indicating circulatory support devices change the outcome mortality in clinical state (3-9 pada Baran). Hence, CS mortality still remains excessively high. The possible explanations why it is difficult to show benefit of these devices because the etiologic of CS patients is varied, from the myocardial infarction, myocardial dysfunction in critically ill patients, or patients ongoing cardiac arrest. Thus, the prognosis and outcomes may vary in these different patient subsets. Another concern of CS patients is the treatment challenge.[1]

The Combination of the right medications and mechanical circulatory support devices would improve the CS patient prognosis. However, choosing the most appropriate management requires good hemodynamic understanding.[3] Therefore, this article will review the importance of hemodynamic profile for guiding management to yield the best outcome of the CS patients.

Cardiogenic shock

Definition

Cardiogenic shock (CS) is a condition where the primary cardiac disorder makes critical end organ hypoperfusion and hypoxia. It is a complicated state caused by the decline of cardiac output as the result of cardiac disease. Basically, the diagnose of CS can be made based on clinical characteristic such as persistent hypotension which doesn't give adequate response to fluid resuscitation and also companied by any sign of end organ hypoperfusion, for the example cold extremities, change of consciousness, or oliguria. Besides, some biomarkers can be detected as the sign of inadequate tissue perfusion, such as elevated

the lactate serum.[2,4] Hemodynamic parameters, such as reduced in cardiac index and elevated pulmonary wedge pressure (PCWP), are proven to help established CS diagnose and also important for defining Right Ventricular (RV) function in CS patients[4] Previous study use the marker of cardiac output and tissue perfusion, determined either clinically, invasively, or biochemically, to defined CS. However, according to SHOCK trial, CS diagnose is made when patients experience: (1) persistent hypotension (systolic blood pressure (SBP) less than 90 mmHg or there is requirement of vasopressor to maintain SBP >90 mmHg); (2) cardiac output (CO) reduction (<1.8 L/min/m^2 without support or 2.0 to 2.2 L/min/m^2 with no support) in the presence of elevated left ventricular end-diastolic pressure (LVEDP).[5] The European Cardiology Society (ESC) Guideline and selected major clinical trial definition of CS are shown on the Figure 1.

Table I Definition of cardiogenic shock in clinical trials and guidelines

SHOCK[13]	TRIUMPH[14]	IABP-SHOCK II[8]	CULPRIT-SHOCK[9]	ESC heart failure guidelines[15]
I. a. SBP <90 mmHg for ≥30 min or b. Support to maintain SBP ≥90 mmHg and II. Endorgan hypoperfusion (urine output <30 mL/h or cool extremities and heart rate >60 b.p.m.) III. Haemodynamic criteria[a]: a. CI of ≤2.2 L/min/m^2 and b. PCWP ≥15 mmHg	I. Patency of IRA spontaneously or after PCI II. Refractory cardiogenic shock >1 h after PCI with SBP <100 mmHg despite vasopressors (dopamine ≥7 μg/kg/min or norepinephrine or epinephrine ≥0.15 μg/kg/min) III. Endorgan hypoperfusion IV. Clinical or haemodynamic criteria for elevated left ventricular filling pressure V. LVEF <40%	I. SBP <90 mmHg for ≥30 min or catecholamines to maintain SBP >90 mmHg and II. Clinical pulmonary congestion and III. Impaired endorgan perfusion with at least one of the following criteria: a. Altered mental status b. Cold/clammy skin and extremities c. Urine output <30 mL/h d. Lactate >2.0 mmol/L	I. Planned early revascularization by PCI II. Multivessel coronary artery disease defined as >70% stenosis in at least two major vessels (≥2 mm diameter) with identifiable culprit lesion III. a. SBP <90 mmHg for >30 min or b. Catecholamines required to maintain SBP >90 mmHg IV. Pulmonary congestion V. Impaired organ perfusion with at least one of the following criteria: a. Altered mental status b. Cold/clammy skin and extremities c. Urine output <30 mL/h d. Lactate >2.0 mmol/L	SBP <90 mmHg with adequate volume and clinical or laboratory signs of hypoperfusion *Clinical hypoperfusion*: Cold extremities, oliguria, mental confusion, dizziness, and narrow pulse pressure. *Laboratory hypoperfusion*: Metabolic acidosis Elevated lactate Elevated creatinine

[a]Not required in anterior infarction or if pulmonary congestion in chest X-ray.
CI, cardiac index; ESC, European Society of Cardiology; IRA, infarct related artery; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure.

Figure 1. The definition of cardiogenic shock based on selected clinical trial and ESC guidelines[4]

Classification

The Society for Cardiovascular Angiography and Interventions (SCAI) direct more uniform definition of CS and also gives a classification scheme similar to the INTERMACS classification which describing profile of advance heart failure patients.[4,6] Based on this SCAI new definition, there are five categories of CS patients range from at risk to extreme CS labelled as A – E (Figure 2). The new classification system will help make a better comparison of different trial in CS and can also stimulate new studies on the pre-shock state CS patients.[4]

The new classification from SCAI helps providing a simple schema to describe patient's status and also make researcher differentiate these subset of CS patients easily. This classification made based on expert consensus and categorized CS patients with at risk of CS into extreme CS condition (which the patients have worsening stage of hemodynamic compromised) purposely to facilitate patient's treatment and clinical research.[4,7] The SCAI CS classification has some advantages, first it was simple and doesn't need any calculation. Second, it is suitable for rapid assessment so that the schema can be applied rapidly at the bed side when patient's condition getting worse. The last, it has prognostic value related to each CS shock state which reflect different morbidity and mortality ranking.[4] A retrospective study, including 10 004 patients admitted to CICU at Mayo Clinic between 2007 and 2015, showed that there was an increase in unadjusted CICU and hospital mortality along with higher SCAI shock stage. The result tells us that hospital mortality was rise from 3.0% in patients with SCAI CS stage A to 67.0% in stage E.[7]

The developed of this novel classification is inspired by the American College of Cardiology/American Heart Association (ACC/AHA) HF classification and the INTERMACS classification. The INTERMACS classification

is easy to use caused it has some key tag to help clinician memorized ways to categorize patients. Profile 1 INTERMACS annotated as "crash and burn", 2 is "sliding on inotropes", and 3 is "dependent stability". But, INTERMACS classification doesn't distinguish patients who placed on ECMO support due to refractory cardiac arrest or who are stable with multiple inotropes and IABP or patients which on IMPELLA catheter while use inotropes to improves CO. In addition, INTERMACS is design to classify patients on single timepoint so that it can't be used to re-assessed patients who experience deterioration condition. Besides, the heterogeneity of patients describes as INTERMACS 1, make it difficult to compare research outcomes.[1]

TABLE 1 Descriptors of shock stages: physical exam, biochemical markers and hemodynamics

Stage	Description	Physical exam/bedside findings	Biochemical markers	Hemodynamics
A At risk	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for its development. These patients may include those with large acute myocardial infarction or prior infarction acute and/or acute on chronic heart failure symptoms.	Normal JVP Lung sounds clear Warm and well perfused • Strong distal pulses • Normal mentation	Normal labs • Normal renal function • Normal lactic acid	Normotensive (SBP≥100 or normal for pt.) If hemodynamics done • cardiac index ≥2.5 • CVP <10 • PA sat ≥65%
B Beginning CS	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	Elevated JVP Rales in lung fields Warm and well perfused • Strong distal pulses • Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP <90 **OR** MAP <60 **OR** >30 mmHg drop from baseline Pulse ≥100 If hemodynamics done • cardiac index ≥2.2 • PA sat ≥65%
C Classic CS	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support, including ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.	***May Include Any of:*** Looks unwell Panicked Ashen, mottled, dusky Volume overload Extensive rales Killip class 3 or 4 BiPap or mechanical ventilation Cold, clammy Acute alteration in mental status Urine output <30 mL/h	***May Include Any of:*** Lactate ≥2 Creatinine doubling **OR** >50% drop in GFR Increased LFTs Elevated BNP	***May Include Any of:*** SBP <90 **OR** MAP <60 **OR** >30 mmHg drop from baseline **AND** drugs/device used to maintain BP above these targets Hemodynamics • cardiac index <2.2 • PCWP >15 • RAP/PCWP ≥0.8 • PAPI <1.85 • cardiac power output ≤0.6
D Deteriorating/ doom	A patient that is similar to category C but are getting worse. They have failure to respond to initial interventions.	***Any of stage C***	***Any of Stage C AND:*** Deteriorating	***Any of Stage C AND:*** Requiring multiple pressors **OR** addition of mechanical circulatory support devices to maintain perfusion
E Extremis	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near Pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	**"Trying to die"** CPR (A-modifier) pH ≤7.2 Lactate ≥5	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support

Figure 2. Description of SCAI cardiogenic shock classification based on physical examination, biomarker, and hemodynamic profile[1]

The Scheme Classification

As showed on the Figure 2, the SCAI classification schema divided CS into 5 stages labelled A – E which will describe below:

1. Stage A (At Risk of CS)

   This stage describes a patient who doesn't experience any sign or symptoms of CS yet but is at risk to developed CS. The stage A patients may have normal physical and laboratory examination. For the example, patients with small infarct may appear well but some of them can fall into CS due to pre-existing left ventricular dysfunction.

2. Stage B (Beginning CS)

   It describes patients who already have pre-shock or compensatory shock characterized by some clinical evidence such as relative hypotension (SBP<90 mmHg OR mean arterial pressure (MAP) ≤60 mmHg or >30 mmHg drops from baseline) or tachycardia without sign of hypoperfusion. The hypoperfusion defined by cold or clamped extremities, oliguria, or mental state deterioration. The physical exam may be showed mild volume overload and the laboratories result can be normal.

3. Stage C (Classic CS)

   Patients with stage C experience hypoperfusion state which requires an initial therapy, such either

inotropes or mechanical support, besides volume resuscitation in order to improve perfusion. These patients mostly present with classic shock phenotype (MAP ≤ 60 mmHg or SBP ≤ 90 mmHg) companied by hypoperfusion sign. The laboratory examination may show elevated lactate serum, creatine, brain natriuretic peptide (BARAN), and/or liver serum. When invasive hemodynamic was performed, it demonstrates depressed cardiac index associated with CS.

4. Stage D (Deteriorating or Doom CS)

   These stage describing patients who has failed to stabilized despite intense initial intervention that has had given. In this stage, the patients must already accept some degree of proper treatment or medical stabilization, but after 30 minutes past, the patient still not responded with improving on hypotension or hypoperfusion of end organ state. Patients with stage D CS needs further escalation therapy, such as increasing dose or number of intravenous medication or mechanical circulatory support.

5. Stage E (Extremis CS)

   Patients in stage E experience circulatory collapse, frequently with refractory cardiac arrest or ongoing cardiopulmonary resuscitation (CPR) or being supported by multiple mechanical support simultaneously (including ECMO-facilitated CPR). These patients have multiple clinicians to treat multiple simultaneous disorder due to their unstable condition.[1,4]

Pathophysiology

Pathophysiology of CS still poorly understood due to lack of high-quality clinical data. But basically, the pathogenesis is different depending on its etiologic.[8] Based on previous studies, the most common etiologic of CS is Acute Coronary Syndrome (ACS).[7,9] The SHOCK trial find that acute left ventricular failure related to an STEMI, especially anterior myocardial infarct, is the largest cause of CS which count about 79% of all CS patients. Besides, mechanical complication of ischemic heart disease (IHD) such as severe mitral regurgitation, right ventricular failure, ventricular septal rupture, and tamponade also quite often become the etiology of CS. Some non-ischemic etiologic, as showed on figure 3, can also resulting CS. This condition may be considered when there is a patient presenting with typical sign of CS but without any abnormality on ECG examination or there is no specific finding on their ECG and cardiac biomarker for myocardial infarct was negative.[9]

The pathogenesis of CS caused by myocardial infarction generally explained by profound depression on myocardial contractility due to reduce of cardiac output, low blood pressure, companied by further coronary ischemia which at the end resulting on additional reduction on contractility. This vigorous cycle can lead to a fatal condition such as cardiac arrest event death. This is a classic paradigm that including compensatory mechanism which is systemic vasoconstriction induced by acute cardiac injury and inadequate stroke volume. Current evidence also shown that disturbing on tissue microcirculation associated with mortality on the first 30-days and temporal changes of Sepsis-Related Organ Failure Assessment (SOFA) score which may be improved with mechanical circulatory support (MCS).[9] Although acute deterioration of the left ventricular (LV) contractility is commonly a main cause of CS, impaired on RV systolic function and deranged on the function of vasculature can also contribute to established and/or make CS worse. It is causing by the reduce of CO which affect coronary perfusion and resulting to further decreased of myocardial contractility.[5] Further understanding on CS pathogenesis together with proper hemodynamic assessment can lead to proper CS therapy and make patient's outcome better.

Table 1
Nonischemic etiologies of cardiogenic shock

Etiology	Examples
Pharmacologic	Beta blockers Calcium channel blockers Digoxin toxicity
Primary ventricular dysfunction	Acute myocarditis Stress cardiomyopathy (ie, Takatsubo cardiomyopathy) Nonischemic cardiomyopathy (eg, sarcoidosis, amyloidosis, hemochromatosis)
Outflow obstruction	Valvular stenosis Left ventricular outflow obstruction (eg, in hypertrophic cardiomyopathy)
Acute valvular regurgitation	Trauma Degenerative disease Endocarditis
Endocrine	Severe hypothyroidism
Pericardial disease	Cardiac tamponade Pericardial constriction
Tachyarrhythmias	Supraventricular/atrial tachyarrhythmias Monomorphic VT Polymorphic VT (ie, Torsades de Pointes)
Bradyarrhythmias	Sinus node dysfunction (eg, sick sinus syndrome) AV node dysfunction (eg, AV nodal block)

Abbreviations: AV, atrioventricular; VT, ventricular tachycardia.

Figure 3. Non-ischemic etiologic of CS[9]

The Role of Hemodynamic Profile on Cardiogenic Shock

The diagnosis of cardiogenic shock can only be established in condition which shock is caused by low cardiac output/index and not due to hypovolemia. Although CS diagnose can made clinically, but it is often difficult to assess volume status without invasive hemodynamic monitoring. Measuring cardiac output and intracardiac pressure is necessary when CS is considered. Current data

suggest that using Pulmonary Artery (PA) catheter may lower mortality in CS patients.[1] PA catheters can measure right atrial (RA), PA, and PCWP directly, cardiac output, systemic vascular resistance (SVR), etc. Non-invasive approach, such as echocardiography, can be helpful to identified any sign of right or left ventricular volume or pressure overload and also determined systolic or diastolic dysfunction that increasing the risk of CS.[1,3] Echocardiography must be performed as one of initial evaluation with suspected CS patients primary to asses intravascular volume status, LVEF, and also pericardial effusion or any obstructive lesion. Using echocardiography to asses inferior vena cava (IVC) can giving information about intravascular volume status and estimating RA pressure. The IVC diameter <2.1 cm and collapses >50% during inspiration suggest a hypovolemia and RA pressure value between 0 and 5 mmHg, whereas if IVC diameter >2.1 cm with <50% collapse at inspiration suggest a RA pressure greater than 10 mmHg.[3]

The Cardiogenic shock, based on the definition of the National Cardiovascular Data Registry, is established when systolic blood pressure ≤ 90 mmHg and cardiac index was less than 2.2 L/min/m^2 and/or there is requirement of intravenous inotropic or vasopressor agent or mechanical support in order to maintain blood pressure or cardiac index above these levels. There are four different hemodynamic type (Figure 4) of CS that difficult to classified unless using invasive hemodynamic monitoring. Moreover, CS patients may change from one category to another one. Beside this category, about 5% patients having uncommon types of CS that is right ventricular shock and normotensive shock. Initial study of CS describing the patient with heart failure (HF) and elevated central venous pressure (CVPs), but with current technology of invasive hemodynamic measure, the CS patient can further be characterized by a low of cardiac index, an elevated of SVR, and a high PCWP called classic "cold and wet" profile which become the most frequent hemodynamic phenotype of CS accounted about two thirds of AMICS patients. This further classification, tell us how important the hemodynamic profile on CS patient, especially to guide a proper therapy. [3,9]

		Volume Status	
		Dry	Wet
Peripheral Perfusion	Warm	**Vasodilatory shock** (not CS) Increased cardiac index, low SVRI, low/ normal PCWP	**Mixed CS** Low cardiac index, low / normal SVRI, Elevated PCWP
	Cold	**Euvolemic CS** Low Cardiac index, high SVRI, low / normal PCWP	**Classic CS** Low cardiac index, High SVRI, Elevated PCWP

Figure 4. Difference Hemodynamic Profile of CS[3]

Pulmonary artery catheter is critically important on this setting to differentiate CS from shock cause by others, to unmask normotensive CS patients as well as determine filling pressure accurately. In addition, PA catheter can also help assess right ventricular function on MI, distinguish classic CS from mixed shock, selecting patients who may have benefit from mechanical supporting, assist the titration of inotrope or vasopressor medication, and also to guide its weaning. But, the use of PA catheter in CS is still remain controversial especially on wider setting. A Study of 89 718 national inpatient sample, showed that only 6.1% AMI with CS patients who received PA catheter and they didn't find any benefit against mortality in this population patients. However, this result was limited due to selection bias where the hemodynamic monitoring is only given to the sicker patients who has the worse prognosis.[3]

Cardiogenic shock management

The optimal management of CS require careful investigation of the etiology and hemodynamic status. Initial goals of the therapy are achieving euvolemia and hemodynamic stabilization in order to optimize end organ perfusion so that multiorgan dysfunction can be avoid. Managing and prevent multiorgan system dysfunction is an important and favorable outcome to achieve in CS patients. a retrospective cohort over 15-years period (2000-2014) by Vallabhajosyula *et al* showed that 31.9% patients AMICS patients was complicated by multiorgan failure. The presence of this complication was associated with 2.23 fold increasing of in-hospital mortality, needed of resource utilization, and less fewer survival into discharge.[2]

The SHOCK trial result showed that therapeutic intervention is the most effective therapy in AMI patients complicated by CS (AMICS) with the most prominent benefit will achieve if the coronary reperfusion was given as early as possible. There was a significant reduction of mortality after 6-months, 1 and 6-years follow up.[4,9] In addition, since the wide spread use of early revascularization, multiple registries already confirmed the significant decrease of mortality from 70 – 80% to 40-50%.[4]

Pharmacotherapy of CS patients including intravenous fluids (IVFs), inotropes, and vasopressor. The role of this medication is primary supportive because there is no randomized trial successfully proof the improvement of CS patients when this therapy given alone. The medical therapy should be used to achieve target MAP between 65 and 75 mmHg. The fluid therapy can also be given to maintenance optimize cardiac filling pressure and euvolemia status. But the dose of IVFs should be individualized for each patient based on hemodynamic parameters, clinical judgement, and echocardiography result. The inotropes and vasopressor medication using to improves CO and tissue perfusion especially by increasing of myocardial contractility and SVR.[3,5]

Medical circulatory support (MCS) may be helpful on CS management in condition there is enough knowledge of fundamental hemodynamic principle. The left ventricle pressure-volume status and any condition change will provide a foundation to understand the hemodynamic deterioration of CS and the mechanism of support device. When EDV and ESV increase followed by decreased of stroke volume and end systolic pressure, the LV

contractility and output are reduced. In this condition, the MCS device is expected to alter hemodynamic so that the CO can improved and perfusion pressure will be normalized. Improving of end organ perfusion will prevent multiorgan failure which is an important step approach of CS management that can lead to improving of prognosis.[2,8]

Conclusions and future direction

Accurate identification of the hemodynamic profile together with collaborative multidisciplinary team is important to the management of CS related to its complexity and high mortality rate. The hemodynamic phenotype and multidisciplinary approach are needed to optimize pharmacologic and nonpharmacologic therapy in CS patients. Intervention managements are aim to limiting myocardial damage, supporting the heart which already failing, and interrupting processes leading to more progressive dysfunction so that irreversible burden of injury can be prevent. Although the initial diagnosis of CS can be made clinically, but invasive hemodynamic monitoring is critically important to tailored this multidisciplinary approach involving intervention, mechanical, and procedural therapy. Consideration of emerged revascularization, supporting of the failing end organ and heart is mandatory to prevent a progressive cardiovascular collapse and also end organ injury that is irreversible.[2,8,9]

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