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Review

Cardiogenic Shock

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Abstract
Cardiogenic shock (CS) is the most common cause of death in hospitalized patients with acute myocardial infarction (AMI). The incidence and mortality of CS in hospitals is also high, although advanced therapy is widely used in CS patients. CS is a condition characterized by inadequate cardiac output due to primary cardiovascular diseases, leading to clinical and biochemical manifestations of insufficient tissue perfusion. CS complicates 5–10% of AMI. STEMI increases the risk of CS approximately twice as much as NSTEMI. In the last 10 years, in-hospital mortality due to CS that occurs in AMI has not changed, that is at 40-50%. The pathophysiology of CS shows several overlaps and can occur simultaneously, that is starting with a cardiac insult that reduces cardiac output, central hemodynamic changes, microcirculatory dysfunction, systemic inflammatory response syndrome, and multi-organ dysfunction. CS classification based on SCAI, divided into 5, that's A(at risk), B(beginning CS), C(classic CS), D(deteriorating), and E(extremis). The key to managing CS is treating the patient as soon as possible, as each higher SCAI shock stage was associated with increased hospital mortality. All patients with suspected ACS-associated CS should have an early invasive strategy with appropriate revascularization. Vasoactive medicines have the potential to improve hemodynamics but at the expense of increased myocardial oxygen consumption and arrhythmogenic risk. Mechanical circulatory support (MCS) has insufficient data as the first-line device solution for CS those patients. However, the use of durable MCS devices in a bridge-to-bridge strategy is becoming more prevalent and is supported by clinical recommendations. APACHE-III and SAPS-II, had the best mortality discrimination values to assess the outcome in CS patients.

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Keywords: cardiogenic shock, classification, heart failure, myocardial infarction, SCAI.

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Introduction

Cardiogenic shock (CS) is the most serious complication of acute myocardial infarction (AMI) and the most common cause of death in hospitalized patients with AMI. The incidence of CS in the last few years has also continued to increase.[19] Although revascularization in AMI patients is widely performed and other advances in therapy, in-hospital mortality from CS remains very high, around 40-50%.[15]

Based on that condition, the authors are interested in discussing and summarizing related to CS, starting from the definition, epidemiology, pathophysiology, classification based on SCAI, management, prognosis and outcome in CS those patients. Articles related to CS were collected and summarized, then compiled into a review article. This is expected to provide an accurate description and selection of appropriate management in patients with CS.

Definition

CS is a condition characterized by inadequate cardiac output due to primary cardiovascular diseases, leading to clinical and biochemical manifestations of insufficient tissue perfusion.[23] Clinical manifestations can be found in the form of signs of hypoperfusion, such as mental confusion, dizziness, narrow pulse pressure, cold extremities, and oliguria. In addition, it can also be characterized by the presence of persistent hypotension, which is unresponsive to the presence of volume replacement and requires pharmacological or mechanical intervention.[23] As for the biochemical manifestations, there is an increase in serum lactate, creatinine, and metabolic acidosis, which conditions reflect hypoxia in tissues and changes in cellular metabolism, which have the potential to cause organ dysfunction.[5] Clinical criteria from several guidelines/ trials can also be seen in the following table **(Table 1)**.

Epidemiology

CS complicates 5 - 10% of acute myocardial infarction. STEMI increases the risk of CS approximately twice as much as NSTEMI.[22] The incidence of CS originating from acute coronary syndrome (ACS) complications is about 4-12%, which is divided into 30-40% at the time of admission and 60-70% during hospitalization.[5] In the last 10 years, in-hospital mortality due to CS that occurs in AMI has not changed, that is at 40-50%, with a higher value during hospitalization.[5] Since 2005 the incidence has continued to increase and the mortality rate since 1975 is down but remains high, with 1 in 2 people not surviving.[7,20] Mortality rates in hospitals vary from 30% and 60%,

Table 1. Clinical Definitions of CS.[23]

ESC HF Guideline	SHOCK Trial (In the setting of MI complicated by predominantly LV dysfunction)	IABP-SHOCK II (In the setting of acute MI)
SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion • **Clinical hypoperfusion:** Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure • **Laboratory hypoperfusion:** Metabolic acidosis, elevated serum lactate, elevated serum creatinine	**1. Clinical criteria:** SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND end-organ hypoperfusion (urine output <30 mL/h or cool extremities) **2. Hemodynamic criteria:** CI of ≤2.2 L/min/m2 AND PCWP ≥15 mm Hg	**Clinical criteria:** SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)

ESC (European Society of Cardiology); HF (heart failure); SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock); IABP-SHOCK II (Intra Aortic Balloon Pump in Cardiogenic Shock II); LV (left ventricular); MI (myocardial infarction); CI (cardiac index); PCWP (pulmonary capillary wedge pressure); SBP (systolic blood pressure)

of which half occur within the first 24 hours.[5] Deaths occurring within 1 year from CS are about 50-60%, with mortality occurring in the first 30-60 days after the onset of CS at about 70-80%.[5]

ICCU data in the US and Canada, showed the most common etiology was ACS, where only one-third of the CS events whose etiology is due to ACS, the remaining 18% are ischemic cardiomyopathy without ACS, 28% are non-ischemic cardiomyopathy, and 17% other cardiac causes.[5]

Pathophysiology

The pathophysiology of CS shows several overlaps and can occur simultaneously, that is starting with a cardiac insult that reduces cardiac output, central hemodynamic changes, microcirculatory dysfunction, systemic inflammatory response syndrome, and multi-organ dysfunction.[5]

The main thing that occurs in CS, that is the contractility of the myocardium is reduced **(Figure 1)**, resulting in decreased cardiac output, hypotension, tachycardia, systemic vasoconstriction, and cardiac ischemia.[9,22] Ineffective cardiac output will cause peripheral vasoconstriction, which is the initial compensation of CS, aiming to increase coronary and peripheral perfusion.[22] These compensatory mechanisms started with sympathetic stimulation to increase heart rate and contractility, as well as fluid retention in the kidneys to increase preload.[9] As CS progresses As this condition develops, these mechanisms become maladaptive and actually worsen the patient's condition. An increase in heart rate and contractility increases myocardial oxygen demand and exacerbates ischemia.[9] This tachycardia and ischemia cause fluid retention and impaired diastolic filling, which can lead to hypoxia and pulmonary congestion.[9] Vasoconstriction conditions that occur to maintain blood pressure, cause an increase in myocardial afterload, which further interferes with the work of the heart and increases myocardial oxygen demand.[9,22] This increase, due to inadequate perfusion, further exacerbates ischemia and starts a vicious cycle, if not stopped it will end in death.[9,22] This decrease in cardiac output also results in impaired systemic perfusion, which can lead to further impairment of systolic performance and the development of lactic acidosis.[9]

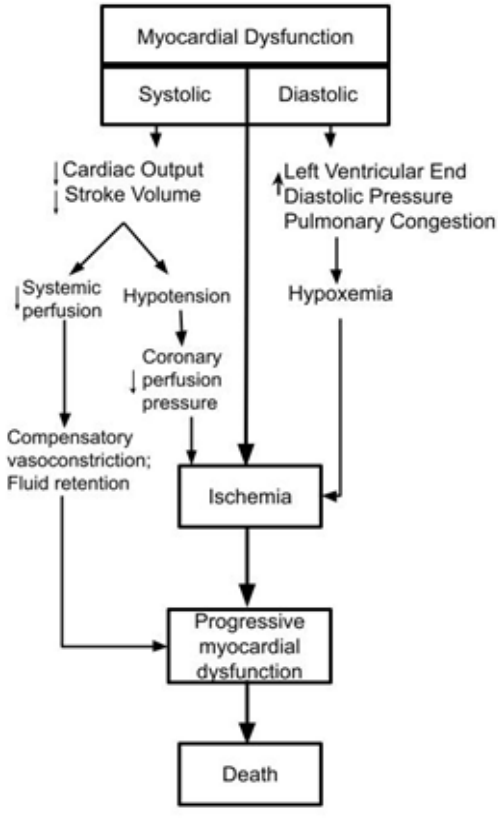

Figure 1. The downward diagram in cardiogenic shock (modified from Hollenberg et al.,1999).

The presence of systemic inflammation, in CS, releases NO synthase and peroxynitrite, which has a cardiotoxic inotropic effect, leading to pathological vasodilation. Other inflammatory mediators, such as interleukins and TNF-alpha, also cause vasodilation and, in CS, may contribute to patient mortality.[9,22]

Classification

CS can be categorized as pre-CS, CS, and refractory CS based on clinical severity and response to treatment. The presence of clinical findings of peripheral hypoperfusion, despite a normal SBP, is referred to as "pre-shock" and precedes the onset of clinical shock. "Refractory CS" is a condition that has been defined as CS with continuous evidence of tissue hypoperfusion after administration of appropriate dosages of two vasoactive medicines and therapy of the underlying etiology. Based on this definition, refractory CS is at the worst end of the severity scale. Early detection

Table 2. Descriptors of shock stages.[1]

Stage	Physical exam	Biochemical markers	Invasive hemodynamic
A "At risk"	Normal physical findings	Normal laboratory result	Normotensive Cardiac index ≥2.5 CVP <10 PA sat ≥65%
B "Beginning CS"	Elevated JVP Rales lung sound Warm and well perfused	Normal lactate Minimal renal function impairment Increased BNP	Hypotension Cardiac index ≥2.2 PA sat ≥65%
C "Classic CS"	Cold, clammy extremities Volume overload Extensive rales Urine output <30 mL/h Mental status impairment	Decreased kidney function Lactate ≥ 2 Elevated BNP Abnormal liver enzymes	Hypotension Cardiac index <2.2 PCWP >15 RAP/PCWP ≥0.8 PAPI <1.85 Cardiac power output ≤0.6
D "Deteriorating"	Any of stage C	Any of stage C and deteriorating	Any of Stage C and: Multiple pressors or the addition of mechanical circulatory support devices are required to sustain perfusion.
E "Extremis"	Near pulselessness Cardiac collapse	pH ≤7.2 Lactate ≥5	Undetectable SBP without resuscitation ECG shows PEA or VT/VF Hypotension persists despite maximal support

of CS permits immediate intervention to reverse the underlying cause and introduce supportive therapy.[5]

The Society for Cardiovascular Angiography and Interventions (SCAI) classification specifies five evolutive stages of CS, from A (at risk of CS) to E (extremis), including a modifier for cardiac arrest (CA). Stage A defines a patient who does not exhibit any indications or symptoms of CS but is at risk for developing it. This classification may include patients with non-STEMI, previous MI, and decompensated heart failure. Stage B refers to a patient who shows relative hypotension or tachycardia in the absence of hypoperfusion. Stage C is a patient with hypoperfusion who requires an initial series of therapies (inotropes, pressors, mechanical support, or ECMO) to restore perfusion. Stage D means that the initial set of interventions chosen has not restored stability and adequate perfusion despite at least 30 minutes of observation and requires additional escalation. Stage E refers to a patient who is extremely unstable and frequently experiencing cardiovascular collapse, often (but not always) in refractory cardiac arrest with continuous cardiopulmonary resuscitation (CPR) or supported by various concurrent acute treatments including ECMO-facilitated CPR (eCPR).[1]

The proposed SCAI Classification of CS aims to create a simple schema that would facilitate clear communication regarding patient status and allow clinical studies to accurately identify patient subsets. In all clinical settings, this classification can be implemented promptly at the bedside upon patient presentation. The SCAI classification uses bedside clinical assessment of hypoperfusion, biochemical markers monitoring, and invasive hemodynamic examination.[5]

Management of Cardiogenic Shock

The key to managing CS is treating the patient as soon as possible, as each higher SCAI shock stage was associated with increased hospital mortality

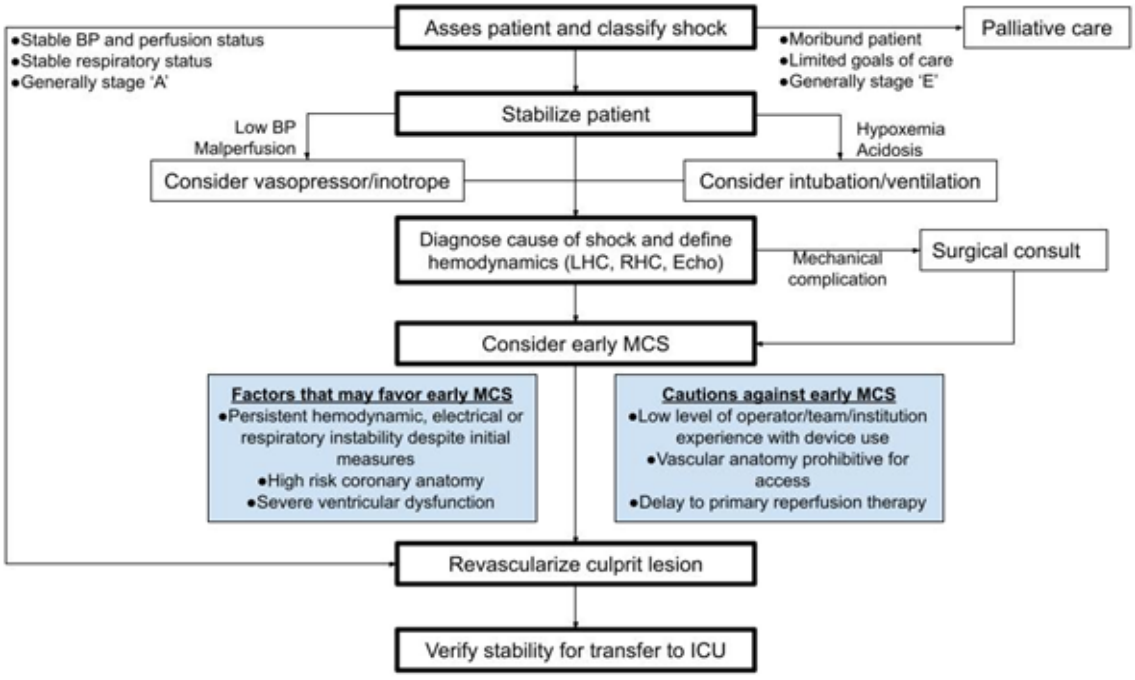

Figure 2. Algorithmic approach to the patient with acute myocardial infarction (AMI) complicated by cardiogenic shock (CS) (modified from Henry et al.,2021).
BP (blood pressure); LHC (left-sided heart catheterization); RHC (right-sided heart catheterization); Echo (echocardiography); MCS (mechanical circulatory support); ICU (intensive care unit).

compared to SCAI shock stage A **(Figure 2)**. The SCAI classification, including the presence or absence of CA, offered substantial hospital mortality risk stratification when assessed at the time of CICU admission. This classification method could be used as a clinical and research instrument to identify, communicate, and estimate the probability of death in patients with and at risk for CS.[12] The oxygenation and circulation are the first things to stabilize, and then physicians should treat the underlying etiology while monitoring vital signs. Patients with CS should be monitored to differentiate causes of hemodynamic instability, enable monitoring of the response to any therapeutic intervention, and determine if they need mechanical circulatory support.[5]

After stabilizing, all CS patients should be rapidly transferred to a tertiary care center that is capable of early invasive catheterization and a dedicated ICU/ICCU with the availability of short-term and long-term mechanical circulatory support. CS centers also should be high volume centers (>107 cases/year) with an experienced multidisciplinary team (MDT), availability of on-site operating rooms, and a nurse-to-patient ratio of 1:1 is also recommended, these factors are associated with improved outcomes. Early identification and treatment of the underlying cause may be advantageous for enhancing outcomes in CS patients.[5]

Reperfusion and Revascularization for Acute MI Patients with CS

The most effective therapeutic intervention for a patient with acute MI who presents with CS is coronary reperfusion. When an early invasive strategy cannot be done in a timely manner, fibrinolysis may be used for CS associated with STEMI. The decision to do fibrinolysis must be individualized depending on bleeding risk, expected reperfusion benefit, and predicted angiography delay time. Although there is a lack of high-quality evidence to support fibrinolysis in CS, fibrinolysis is commonly used in the treatment of CS. In MI with CS, the best way to restore blood flow to the heart is still

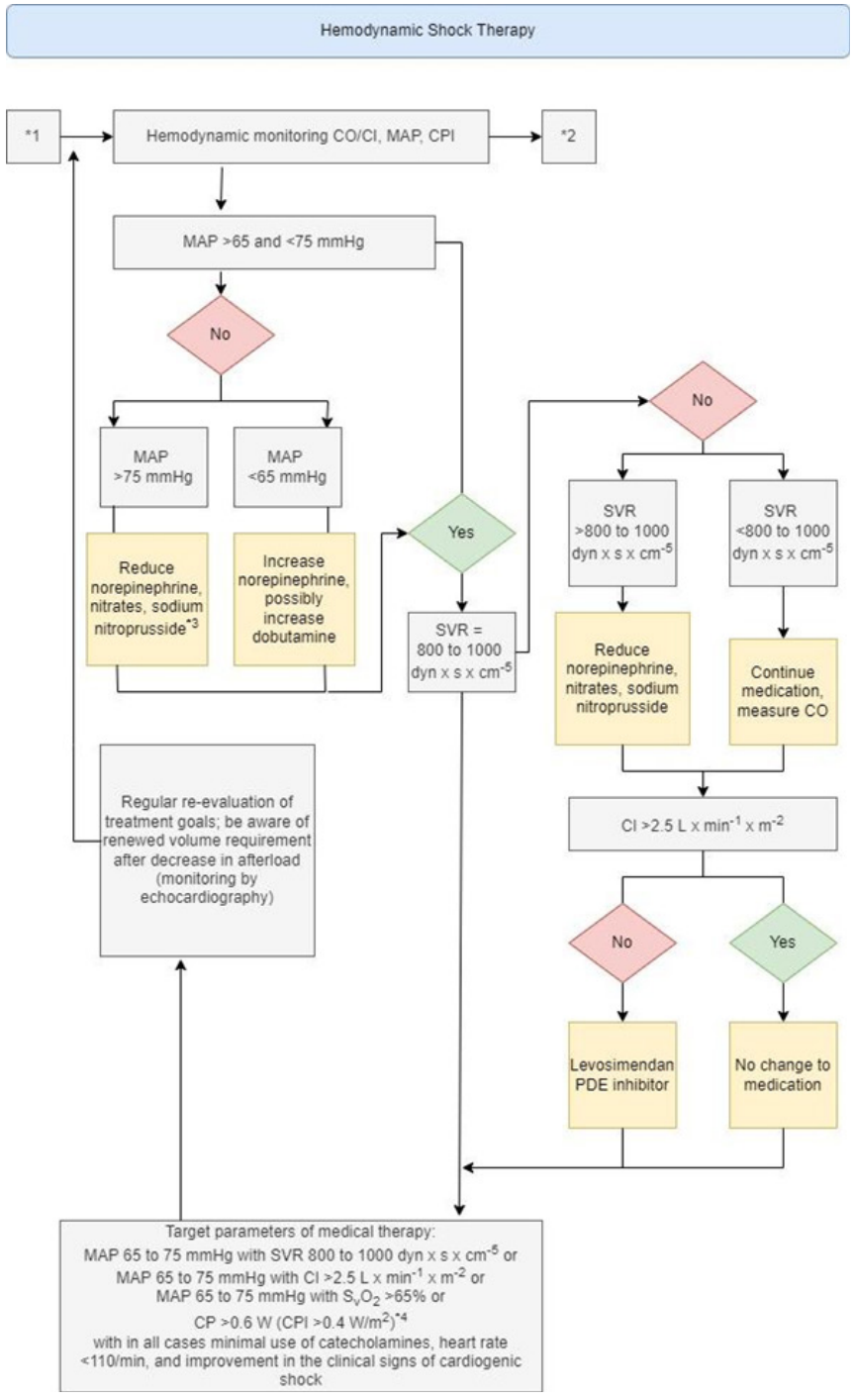

Figure 3. Hemodynamic shock therapy (modified from Werdan et al., 2012)
Hemodynamic shock therapy focuses on achieving adequate organ perfusion using the minimum of catecholamines.
*1 Shock after revascularization;
*2 Treatment of MODS (Multiorgan Dysfunction Syndrome);
*3 In patients with raised SVR, norepinephrine treatment is always ended before treatment with nitrates or sodium nitroprusside is started.
*4 CP > 0.6 W corresponds to a cardiac output of 5 L/min with an MAP of 65 mm Hg and SVR of 880 dyn × s × cm-5

through surgery.[23]

All patients with suspected ACS-associated CS should have an early invasive strategy with appropriate revascularization, including those with uncertain neurological status or those who have received prior fibrinolysis due to the time delay from MI onset.

In patients with MI-associated CS who have multivessel or left main disease, PCI or CABG revascularization decisions should be made collaboratively between cardiologists and surgeons, considering the patient's medical history, coronary anatomy, procedural risks, potential treatment-related delays, and expressed preferences.

Medical Treatment of the CS Patient

Inotropes and/or vasopressors are administered to around 80–90% of CS patients. Vasoactive medicines have the potential to improve hemodynamics but at the expense of increased myocardial oxygen consumption and arrhythmogenic risk. Therefore, the usual recommendation is to avoid using them once tissue perfusion has been restored and to limit the amount and duration of the infusion.[5] The following figure **(Figure 3)** shows recommendations for the use of inotropes and/or vasopressors in shock patients.[25]

Mechanical Circulatory Support

Mechanical Circulatory Support (MCS) devices can be classified either as temporary or permanent. Temporary MCS devices can be implanted percutaneously or surgically and used as a bridge to recovery, or a bridge to a bridge, in which case patients have a temporary device inserted and a plan to transition to a durable MCS following clinical stabilization. Surgically implanted, long-lasting MCS devices can be used as a bridge to recovery or as the final treatment. In the CS population, the regular use of MCS devices as a therapeutic adjunct is not supported by high-quality evidence. Therefore, we completely agree with the American Heart Association and the International Society of Heart and Lung Transplantation in recommending that patients with persistent CS, with or without end-organ hypoperfusion, be evaluated for MCS candidacy by a multidisciplinary team with expertise in MCS device selection, implantation, and management.[5]

There are currently insufficient data to decide if durable MCS should be the first-line device solution for patients with CS. However, the use of durable MCS devices in a bridge-to-bridge strategy is becoming more prevalent and is supported by clinical recommendations. After temporary or permanent implantation of an MCS device, heart transplantation may be conducted on appropriate patients whose heart function is not expected to recover.[23]

Mechanical Ventilatory Support

Acute respiratory failure is found in nearly all patients with CS. Hypoxemia and hypercapnia originate from intrapulmonary shunting due to pulmonary congestion, the reduction in lung space with increasing ventilation–perfusion mismatch, and the change of respiratory drive due to cerebral hypoperfusion. In addition, lactic acidosis raises the respiratory load by compensating for hyperventilation, hence raising the organism's overall oxygen demand. The decision to intubate CS patients must be based on critical care standards.[23]

Management of Liver Injury and Renal Dysfunction in CS Patient

Ischaemic hepatitis is the diffuse hepatic injury produced by a fast reduction in CO and is accompanied by a sharp increase in the serum levels of alanine aminotransferase, aspartate aminotransferase, and lactic dehydrogenase. Congestive hepatopathy, on the other hand, is frequently observed in patients with high venous pressure, particularly in CS patients with RV dysfunction. High amounts of direct bilirubin, gamma-glutamyl transferase, and alkaline phosphatase accompany it. However, these anomalies frequently coexist, and poor liver function in CS involves a confluence of congestion and decreased CO.[5] Since there isn't a specific treatment for liver damage in CS, the RV function, especially the decrease in pulmonary vascular resistance and right atrial pressure, needs to be taken into account.[3,10]

Acute kidney injury (AKI) affects around one-third of people with CS. However, many survivors of CS experience a gradual renal recovery. Systemic hypoperfusion, retrograde congestion, nephrotoxic medicines, contrast agents, and use of MCS in CS may cause AKI. Patients requiring renal replacement therapy had a reduced chance of surviving to hospital

discharge and a higher risk of having long-term dialysis and passing away. Hemodynamically, patients with CS typically cannot tolerate the fluid fluctuations that can occur during intermittent hemodialysis. Continuous renal replacement therapy, which utilizes a veno-venous driving force and an external pump to gradually remove fluid and toxins, is increasingly utilized for patients with CS. Continuous veno-venous hemodiafiltration is recommended for patients with severe AKI (creatinine 2 at baseline and urine output of 0.5 mL/kg/h for 12 h) or when there are life-threatening abnormalities in fluid, electrolyte, and acid–base balance.[5]

Prognosis And Outcome

The prognosis in patients with CS is adjusted according to the underlying etiology. A cohort study showed two common etiologies, that is acute decompensation of CHF and ACS. CS with acute decompensation of CHF etiology has a poorer prognosis, with a higher mortality rate after a 6-month follow-up. These results compared with CS occurring after ACS within the first 1 month of follow-up suggest a relatively good prognosis.[4]

Several parameters that can be used to assess the outcome in CS those patients are APACHE (Acute Physiological and Chronic Health Status Evaluation)-II, APACHE-III, and SAPS (Simplified Acute Physiology Score)-II. The results of a study comparing these three parameters, it was shown that APACHE III and SAPS II, had the best mortality discrimination values.[23] The GRACE (Global Registry of Acute Coronary Events) score can be used to predict the incidence of mortality in CS patients with ACS, in-hospital and long-term mortality, which has a good discriminatory value.[23]

The other scoring systems that are also proposed to assess mortality in CS are the CardShock risk score and IABP-SHOCK II (Intra Aortic Balloon Pump in Cardiogenic) risk score. These two scores can be used to determine the short-term mortality rate in CS patients. The IABP-SHOCK II risk score has a strong correlation with short-term prognosis and has 6 variables that have been shown to be predictors of 30-day mortality: age, history of the previous stroke, TIMI flow grade less than 3 after PCI, glucose, creatinine, and lactate levels at admission.[17]

Conclusion

BCS is characterized by inadequate cardiac output due to primary cardiovascular diseases, causing insufficient tissue perfusion. CS The pathophysiology includes a cardiac insult that reduces cardiac output, central hemodynamic changes, microcirculatory dysfunction, systemic inflammatory response syndrome, and multi-organ dysfunction. The SCAI classification creates a simple schema that allows clinical studies to accurately identify patient subsets. This classification specifies five CS stages, from A to E, with a CA modifier. Rapid diagnosis of CS patients can improve patient prognosis since hospital mortality increases with each higher SCAI shock stage. Therefore, the key to controlling CS is rapid treatment. Oxygenation and circulation should be stabilized initially, followed by treatment of the underlying cause while monitoring vital signs. Several scoring systems can be used to predict the prognosis of CS patients. In general, we strongly advise the clinical application of the SCAI classification.

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