Intensive Care Management of Cardiogenic Shock in The Critically-ill Patients

An Essay

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Contents

Page No.

List of Abbreviations

ACLI	Acute cardiogenic liver injury
ADHF	Acute decompensated heart failure
ADQI	Acute Dialysis Quality Initiative
AKI	Acute kidney injury
AlP	Aluminium phosphide
ALT	Alanine aminotransferase
AMI	Acute myocardial infarction
AR	Aortic regurgitation
ARDS	Acute respiratory distress syndrome
AST	Aspartate aminotransferase
ATP	Adenosine triphosphate
AV	Atrioventricular
BNP	Brain-type natriuretic peptide
Bpm	Beats per minute
CABG	Coronary artery bypass grafting
CAD	Coronary artery disease
cAMP	Cyclic adenosine monophosphate
CCB	Calcium channel blocker
CHF	Congestive heart failure
CO	Cardiac output
CRS	Cardiorenal syndrome
CS	Cardiogenic shock
CT	Computerized tomography
cTn	Cardiac troponin
DAMPs	Damage-associated molecular patterns
DCM	Dilated cardiomyopathy
DO_2	Oxygen delivery
ECG	Electrocardiogram
EDV	End-diastolic volume
EF	Ejection fraction

FGF 23	Fibroblast growth factor 23
FiO_2	Fraction of inspired oxygen
Hb	Hemoglobin
HF	Heart failure
HR	Heart rate
IABP	Intra-aortic balloon pump
ICU	Intensive care unit
IV	Intravenous
IVC	Inferior vena cava
LBBB	Left bundle branch block
LDH	Lactate dehydrogenase
LVAD	Left ventricular assist device
LVEF	Left ventricular ejection fraction
MAP	Mean arterial pressure
MR	Mitral regurgitation
MRI	Magnetic resonance imaging
NIPPV	Noninvasive positive-pressure ventilation
NSTACS	Non-ST segment elevation acute coronary syndrome
PAMPs	Pathogen-associated molecular patterns
PaO_2	Partial pressure of arterial oxygen
PAOP	Pulmonary artery occlusion pressure
PCI	Percutaneous coronary intervention
PCWP	Pulmonary capillary wedge pressure
PPCM	Peripartum cardiomyopathy
PRRs	Pattern-recognition receptors
pVADs	Percutaneous ventricular assist devices
PVCs	Premature ventricular contractions
RA	Right atrium
RCM	Restrictive cardiomyopathy
RV	Right ventricle
SAH	Subarachnoid hemorrhage
SaO_2	Arterial hemoglobin oxygen saturation

SK	Streptokinase
STEMI	ST segment elevation myocardial infarction
SV	Stroke volume
SVR	Systemic vascular resistance
TEE	Trans-esophageal echocardiography
TNK	Tenecteplase
TT	Thrombolytic therapy
TTE	Trans-thoracic echocardiography
VA ECMO	Veno-arterial extracorporeal membrane oxygenation
VO_2	Oxygen consumption
VSR	Ventricular septal rupture
VT	Ventricular tachycardia

List of Tables

List of Figures

INTRODUCTION

Cardiogenic shock is a consequence of severe cardiac pump failure and is characterized by decreased cardiac output (CO), increased systemic vascular resistance (SVR), and increased pulmonary capillary wedge pressure (PCWP) **(Givertz, 2014).**

The heart is central to the circulatory supply of O_2 and if the pump fails then there are few compensatory mechanisms available. Hence, cardiogenic shock has a very high in-hospital mortality rate ranging from 45–100%, depending on the etiology. Myocardial ischemia is the most common cause of cardiogenic shock, but other etiologies must be considered **(Maiden and Peake, 2014).**

Complications of acute myocardial infarction (AMI), such as arrhythmias, ventricular septal defects, papillary muscle dysfunction, or myocardial rupture causing pericardial tamponade, may also trigger the onset of shock. Less frequently, cardiogenic shock may be caused by severe cardiomyopathy (dilated, restrictive, or stress induced), acute myocarditis, or severe valvular disease (**Viswanathan and Bach, 2012).**

Any patient presenting with cardiogenic shock must receive an early working diagnosis, urgent resuscitation, and subsequent confirmation of the working diagnosis. In

addition to laboratory studies, workup in cardiogenic shock can include imaging studies such as echocardiography, chest radiography, and angiography; electrocardiography (ECG); and invasive hemodynamic monitoring **(Ren and Lenneman, 2017).**

Early and definitive restoration of coronary blood flow is the most important intervention for achieving an improved survival rate. Correction of electrolyte and acid-base abnormalities is essential in cardiogenic shock **(Ren and Lenneman, 2017).**

For cardiogenic shock, medications may be required to increase contractility (e.g. Inotropic and vasopressor medications), reduce afterload, maintain adequate systemic and coronary perfusion pressure, increase diastolic relaxation and increase or decrease heart rate **(Maiden and Peake, 2014).**

When intravenous (IV) medications are insufficient to maintain vital organ perfusion, consideration should be given to temporary mechanical circulatory support with an intra-aortic balloon pump (IABP) , percutaneous ventricular assist device (VAD) , or extracorporeal membrane oxygenation (ECMO) **(Givertz, 2014).**

AIM OF THE ESSAY

The aim of this essay is to put a reliable and conclusive discussion about cardiogenic shock as a serious, life-threatening clinical illness and several new modalities used for diagnosis, management and prognosis of such cases.

PATHOPHYSIOLOGY OF CARDIOGENIC SHOCK

Shock definition:

The answer commonly provided to the question 'what is shock?' is often a vague 'patient with a low blood pressure', followed by some threshold blood pressure below which 'shock' is said to be occurring. The reason for the uncertainty about what should be a simple concept relates to the fact that the term 'shock' is used in a multitude of contexts (e.g. septic, hemorrhagic, distributive, hypovolemic, cytotoxic, cardiogenic, anaphylactic, toxic, spinal, neurogenic, cervical and, even electrical shock). However, the unifying feature of shock, irrespective of the initiating disease or clinical features, is a failure to deliver and/or utilize adequate amounts of oxygen **(Maiden and Peake, 2014).**

Hence, cardiogenic shock is a life-threatening condition that should be treated as a medical emergency. The most common etiology is severe left ventricular (LV) dysfunction that leads to pulmonary congestion and/or systemic hypoperfusion **(Fig. 1) (Hochman and Ingbar, 2012).**

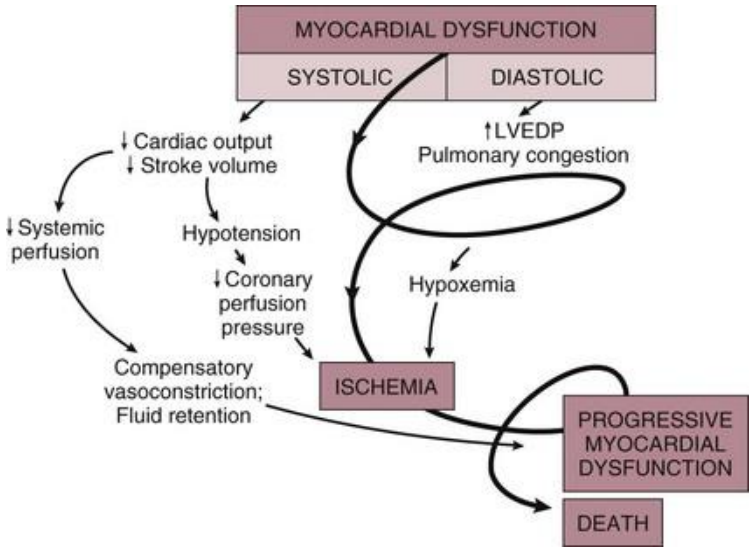

Fig. 1: Pathophysiology of cardiogenic shock **(Hochman and Ingbar, 2012).**

Cardiogenic shock (CS) is characterized by systemic hypoperfusion due to severe depression of the cardiac index (<2.2 L/min/m^2) and sustained systolic arterial hypotension (<90 mmHg) despite an elevated filling pressure (pulmonary capillary wedge pressure [PCWP] >18 mmHg). It is associated with in-hospital mortality rates >50%. The major causes of CS are listed in **Table 1 (Hochman and Ingbar, 2012).**

Table (1): Etiological causes of cardiogenic shock

Etiological causes of cardiogenic shock:
Acute myocardial infarction (Left ventricular infarction).
Acute valve dysfunction (e.g. chordae rupture, acute mitral regurgitation, acute aortic regurgitation).
Cardiomyopathies.
Myocarditis.
Myocardial contusion.
Right ventricular infarction.
Aortic dissection.
Ventricular septal rupture.
Ventricular free wall rupture.
Pericardial tamponading effusion.
Dysrhythmias (Bradyarrhythmias and tachyarrhythmias).
Toxic/metabolic (Severe acidosis, severe hypoxemia).
Drugs (e.g. Ca^{2+} channel blocker or beta blocker overdose).

(Hochman and Ingbar, 2012).

Cardiogenic shock due to left ventricular infarction suggests that more than 40% of the left ventricle is involved. On physical examination, signs of peripheral vasoconstriction are evident and oliguria is common. The typical hemodynamic profile includes systemic hypotension with decreased CO and elevated Pulmonary artery occlusion pressure (PAOP). Physical examination findings of pulmonary and peripheral edema as well as hepatomegaly may suggest volume overload, but are commonly due to third spacing of fluid due to shock with relative intravascular

volume depletion being present. In such situations, hemodynamic monitoring using echocardiography or a volumetric pulmonary artery catheter may provide additional diagnostic information clarifying the patient's true volume status **(Cheatham et al., 2012).**

Right ventricular dysfunction as a consequence of inferior wall myocardial infarction carries a better prognosis than left-sided failure. Diagnosis may be suggested by elevated right ventricular diastolic pressure with decreased pulmonary artery pressure. Hypotension caused by right-sided heart failure must be distinguished from left-sided failure because of the significant differences in their management. Shock from right-sided failure is corrected by volume resuscitation to maintain right ventricular preload while left-sided failure is treated by volume restriction to reduce myocardial work. If inotropes are indicated, agents that do not increase pulmonary vascular resistance should be chosen. Dysrhythmias are another source of cardiogenic shock. In addition to malignant dysrhythmias, such as ventricular fibrillation, atrial dysrhythmias such as atrial fibrillation or flutter as well as supraventricular tachycardia are common in the critically ill and may result in shortened diastolic filling time with a profound decrease in CO **(Cheatham et al., 2012).**