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Management of Cardiogenic Shock: A Review

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ABSTRACT
INTRODUCTION: Cardiogenic shock (CS) is defined as a state of critical end organ hypoperfusion due to reduced cardiac output.The most frequent cause is acute myocardial infarction (AMI) with subsequent ventricular dysfunction in about 80% of cases. In spite of the advances made in the treatment of AMI, cardiogenic shock remains a leading cause of death with mortality rates approaching 40- 50%.
AIM: The purpose of this review is to highlight the current concepts in the management of cardiogenic shock.
MATERIALS AND METHODS: A systematic review of published literature using PubMed and Med Line was done using search items like "cardiogenic and shock". Secondary references obtained from this publication were identified by manual search and reviewed as relevant.
RESULTS: Cardiogenic shock is characterized by inadequate tissue perfusion in the setting of adequate intravascular volume. The treatment involves general supportive measures which include; adequate oxygenation and ventilation, correction of electrolytes and acid-base abnormalities, pain reliefand restoration of sinus rhythm. Revascularizationand mechanical supports are also necessary.
CONCLUSION: The diagnosis and management of cardiogenic shock are difficult and require extensive knowledge and clinical experience. In spite of the significant advances made, the management still remains a challenge.
KEY WORDS: Cardiogenicshock

I. Introduction

Cardiogenic shock (CS) is defined as a state of critical end organ hypoperfusion due to reduced cardiac output[1]. The diagnostic criteria includes: i) Systolic blood pressure < 90mmHg for >30minutes or vasopressors required to achieve a blood pressure≥ 90mmHg ii) Pulmonary congestion or elevated left ventricular filling pressures iii)Signs of impaired organ perfusion with at least one of the following criteria a) altered mental status b)cold clammy skinc) oliguria d) increased serum lactate. Cardiac index(CI) and pulmonary capillary wedge pressure(PCWP) are usually required to make a diagnosis of cardiogenic shock. This easy to assess clinical criteria may be useful in making a diagnosis without advanced hemodynamic monitoring[2]. The most frequent causeof cardiogenic shock is acute myocardial infarction (AMI) with subsequent ventricular dysfunction in about 80% of cases[1].Less frequent causes include mechanical complications like ventricular septal defect (4%), free wall rupture(2%) and acute severe mitral regurgitation[3]. Other causes of CS include decompensated valvular heart disease, acute myocarditis, arrhythmias with heterogenous treatment options[1]. In spite of the advances made in the treatment of AMI , cardiogenic shock remains a leading cause of death with mortality rates approaching 40- 50%[4,5,6].The purpose of this review is to highlight the current concepts in the management of cardiogenic shock.

II. Materials And Methods

A systematic review of published literature using PubMed and MedLine was done using search items like "cardiogenic and shock". Secondary references obtained from this publication were identified by manual search and reviewed as relevant.

III. RESULTS

PATHOPHYSIOLOGY

Myocardial ischemia leads to abnormal functioning of the cardiac myocytes. This leads to further deterioration of the left ventricular function, creating a ''downward spiral''[7]. Inadequate pumping of the left ventricular myocardium following ischemia leads to a decline in stroke volume and cardiac output. The pump failure reduces the ability of the heart to push blood forward out of the ventricle, thereby increasing the ventricular diastolic pressure. This increase in ventricular diastolic pressure reduces coronary perfusion pressure, increases ventricular wall stress and myocardial oxygen requirement. This further worsens myocardial ischemia.[2,7] The cardiac pump failure and consequent hypoperfusion of the peripheral tissues causes the release of catecholamines such as noepinephrine. This results in an increase in the hearts contractility, constriction of arterioles and angiotensin II release with an aim of maintaining cardiac perfusion. This however leads to an increase in the hearts oxygen demand with proarrhythmic and myocardial toxic consequences[2]. The resultant ischemia from these processes increases diastolic stiffness of the left ventricular wall and along with left ventricular dysfunction increases

the left atrial pressure. The increased left atrial pressure propagates through the pulmonary vein causing pulmonary congestion which reduces oxygen exchange resulting in hypoxia. Hypoxia further worsens the ischemia of the myocardium. The pulmonary congestion propagates its effect through the pulmonary arteries to the right ventricle, thus jeopardizing its performance. Prolonged systemic hypoperfusion and hypoxia would cause a shift in cellular metabolism leading to lactic acidosis which inhibits cardiac contractility.

Right ventricular (RV) myocardial infarction accounts for about 5% of cases of cardiogenic shock[8] but presents with as high a mortality rate as that of the left ventricle. The right ventricular regions more commonly affected by infarction are the inferior and inferior posterior aspects. The right coronary artery or left circumflex coronary artery in a left dominant system are the arteries frequently occluded in this setting[9,10]. In a right dominant system, patients with right coronary artery occlusion are at a higher risk of developing papillary muscle rupture and therefore undergoing valvular heart disease such as mitral regurgitation[11,12].

Systemic inflammatory response syndrome (SIRS) is thought to play a role in myocardial infarction associated with cardiogenic shock. Vasodilatation as part of SIRS leads to impaired perfusion of the intestinal tract, which enables transmigration of bacteria and sepsis. Tumour necrosis factor-α and interleukin 6 have myocardial depressant action and induce coronary endothelial dysfunction which may further diminish coronary flow[13].

Nitric oxide, complement, procalcitonin, neopterin and C-reactive protein also contribute to SIRS in cardiogenic shock[14]. Complement (C5) inhibition using pexelizumab in patients with myocardial infarction did not reduce the development of shock or mortality[14,15].

CLINICAL ASSESSMENT

Cardiogenic shock is characterized by inadequate tissue perfusion in the setting of adequate intravascular volume[16]. Specifically, shock in the peri-infarction setting is defined as sustained hypotension (systolic blood pressure ≤ 90mmHg for ≥ 30 minuites), accompanied by signs of peripheral hypoperfusion (altered mental status, cool peripheries, oliguria)[17]. This clinical entity is unresponsive to fluid resuscitation alone, with a cardiac index of < 2.2L/min/m^2. Subjects requiring pharmacological or mechanical circulatory support to maintain blood pressure are also involved in this category.
Some especially those with anterior myocardial infarction develop signs of end organ hyperperfusion in the setting of unsupported blood pressure measurements > 90mmHg. The urine output is low and the heart rate > 90 beats per minuite. This 'Pre Shock' presentation is associated with high risk in-hospital morbidity and mortality (43%)[18].

In the SHOCK trial registry, 64% of patients presented with hypotension, evidence of inadequate cardiac output (resting tachycardia, altered mental status, oliguria, cool peripheries) and pulmonary congestion[19]. A substantial minority (28%) presented with evidence of hypoperfusion in the absence of pulmonary congestion – the 'Silent Lung Syndrome'[19]. These latter patients have an equal distribution of anterior (50%) and non-anterior index infarction (50%) with pulmonary capillary wedge pressure in the range of 21.5 ± 6.7mmHg[19].

TREATMENT GENERAL SUPPORTIVE MEASURES

Supportive and resuscitative measures should be started immediately at the same time as the diagnostic evaluation[20]. This includes adequate oxygenation and ventilation, correction of electrolyte and acid-base abnormalities, relief of pain and restoration of sinus rhythm[20].

In patients with inadequate tissue perfusion and adequate intravascular volume, infusion of ionotropic or vasopressor drugs should start immediately[20]. Dobutamine is preferred except when there is significant hypotension(systolic blood pressure below 80mmHg); it augments coronary collateral blood flow to the ischaemic area while increasing myocardial contractility, raising cardiac output and lowering left ventricular filling pressures. It has the advantage of not affecting myocardial oxygen demand as dopamine does, however tachycardia may preclude the use of this ionotropic agent[21,22]. Dopamine is preferable when moderate hypotension and hypoperfusion are present as vasoconstriction in the peripheral vessels is often needed to maintain vital organ tissue perfusion[20]. Phosphodiesterase inhibitors, Amrinone and Milrinone can increase contractility without adrenergic stimulation leading to improved cardiac output and pulmonary pressure[23], with less effect on myocardial work[20]. These agents should be reserved for those in whom catecholamine have failed to improve cardiac performance or those in whom arrhythmia or ischemia limits the catecholamine dose because of their longer half-life especially in patients with renal impairment. The use of these agents is indicated in CS but it is important to note that a survival benefit has not been established. The routine use in patients with haemodynamically stable, decompensated heart failure was associated with greater morbidity and no clinical benefit(Outcomes of a Prospective Trial of intravenous Milirinone for Exacerbations of Chronic Heart Failure OPTIME- CHF)[24,25].Levosimendan may be used in conjunction with vasopressors to improve coronary blood flow. Levosimendan is a potent ionotrope that stabilizes troponin C and the kinetics of actin myosin cross bridges without increasing myocardial oxygen consumption of adenosine triphosphate. It is a vasodilator of the arterial, venous and coronary circulation. It should however be used with caution as it can cause hypotension[26,27].

Eventhough vasodilators may be beneficial for patients who are in shock, they should be used with extreme caution because of the risk of precipitating further hypotension and thereby reducing coronary blood flow. Intravenous nitroglycerin or sodium nitroprusside can be used but nitroglycerin is less potent as an arterial vasodilator[28].It may also have the advantage of not producing coronary 'steal' (preferential coronary blood flow to non-ischaemic vascular beds)[29]. Vasodilatorsare particularly important when mitral regurgitation is a major part of the pathophysiologic process.

Vasodilators should be withheld until the blood pressure is stabilized and haemodynamic monitoring is begun so as to ensure the beneficial effects of the drug. Patientswith cardiogenic shock from right ventricular infarction are particularly sensitive to volume depletion and prone to haemodynamic deterioration resulting from bradycardia and the loss of atrioventricular synchrony precipitated by advanced heart block. Thetreatment is thus directed towards immediate restoration of adequate left ventricular filling pressure, maintenance of sinus rhythm or synchronized pacing and the use of dobutamine to stimulate right ventricular systolic function[30-32]. Treatment basically includes maintaining right ventricular preload, reducing RV afterload, providing ionotropic support when needed, and immediate reperfusion. Cardioversion and AV synchrony for atrial fibrillation may also be required[33].

IV. REVASCULARIZATION

Early revascularization by either Percutaneous Coronary Intervention (PCI) or Coronary Artery Bypass (CABG) is recommended[34,35]. The rates of early revascularization in CS are still unsatisfactory ranging from 50 to 70% in registries despite the fact that it has markedly increased in clinical practice[1,36].

i) REVASCULARIZATION IN MULTIVESSEL CORONARY ARTERY DISEASE: About 70-80% of patients with cardiogenic shock present with multi vessel disease (coronary stenosis/occlusion in more than one vessel)[6,34]. Currently, early vascularization by PCI or CABG depending on coronary anatomy and amenability to PCI is recommended[6]. The outcome might be influenced by the type of revascularization theoretically. CABG is rarely performed in cardiogenic shock with rates <5% in registries and randomized trials[6,37]. Therefore, the accepted standard practice is PCI of the culprit lesion, while optimal management of additional non-culprit lesions is not clear[1]. Current guidelines encourage multi vessel PCI of all critical stenosis or highly unstable lesions in addition to the culprit lesions in cardiogenic shock[38]. In spite of these recommendations, multi vessel PCI is currently performed in only one-third to one-fourth of cardiogenic shock patients with multi vessel disease[6].

In the SHOCK trial, patients with cardiogenic shock were randomly allocated to early revascularisation(PCI or CABG) or medical treatment[39,40]. The result showed no significant difference in the primary end point of the 30 day mortality between the 2 groups (46.7% vs 56.0% p= 0.11). On follow up, the survival difference in form of early revascularization strategy became larger and significant at 6 months (36.9% vs 49.7%, P=0.027) and at one year (33.6% vs 46.7%), an absolute reduction of 13.2% (95% confidence interval 2.2% to 24.1%, P<0.03)[39,40]. This benefit of early revascularization was however not apparent for the elderly >75years[39]. Several studies have shown that revascularization in selected elderly patients is beneficial (20% - 30%) meaning that clinicians are capable of identifying older patients who are appropriate for revascularization[40]. Based on the American College of Cardiology (ACC)/American Heart Association guidelines (AHA) guidelines, early revascularization in cardiogenic shock for those < 75 years of age (class 1) and suitable candidates ≥ 75 years of age (class lla) is recommended[41].

The SMASH (Swiss Multicentre Angioplasty for Shock) trial compared initial strategies of coronary angioplasty with medical treatment. It showed a non-significant mortality difference (69% vs 78% relative risk 0.88, 95% CI). The higher mortality rate may have been due to the inclusion of sicker patients that remained hypotensive despite inotropic support and volume replacement. This study terminated early because of difficulties in patient recruitment[42].

ii) PERI INTERVENTIONAL ANTIPLATELET AND ANTITHROMBIN MEDICATION:

Antithrombotic therapy (antiplatelets and anticoagulation) is a cornerstone during PCI[1].

Prasugrel/ticagrel or clopidogrel is indicated in addition to aspirin in all cases undergoing PCIwhen there are contra indications for the newer oral anti platelets[38,43]. In intubated patients, crushed tablets need to be administered through a nasogastric tube. As a result of the late and impaired onset of action of anti-platelets, glycoprotein llb/llla inhibitors may be beneficial in cardiogenic shock[1.] Observational data support a potential mortality benefit by use of IV platelet inhibitors in cardiogenic shock[44]. Current considerations and experience suggest a liberal use of glycoprotein llb/llla inhibitors in patients with high thrombus burden and slow flow after PCI in particular for cardiogenic shock patients[1].

MECHANICAL SUPPORT

Mechanical circulatory support to improve haemodynamicsbecame attractive in order to overcome the limitations of ionotropes and vasopressors with limited effects to maintain adequate perfusion pressure, prevent or reverse multiorgan system dysfunction. Despite the lack of data derived from randomized clinical trials on the efficacy, safety and differential indications for different devices, percutaneous mechanical support with active devices is increasingly being performed[45].

i) INTRAAORTIC BALLOON PUMPING: Based on a national survey in United States of America (USA)[46], intraaortic balloon pumping is the most widely used device for mechanical support at stable implantation rates from 2007 to 2011 of about 50,000 per year. It improves the diastolic and lowers the endsystolic pressure without affecting the mean blood pressure. In a study, it has been shown not to improve relevant haemodynamic parameters like cardiac index or cardiac power index[46].

PERCUTANEOUS LEFT-VENTRICULAR ASSIST DEVICES: The devices are introduced percutaneously through the femoral artery and can provide a pulsatile support of 2L/min using an extracorporeal membrane pump via a 17F cannula. When the heart is in the systolic phase, blood is aspirated from the left ventricle through the catheter lumen into the membrane pump[1]. During the diastolic phase, the pump ejects the blood back through the catheter, subsequently opening the catheter valve and delivering the blood to the ascending aorta through the side outflow port, thereby creating an extra heart beat[1].the device directly unloads the ventricle

by active aspiration and simultaneously creates a counter pulsating flow in the ascending aorta[1]. Patients treated with LVADs have been noted to demonstrate higher cardiac index and mean arterial pressure but lower pulmonary capillary wedge pressure[47]. Conversely, bleeding complications and inflammation were more frequent with LVAD therapy with no difference in30 day mortality[47].

ii) EXTRACORPOREAL LIFE-SUPPORT SYSTEMS: The integral features of extracorporeal life support (ECLS) systems are the blood pump, heat exchanger and oxygenator[48]. The main drawbacks of these devices are large cannula sizes potentially causing lower limb ischemia and bleeding complications, frequent requirement of perfusionists, lack of direct left-ventricular unloading, rise in afterload and a limited support time. Complication rates may be lowered by greater experience in percutaneous implantation and by obligatory insertion of an antegrade perfusion cannula. The low cost in comparison to other percutaneous LVADs and high flow are the major advantages of this system.

Acute thoracic aortic dissection involving the ascending aorta is a life threatening cause of cardiogenic shock and requires emergency surgery (emergency aortic valve repair/replacement). The acute onset of severe aortic regurgitation is usually a medical emergency as the left ventricle is unable to adapt quickly to the sudden increase in end diastolic volume caused by the regurgitant blood. Temporary stabilization while awaiting surgery may be attempted using intravenous vasodilators such as nitroprusside, ionotropic agents like dopamine or dobutamine, to decrease left ventricular end diastolic pressure and enhance forward flow. The use of IntraAortic Balloon Counterpulsation is however contraindicated as balloon inflation in diastole will worsen the severity of aortic regurgitation[49,50].

Left Ventricular Assisted Devices are also not useful because of retrograde filling of the left ventricle across the incompetent valve, without improvement in left ventricular diastolic pressure and forward cardiac output[49,50].

Acute severe mitral regurgitation involving the posterior papillary muscle rupture occurs in cardiogenic shock from ST segment elevation myocardial infarction. Emergency mitral valve replacement rather than repair is required. This improves survival compared to medical therapy with 5 year post-operative survival rates of 60-70%[33].

Ventricular septal rupture may occur following acute myocardial infarction and may present with cardiogenic shock. Emergency surgical repair is usually required. Percutaneous trans catheter closure is usually beneficial. Devices with a diameter greater than the ventricular septal defect have been associated with relatively good outcome[2,51].

Left ventricular wall rupture is associated with a rapid progression to haemodynamic collapse, electrochemical dissociation and death. Chest pain and persistent ST wave changes usually occur. Emergency surgery should be considered for pseudoaneurysm formation with rupture and tamponade, although, mortality rates approach 60% even for those that have surgery[33].

V. CONCLUSION

The diagnosis and management of cardiogenic shock requires extensive knowledge and clinical experience. In spite of the significant advances made, the management still remains a challenge and some authors have indicated that mortality trends in cardiogenic shock have not improved significantly in recent decades. Prevention, early recognition andappropriate patient selection are key to the improvement in mortality. Newer therapeutic methods are still being awaited.

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