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Cardiogenic Shock in Acute Myocardial Infarction: A Review.

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Abstract

Cardiogenic shock is the most common cause of death in patients hospitalized with acute myocardial infarction. Cardiogenic shock is characterized by inadequate tissue perfusion due to cardiac dysfunction, and it is often caused by acute myocardial infarction. It has also been frequently associated with ST-segment elevation myocardial infarction and patients with comorbidities.

Cardiogenic shock presents with low systolic blood pressure and clinical signs of hypo perfusion. The Initialization on stability can be followed by reperfusion by fibrinolytic therapy, emergent percutaneous intervention (PCI) or coronary artery bypass grafting (CABG) is the treatment option. The PCI and CABG have been found to decrease mortality in the long term.

Research is being carried out on the role of inflammatory mediators in the clinical manifestation of cardiogenic shock. Cardiogenic shock (CS) is a complex and highly morbid entity conceptualized as a vicious cycle of injury, cardiac and systemic decompensation, and further injury and decompensation. The pathophysiology of CS is incompletely understood but limited clinical trial experience suggests that early and robust support of the failing heart to allow for restoration of systemic homoeostasis appears critical for survival. Mechanical support devices also show promise in the future.

The pathophysiology of cardiogenic shock involves a vicious spiral circle: ischemia causes myocardial dysfunction, which in turn aggravates myocardial ischemia. The review is to understand the pathophysiology of cardiogenic shock, clinical features and management of CS as a systemic process. The key to an optimal treatment of cardiogenic shock patients is a structured approach will include rapid diagnosis and prompt initiation of therapy to increase blood pressure and augment cardiac output with subsequently improved perfusion. A Rapid diagnosis and supportive therapy in the form of medications is the need of hour such as airway support and intra-aortic balloon counter pulsation is necessary. The review mainly emphasis the epidemiology, pathophysiology, and treatment management strategies for cardiogenic shock.

Keywords: Acute myocardial infarction, cardiogenic shock, CVD

Introduction

Cardiogenic shock is the most common cause of death in patients with acute myocardial infarction (AMI) and has a frequency of around 7-10% [1-4] [5]. Acute myocardial infarction is one of the most common diseases among the developing countries. These diseases have caused mortality in developed countries more than other diseases and impose numerous social and economic costs. This heart disease has emerged as a major health problem in developing countries including India. It continues to cause significant mortality despite advances in pharmacological, mechanical and reperfusion activities. Cardiogenic shock is defined as a systolic blood pressure of less than 90 mmHg for at least 30 minutes, which is secondary to myocardial dysfunction. The Cardiogenic shock is associated with clinical signs of hypoperfusion, which include decreased urine output, altered mental status and peripheral vasoconstriction. It is usually unresponsive to fluids, an important differentiating quality from other types of shock. However, it frequently responds to inotropes. The cardiac index (CI) and the pulmonary capillary wedge pressure (PCWP) are usually less than 2.2 l/min/m^2 and greater than 15 mmHg respectively [6].

Precious life is snatched away when person is in most productive stage of life, when the social and family responsibilities are the peak and greatest. These diseases are now seen in countries with low or average income which also have the majority of population. These diseases will probably turn into the most common cause of death in world till 2020 and beyond [2].

The incidence of cardiogenic shock was likewise 7.2%, and consistent with other studies [7]. However, it is difficult to assess the actual number, as a certain proportion of patients die before reaching the hospital and cannot be categorized as having cardiogenic shock [8]. Over the years, there has been little decrease in the time to present to the hospital [9]. A larger number of patients develop shock after reaching the hospital. This highlights an important fact that medical contact may have been established before shock development and opens the door to its possible prevention. In the GUSTO trial, 11% of patients had shock on presentation while 89% of patients subsequently developed shock. Similarly in case of emergency revascularise occluded Coronaries for cardiogenic shock? (SHOCK) trial registry, more than half the patients developed shock within a day of presenting to the hospital [10]. Early shock is treated as occurring in <24 hours, was found in 74.1% of patients in a recent study [11].

In addition, we observed that there is a slight increase in the number of deaths among patients who present with early shock [12]. Despite emerging innovative treatments, in-hospital mortality in patients with cardiogenic shock continues to be as high as 70-80%. A study carried out at a tertiary care hospital in other countries had an in-hospital mortality rate of 55% [13]. In a study, the overall in-hospital mortality was high (63%) but was found to reduce (P=0.004) over time. This was partially attributed to the greater use of revascularization procedures, which are known to improvise the outcomes [14]. Cardiogenic shock seems to occur with a greater frequency amongst patients with ST-segment elevation myocardial infarction (STEMI). It was observed that shock developed in 7.5% of patients with STEMI and in 2.5% of patients with non-ST-segment elevation myocardial infarction (NSTEMI) [15]. A significant delay precedes shock development in patients with NSTEMI [16]. The underlying reason may be the rapid cell necrosis that takes place in STEMI contrasting

with a slower cell loss in NSTEMI. Thus, the highest creatine kinase (CK) level is found in STEMI as compared to NSTEMI (Table-1)

Table 1. The list of different causes of Cardiogenic Shock observed generally.

S.No	Causes
1.	Acute Myocardial Infarction
2.	Left ventricular dysfunction
3.	Acute mitral regurgitation
4.	Ventricular septal rupture
5.	Right ventricular shock C
6.	Ardiac Tamponade Cardiac Rupture

In our country the management of the cardiogenic shock will vary depending on the available team of medical experts and mainly the CATH lab facilities. A well trained qualified personals and lab facilities are available, this strategy comprises of a very quick diagnostic workup with hemodynamic stabilization with inotropes and IABP along with all supportive measures and immediate transfer to a cath lab for early angiography and angioplasty with GpIIb IIIa antagonists and stenting. Where as in centers without cath lab facilities the best option would be inotropic hemodynamic stabilization (+ Early use of IABP if available) with thrombolysis without delay and transfer to a center with angio lab facilities as soon as possible.

The incidence of myocardial infarction in the world varies greatly. According to Global Burden of Diseases (GBD) study the estimated mortality from CHD in India at 1.6 million in the year 2000. It has been predicted that by 2020 there would be a 111 percent increase in cardiovascular deaths in India. This increase is much more than 77 percent for China, 106 percent for other Asian countries and 15 percent for economically developed countries [17].

In the developed countries like USA and UK, nearly 6.5 and 1.8 lakh patients get an acute myocardial infarction every year, respectively [18]. Worldwide, more than 3 million people have STEMIs and 4 million have NSTEMIs [19]. Indians are four time more prone to AMI as compared to the people of other countries due to a combination of the genetic and lifestyle factors that promote metabolic dysfunction [20]. The incidence of myocardial infarction in India is 0.064% [21]. The mortality rate of myocardial infarction is approximately 30% and for every 1 in 25 patients who survive the initial hospitalization, dies in the first year after AMI. In India.

Some disease factors contribute to the risk of myocardial infarction and they include diabetes mellitus (type 1 or 2), high blood pressure, dyslipidemia/hypercholesterolemia and particularly high amount of low-density lipoprotein, low amount of high density lipoprotein, high triglycerides, and obesity [22]. Atherosclerosis is by far the most common cause of myocardial infarction. According to the INTERHEART study report, nine factors are responsible for 90% of myocardial infarctions. Modifiable risk factors include Diabetes mellitus, smoking, hypertension, hyperlipidemia, sedentary life style, obesity, stress and depression. The combination of several risk factors further enhances the risk.

ST Elevation Myocardial Infarction (STEMI) is one of the most challenging clinical conditions in cardiology but also the most gratifying when treated promptly and appropriately. Recent advances in the management of STEMI have shown the

prompt treatment can save lives and restore normal cardiac function. However, for treatment to be successful time is crucial, and for this teamwork is critical. Delay or sub optimal management at any stage can affect outcomes.

The National Commission on Macro-economics and Health has projected the evolving epidemic of coronary artery disease in India - 80 million patients with CHD by the year 2030 with a yearly mortality close to 5 million. A substantial number of this will be due to acute STEMI or the delayed consequences of STEMI. Though the US and Europe have substantantially fewer STEMI, the national bodies have formulated appropriate guidelines and there are audits and attempts to encourage and monitor adherence. Unfortunately there is less or no focused training program for STEMI and there is no national guideline or policy in our country.

Epidemiology of Cardiogenic Shock:

Cardiogenic Shock complicates 5% to 10% of cases of acute myocardial infraction and is the leading cause of death after MI. (23) ST-segment–elevation myocardial infarction (STEMI) is associated with a 2-fold increased risk for development of Cardiogenic Shock compared with non–ST-segment–elevation myocardial infarction (NSTEMI). Patients with NSTEMI-associated Cardiogenic Shock are less likely to undergo early cardiac catheterization, delaying PCI and coronary artery bypass graft and increasing the risk of mortality compared with patients with STEMI-associated cardiogenic shock.(24) Higher incidences of Cardiogenic Shock are observed in women, Asian/Pacific Islanders, and patients aged >75 years. The incidence of Cardiogenic Shock has increased in recent years, while the reason for increasing incidence is unclear, improved diagnosis and better access to care are both likely contributory. While the in-hospital mortality has improved, the 6- to 12-month mortality in cardiogenic shock has remained unchanged at ≈50% over the past 2 decades. (25)

Survivors of MI-associated Cardiogenic shock have an 18.6% risk of 30-day readmission after discharge, with a median time of 10 days. The risk of readmission is slightly lower among patients with STEMI versus NSTEMI. The most common causes of readmission are congestive heart failure and new myocardial infarction. Female sex, low socioeconomic status, mechanical circulatory support (MCS) device placement, atrial fibrillation, and ventricular tachycardia are predictors of readmission.(26)

Pathophysiology of Cardiogenic shock

Our understanding of the complexity and pathophysiology of MI-associated cardiogenic shock in particular has evolved over the past two decades. (27) In general, there is a profound depression of myocardial contractility resulting in a potentially deleterious spiral of reduced cardiac output, low blood pressure, and further coronary ischemia, followed by additional reductions in contractility This cycle may lead to death. This classic paradigm also includes compensatory, although pathological, systemic vasoconstriction that results from acute cardiac injury and ineffective stroke volume. Emerging evidence has also shown that impairment of tissue microcirculation is associated with 30-day mortality and temporal changes in SOFA (Sepsis Related Organ Failure Assessment) scores and may be improved with MCS.(28) In fact, it is now well established that CS can result in both acute and subacute derangements to the entire circulatory system, including the peripheral vasculature. Extremity and vital organ hypoperfusion remains a clinical hallmark. Although ineffective stroke volume is the inciting event, inadequate circulatory compensation may also contribute to shock.

Peripheral vasoconstriction may improve coronary and peripheral perfusion at the cost of increased afterload (Figure-.1, Table-2))

Alternatively, systemic inflammation triggered by acute cardiac injury may induce pathological vasodilatation. Endothelial and inducible nitric oxide (NO) synthase may play a major role in the production of high NO levels, along with peroxynitrite, which has a negative inotropic effect and is cardiotoxic.(29). Other inflammatory mediators such as interleukins and tumor necrosis factor can also contribute to systemic vasodilation and have been associated with mortality in CS.(30))In addition, bleeding and transfusions may be associated with mortality.(31) Alterations in erythrocyte NO biology of stored blood can lead to vasoconstriction, platelet aggregation, and ineffective oxygen delivery, whereas transfusion of stored blood may also contribute to inflammation.

Shock is a clinical state in which target tissue perfusion is inadequate to supply vital substrates and remove metabolic waste. Inadequate cellular oxygenation leads to marked generalized impairment of cellular function and multiorgan failure. Autopsy studies indicate that combined new and old extensive myocardial infarctions (MI) consistently involve more than 40% of the left ventricular (LV) myocardium. The underlying pathophysiology of cardiogenic shock is:

1. Profound depression of myocardial contractility, resulting in a vicious spiral of reduced cardiac output and low blood pressure.
2. Further coronary insufficiency, and further reduction in contractility and cardiac output occurs.
3. In response to the severe reduction of cardiac output (CO) compensatory systemic vasoconstriction with increased systemic vascular resistance occurs.
4. The dogma that acute reduction in CO causes marked compensatory vasoconstriction was not confirmed, however, in many patients in the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) registry and trial. Over time, myocardial hyper contractility ceases because of utilization of glucose over fatty acids, loss of Krebs cycle intermediates, and depletion of substrate required for adenosine triphosphate (ATP) production.

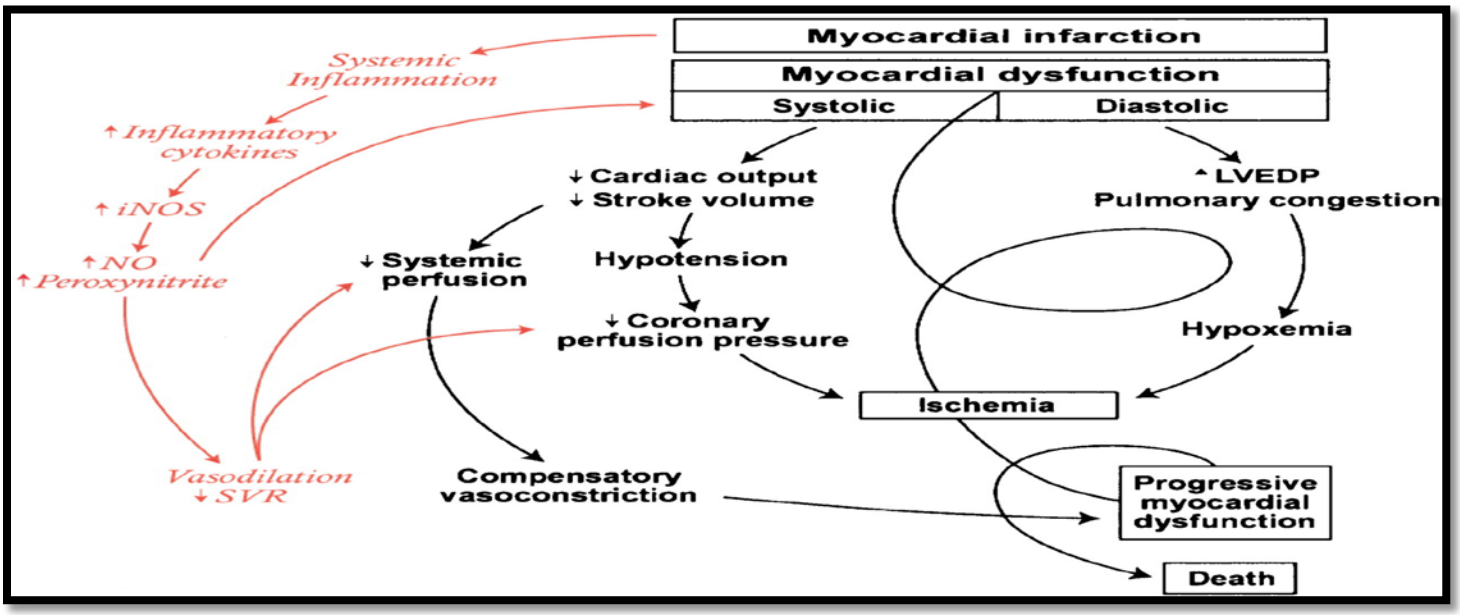

Figure 1: Pathophisiology shock spiral of cardiogenic shock

Table-2: The various causes of Cardiogenic Shock

S.No.	Causes
1.	Myocardial disorders
2.	Acute myocardial infarction and complications
3.	a Dilated and hypertrophic cardiomyopathy
4.	Acute aortic regurgitation
5.	Prosthetic valve dysfunction
6.	Valvular
7.	Acute mitral regurgitation
8.	Severe aortic stenosis
9.	Preload reduction Restriction to filling Cardiac tamponade
10.	Mitral stenosis,
11.	left atrial myxoma, or thrombus Alteration of compliance
12.	Acute myocardial infarction, especially in the presence of right ventricular infarction Hypertrophic cardiomyopathy
13.	Decrease diastolic filling with tachyarrhythmias Tachyarrhythmias, bradyarrhythmias
14.	Other cardiovascular causes of shock Aortic dissection Pulmonary embolism Primary pulmonary hypertension.

Management of Cardiogenic Schock

Assessment Cardiogenic shock is an emergency and it needs rapid diagnosis and institution of therapy (Figure-2). Improved long-term outcomes require immediate diagnosis and management and if needed, transfer to a tertiary care hospital. History will usually reveal symptoms of a preceding AMI. A diagnosis of cardiogenic shock is made when myocardial dysfunction is observed in the absence of other causes such as hemorrhage, sepsis, pulmonary embolism, tamponade, aortic dissection, and preexisting valvular disease [31]. On a physical exam, the patient may be cyanotic with cold extremities and pulses are usually rapid and faint. If LVD is the etiology, then jugular venous distention (JVD) and rales in the lung field due to pulmonary congestion are observed. If right ventricular failure is the underlying cause then JVD and kussmaul's sign is present and pulmonary rales are not found. Other findings include distant heart sounds and the presence of third and fourth heart sounds. Mitral regurgitation or a VSD can lead to a new systolic murmur. Arrhythmias are a frequent occurrence and need immediate attention.

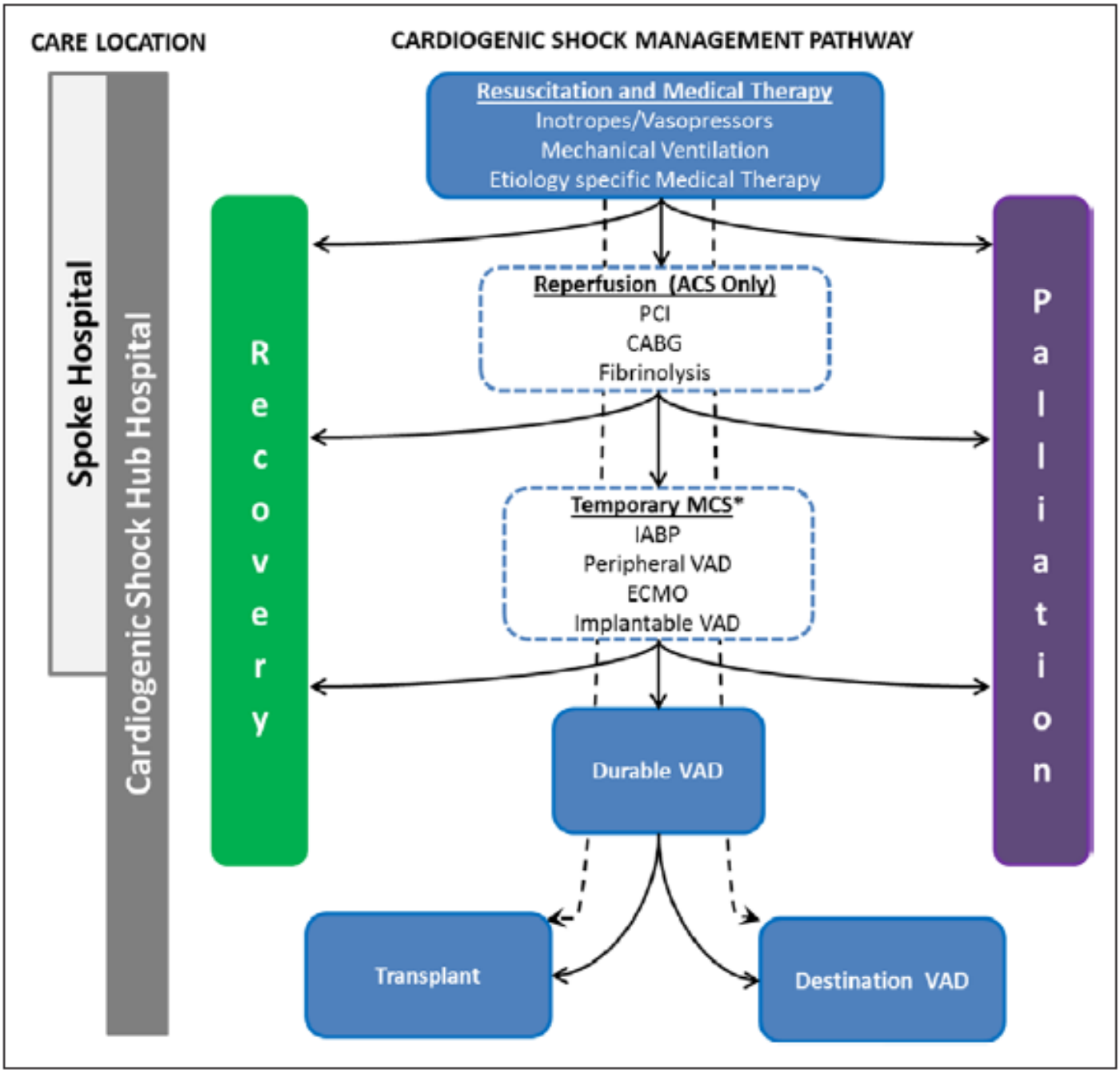

Figure 2 : Managemental Pathway of Cardiogenic Shock at care location and care providers.

ACS: Acute Coronary Syndrome, CABG:Coronary artery bypass graft, ECMO: Extracorpeal Membrane oxygenation, IABP:Intra-aortic balloon pump MCS:Mechanical Circulatory Support PCI:Percutaneous coronary invention VAD: ventricular assist device

Diagnosis of cardiogenic shock

Cardiogenic shock is usually diagnosed in an emergency setting. Doctors will check for signs and symptoms of shock, and will then perform tests to find the cause (Figure-3). Tests might include:

- **Blood pressure measurement.** People in shock have very low blood pressure.

- **Electrocardiogram (ECG).** This test records the electrical activity of your heart via electrodes attached to your skin. If you have damaged heart muscle, electric problems or fluid buildup around your heart, it won't conduct electrical impulses normally.
- **Chest X-ray.** This allows your doctor to check the size and shape of your heart and its blood vessels and whether there's fluid in your lungs.
- **Blood tests.** You'll have blood drawn to check for organ damage, infection and heart attack. Another type of blood test called arterial blood gas might be used to measure oxygen in your blood.
- **Echocardiogram.** Sound waves produce an image of your heart that can help identify damage from a heart attack.
- **Cardiac catheterization (angiogram).** A liquid dye is injected into the arteries of your heart through a long, thin tube (catheter) that's inserted through an artery, usually in your leg. The dye makes your arteries visible on X-ray, revealing areas of blockage or narrowing.

Cardiogenic shock treatment focuses on minimizing the damage from lack of oxygen to your heart muscle and other organs.

Emergency life support

Most people who have cardiogenic shock need extra oxygen. If necessary, you'll be connected to a breathing machine (ventilator). You'll receive medications and fluid through an intravenous (IV) line in your arm.

Medications

Fluids and plasma, given through an IV, and medications to treat cardiogenic shock, work to increase your heart's pumping ability.

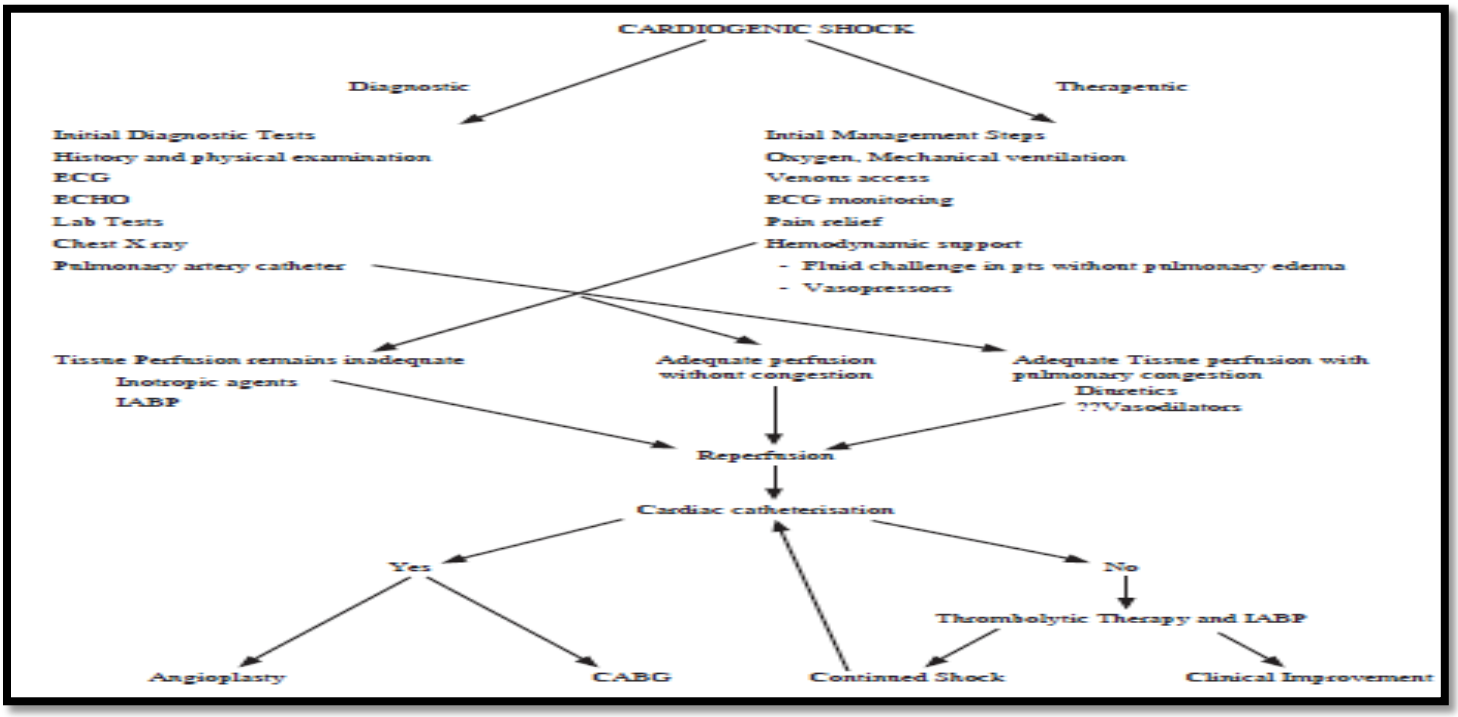

Figure-3: A possible way and strategic approach for the diagnosis and treatment of cardiogenic shock.

- **Inotropic agents.** You might be given medications to improve your heart function, such as norepinephrine (Levophed) or dopamine, until other treatments start to work.

- **Aspirin.** Emergency medical workers might give you aspirin immediately to reduce blood clotting and keep your blood flowing through a narrowed artery. Take an aspirin yourself while waiting for help to arrive only if your doctor has previously told you to do so for symptoms of a heart attack.
- **Thrombolytics.** These drugs, also called clot busters or fibrinolytics, help dissolve a blood clot that's blocking blood flow to your heart. The sooner you receive a thrombolytic drug after a heart attack, the greater your chances of survival. You'll likely receive thrombolytics, such as alteplase (Activase) or reteplase (Retavase), only if emergency cardiac catheterization isn't available.
- **Antiplatelet medication.** Emergency room doctors might give you drugs similar to aspirin to help prevent new clots from forming. These include medications, such as oral clopidogrel (Plavix), and platelet glycoprotein IIb/IIIa receptor blockers, such as abciximab (Reopro), tirofiban (Aggrastat) and eptifibatide (Integrilin), which are given through a vein (intravenously).
- **Other blood-thinning medications.** You'll likely be given other medications, such as heparin, to make your blood less likely to form clots. IV or injectable heparin usually is given during the first few days after a heart attack.

Medical procedures

Medical procedures to treat cardiogenic shock usually focus on restoring blood flow through your heart. They include:

- **Angioplasty and stenting.** If a blockage is found during a cardiac catheterization, your doctor can insert a long, thin tube (catheter) equipped with a special balloon through an artery, usually in your leg, to a blocked artery in your heart. Once in position, the balloon is briefly inflated to open the blockage.

  A metal mesh stent might be inserted into the artery to keep it open over time. In most cases, you doctor will place a stent coated with a slow-releasing medication to help keep your artery open.
- **Balloon pump.** Your doctor inserts a balloon pump in the main artery off of your heart (aorta). The pump inflates and deflates within the aorta, helping blood flow and taking some of the workload off your heart.
- **Mechanical circulatory support.** Methods newer than the balloon pump are being used to help improve blood flow and supply oxygen to the body, such as extracorporeal membrane oxygenation (ECMO).

Surgery

If medications and medical procedures don't work to treat cardiogenic shock, your doctor might recommend surgery.

- **Coronary artery bypass surgery.** This involves sewing veins or arteries in place at a site beyond a blocked coronary artery. Your doctor might suggest this procedure after your heart has had time to recover from your heart attack. Occasionally, bypass surgery is performed on an emergency basis.
- **Surgery to repair an injury to your heart.** Sometimes an injury, such as a tear in one of your heart's chambers or a damaged heart valve, can cause cardiogenic shock. Surgery might correct the problem.
- **Ventricular assist device.** A mechanical device can be implanted into the abdomen and attached to the heart to help it pump. This might extend and improve the lives of some people with end-stage heart failure who are waiting for new hearts or aren't able to have heart transplantation.
- **Heart transplant.** If your heart is so damaged that no other treatments work, a heart transplant may be a last resort.

Suggestions For Clinical Practice

It is suggested that all patients with CS be evaluated with an ECG, chest x-ray, and comprehensive echocardiogram with the specific purpose of understanding the dominant mechanism responsible for acute hemodynamic instability. In the absence of contraindications, additional imaging with a computed tomography scan or transesophageal echocardiogram (as appropriate) if an acute aortic syndrome or pulmonary embolism is suspected is appropriate. Suggested laboratory tests include a complete blood count, electrolytes, creatinine, hepatic function tests, arterial blood gas and lactate, and serial cardiac troponin levels.

Conclusion

The pathophisiology and management of CS is fundamentally a systemic and derangement of maladaptive compensatory mechanisms resulting in perpetuation of coronary and systemic perfusion mismatch. Cardiogenic shock due to AMI continues to be the main cause of death in these patients. Immediate diagnosis and management is required. There are two strategies in treating cardiogenic shock: medical versus invasive. If institutions lack revascularization facilities, fibrinolytic therapy and IABP should be used while provisions are made for invasive treatment. However, current guidelines favor an invasive approach. .Although the optimal treatment approach is incompletely defined, especially in non-ACS presentations, clinical trials are difficult to conduct in this patient population. While percutaneous MCS device trials to date have failed to show survival benefit in small randomised trials, haemodynamics improve with use of these devices. Current understanding of the pathophysiological process of CS suggests that interrupting the ‘shock spiral’ and restoration of cardiovascular homoeostasis early before end-organ hypoperfusion occurs may be ultimately critical to survival. To ensure appropriate use of these devices and uniformity in CS management, a systematic and consistent approach using contemporary haemodynamic parameters to guide management should be considered. In addition, risk/safety as well as cost must be factored into the institutional approach to CS before tailoring to the specific needs of the patient.

There are various risk factors of acute myocardial infarction which should be taken into consideration while treating patients of AMI. Expression of coronary heart disease phenotype is feature of ageing and therefore increased life expectancy has resulted in the greater exposure to the risk factors of coronary heart disease. As a result of population growth and demographic trends, urban population of India is expected to double to 600 million by 2020, further expanding the coronary heart disease epidemic.13 In the present study except family history all other risk factors are modifiable, illustrating the enormous potential for prevention of acute myocardial infarction. Prognosis is established by the outcome of revascularization regardless of the procedure used, such as PCI or surgery. Newer devices are being developed for mechanical support. Inhibitors of nitric oxide have also shown favorable outcomes. These newer therapies may help in decreasing the significant mortality of cardiogenic shock in the future.

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