

CARDIOGENIC SHOCK ETIOLOGY AND PRINCIPLES OF HOW TO HELP THE PATIENT IN THIS CASE

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Annotation: This article provides information on how cardiogenic shock is caused , how to help the patient in this case. The article is written in the case of using internet resources.

Key words: cardiogen shock, blood, heart.

Cardiogenic shock is defined as a primary cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion. Clinical criteria include a systolic blood pressure of less than or equal to 90 mm Hg for greater than or equal to 30 minutes or support to maintain systolic blood pressure less than or equal to 90 mm Hg and urine output less than or equal to 30 mL/hr or cool extremities. Hemodynamic criteria include a depressed cardiac index (less than or equal to 2.2 liters per minute per square meter of body surface area) and an elevated pulmonary-capillary wedge pressure greater than 15 mm Hg.

Cardiogenic shock is a clinical entity characterized by a low cardiac output state of circulatory failure that results in end-organ hypoperfusion and tissue hypoxia. The most common cause of cardiogenic shock is acute myocardial infarction, though other disorders leading to impairment of the myocardium, valves, conduction system, or pericardium also can result in cardiogenic shock. Despite advances in reperfusion therapy and mechanical circulatory support treatments, morbidity, and mortality among patients with cardiogenic shock remain high.

Various forms of cardiac dysfunction can cause cardiogenic shock.

The most common causes of cardiogenic shock include:

- Acute myocardial ischemia
- Mechanical defect: acute mitral regurgitation (papillary muscle rupture), ventricular wall rupture (septal or free wall), cardiac tamponade, left ventricular outflow obstruction (hypertrophic obstructive cardiomyopathy [HOCM], aortic stenosis [AS]), Left ventricular inflow obstruction (MS, atrial myxoma)
- Contractility defect: ischemic and non-ischemic cardiomyopathy, arrhythmias, septic shock with myocardial depression, myocarditis
- Pulmonary embolus (right ventricular with or without left ventricular failure)
- Right ventricular failure
- Aortic dissection

- Other causes include cardiotoxic drugs (doxorubicin), medication overdose (beta/calcium channel blockers), metabolic derangements (acidosis), electrolyte abnormalities (calcium or phosphate)

Risk of Cardiogenic shock after ST-elevation myocardial infarction (STEMI):

- Age more than 70 years
- Systolic blood pressure less than 120 mmHg
- Sinus tachycardia or bradycardia
- A long duration of symptoms before treatment

The pathophysiology of cardiogenic shock is complex and not fully understood. Ischemia to the myocardium causes derangement to both systolic and diastolic left ventricular function, resulting in a profound depression of myocardial contractility. This, in turn, leads to a potentially catastrophic and vicious spiral of reduced cardiac output and low blood pressure, perpetuating further coronary ischemia and impairment of contractility. Several physiologic compensatory processes ensue. These include:

- The activation of the sympathetic system leading to peripheral vasoconstriction may improve coronary perfusion at the cost of increased afterload, and
- Tachycardia increases myocardial oxygen demand and subsequently worsens myocardial ischemia.

These compensatory mechanisms are subsequently counteracted by pathologic vasodilation that occurs from the release of potent systemic inflammatory markers such as interleukin-1, tumor necrosis factor- α , and interleukin-6. Additionally, higher levels of nitric oxide and peroxynitrite are released, which also contribute to pathologic vasodilation and are known to be cardiotoxic. Unless interrupted by adequate treatment measures, this self-perpetuating cycle leads to global hypoperfusion and the inability to effectively meet the metabolic demands of the tissues, progressing to multiorgan failure and eventually death.

The presenting symptoms of cardiogenic shock are variable. The most common clinical manifestations of shock, such as hypotension, altered mental status, oliguria, and cold, clammy skin, can be seen in patients with cardiogenic shock.

History plays a very important role in understanding the etiology of the shock and thus helps in the management of cardiogenic shock.

The patient should also be assessed for cardiac risk factors:

- Diabetes mellitus
- Tobacco smoking
- Hypertension

- Hyperlipidemia
- A family history of premature coronary artery disease
- Age older than 45 in men and older than 55 in women
- Physical inactivity

Physical examination findings in patients with cardiogenic shock include the following:

- Altered mental status, cyanosis, cold and clammy skin, mottled extremities
- Peripheral pulses are faint, rapid, and sometimes irregular if there is an underlying arrhythmia.
- Jugular venous distension
- Diminished heart sounds, S3 or S4, may be present, murmurs in the presence of valvular disorders such as mitral regurgitation or aortic stenosis.
- Pulmonary vascular congestion may be associated with rales.
- Peripheral edema may be present in the setting of fluid overload

Cardiogenic shock is an emergency requiring immediate resuscitative therapy before the irreversible damage of vital organs. Rapid diagnosis with prompt initiation of pharmacological therapy to maintain blood pressure and to maintain respiratory support along with a reversal of underlying cause plays a vital role in the prognosis of patients with cardiogenic shock.

Early restoration of coronary blood is the most important intervention and is the standard therapy for patients with cardiogenic shock due to myocardial infarction.

The management of cardiogenic shock involves the following:

Medical Management

The goal of medical management is to restore cardiac output and prevent irreversible end-organ damage rapidly.

- The optimal choice of a vasoactive agent in cardiogenic shock is unclear.
- Norepinephrine is preferred over dopamine in patients with severe hypotension (systolic blood pressure less than 70 mm Hg) or hypotension unresponsive to other medications as dopamine has been associated with higher rates of arrhythmias and a higher risk of mortality in this patient population. However, norepinephrine should be used with caution as it can cause tachycardia and increased myocardial oxygen demand in patients with recent myocardial infarctions.
- Dobutamine is widely used, has beta-1 and beta-2 agonist properties, which can improve myocardial contractility, lower left ventricular end-diastolic pressure, and increased

cardiac output.

- Milrinone, also a widely used inotrope, has been shown to reduce left ventricular filling pressures.
- Saline or lactated ringer solution greater than 200 ml per 15 to 30 minutes is indicated in patients with no signs of fluid overload.
- Fibrinolytic therapy should be administered to patients who are unsuitable candidates for either percutaneous coronary intervention or coronary artery bypass graft if there are no contraindications.
- Patients with myocardial infarction or acute coronary syndrome are given aspirin and heparin. They have been shown to be effective in reducing mortality.
- Diuretics such as furosemide play a role in decreasing plasma volume and edema and thereby decreasing cardiac output and blood pressure. This is associated with a compensatory increase in peripheral vascular resistance. With continuous therapy, extracellular fluid and plasma volumes return almost to pretreatment levels.
- Therapeutic hypothermia is established for out-of-hospital cardiac arrest patients with shockable rhythm to prevent brain injury and improve survival.

Procedures

- Central line placement plays a role in fluid resuscitation, access for multiple infusions, and allows invasive monitoring of central venous pressure.
- Arterial line placement is useful in providing continuous blood pressure monitoring, especially in patients requiring inotropic agents.
- Mechanical ventilation is indicated in patients with cardiogenic shock for oxygenation and airway protection.

Percutaneous Coronary Intervention and Coronary Artery Bypass

- Primary percutaneous coronary intervention (PCI) should be performed, irrespective of time delay from the onset of myocardial infarction
- Urgent coronary artery bypass grafting is indicated in patients with coronary anatomy not amenable to PCI.

SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial data confirmed an approach that combines early revascularization with medical management in patients with cardiogenic shock is optimal.

Mechanical Circulatory Support

Due to the poor prognosis associated with cardiogenic shock, medical therapy is often inadequate, and mechanical circulatory support (MCS) therapy to improve end-organ perfusion may be required. An experienced interprofessional team should evaluate MCS.

- Percutaneous circulatory assist devices provide superior hemodynamic support compared with pharmacologic therapy.
- Intra-aortic balloon pumps can be considered but are less likely to provide a benefit compared to other MCS devices and should not be routinely used but may still play an important role in patients with acute severe mitral regurgitation, ventricular septal defects, or when other MCS devices cannot be placed.
- Extracorporeal membrane oxygenation (ECMO) is indicated in patients with poor oxygenation not expected to improve with alternative temporary mechanical support devices rapidly.
- In properly selected patients not likely to recover from cardiogenic shock without long-term MCS support, a ventricular assist device can be implanted as a bridge to recovery, bridge to bridge, bridge to transplant, or destination therapy.
- Cardiac transplantation may be performed in suitable candidates not expected to recover after MCS device implantation may be the only hope for meaningful long-term recovery. However, it remains a very limited option owing to the low number of available hearts.

Short-term mechanical circulatory assist

- IABP (Intra-aortic balloon pump)
- Non-IABP percutaneous mechanical devices
- ECMO

IABP- Intra-aortic balloon pump

- Most commonly used and least expensive of all mechanical support devices.
- Easy and rapid insertion.
- It is the device with which interventional cardiologists are most familiar.

Some of the other indications apart from cardiogenic shock include intractable angina, adjunctive therapy in high risk or complicated angioplasty, refractory heart failure as a bridge to future therapy, intractable ventricular arrhythmias as a bridge to therapy.

Mechanism: Blood is displaced into the proximal aorta by inflation during diastole, during systolic, rapid balloon deflation occurs, creating a vacuum effect and reducing the afterload.

Contraindications include severe aortic regurgitation, aortic dissection, uncontrolled sepsis, uncontrolled bleeding disorders.

The patient needs to be on therapeutic anticoagulation to avoid thrombosis, and heparin is the most common anticoagulation used. Daily labs, including creatinine, platelet count, and hemoglobin, need to be checked. Uncommon events like cholesterol embolization and balloon rupture are rare occurrences.

Limitations of IABP include variable effect on total coronary blood flow, no mortality benefit, modest hemodynamic support.

Non-IABP percutaneous mechanical devices

Left ventricular assist devices

1. Left ventricle to aorta- percutaneous transvalvular left ventricular assistance device (LVAD)
2. Left-atrium-to-aorta assist device-
3. Percutaneous cardiopulmonary bypass support with the use of an extracorporeal membrane oxygenator (ECMO).

Right ventricular assist device

-The LVAD system is available in 2 different versions (2.5 and 5.0). The larger version needs surgical implantation, whereas the smaller motion could be placed percutaneously.-This has an axial flow pump that revolves at a high speed displacing blood from the left ventricle to the proximal ascending aorta and works on the principle of Archimedes screw.

-Protective to trial compared to a 30-day incidence of major adverse events between intractable on the pump and the 2.5 LVAD. It showed no difference between the 2 groups; trends for improved outcomes were observed for the 2.5 supported patients at 90 days.

-Placement of the LVAD system takes a longer time compared to IABP and needs more experience. However, it provides a better improvement in cardiac index.

Left-atrium-to-aorta assist device

With a percutaneous left atrium to aortic assistive device, blood is pumped from the left atrium to the iliofemoral system. It is approved by the US FDA for 6 hours of support. A randomized control trial comparing this device with an IABP in 41 patients with cardiogenic shock after acute myocardial infarction. Hemodynamic and metabolic parameters were more effectively reversed with the left atrium to aorta assist device. Complications such as severe bleeding and acute limb ischemia are more common with this device, and there is no difference in 30-day mortality (VAT 43% versus balloon pump 45%).

ECMO-venoarterial bypass configuration

Pulmonary circulation is bypassed, and oxygenated blood returns to the patient via an arterial or venous route. The extracorporeal pump is employed to support systemic perfusion. Cannulas are placed into the central vein and central artery. Blood from the venous catheter is pumped through a heat exchanger and oxygenator. Blood is returned to the systemic arterial circulation via

arterial cannula. This is used in patients with circulatory and respiratory failure.

Palliative Care in Cardiogenic shock

- Early referral to a palliative care specialist is recommended as a strategy to reduce physical and emotional distress, optimize symptom control, and improve quality of life.

Current Management Guidelines

1. Immediate transfer for PCI
2. Consider open heart surgery if PCI not available
3. Start fibrinolytic therapy if PCI and open surgery not available
4. Do not start beta-blockers
5. May use an IABP to stabilize the patient
6. Consider LV assist device if no contraindications

Cardiogenic shock is a life-threatening disorder and is the main cause of death after an acute MI. Even in the best of hands and the latest treatment, the condition carries a mortality rate in excess of 30%. The key to survival is to have prompt resuscitation with coronary artery revascularization. Unfortunately, even with revascularization, multiorgan failure is common, and long-term survival is not guaranteed. Because cardiogenic shock affects almost every other organ in the body, the condition is best managed by an interprofessional team that also includes ICU nurses.

Once cardiogenic shock has been diagnosed, patient monitoring is vital. The surgeon and cardiologist need to be notified right away. The cardiac catheterization nurses need to be informed of this emergency as the initial treatment of choice is PCI. The pharmacist should make sure that the patient is on no medications that depress the heart function like beta-blockers. If an IABP is contemplated, the perfusionist should be notified. Most patients require mechanical ventilation, and hence, respiratory therapists should be involved to ensure that positive pressure ventilation is avoided.

If the patient undergoes PCI or open-heart surgery, monitoring is required as complications are common in the post-operative period. The nephrologist, pulmonologist, and internist should be involved to ensure optimal care. The nurse should monitor the patient for oliguria, coagulopathy, poor oxygenation, loss of pulses, abdominal pain (mesenteric ischemia), and stroke. Close communication is vital between the interprofessional team to improve outcomes.

Even though cardiogenic shock cannot be entirely prevented, clinicians should educate patients on reducing risk factors for heart disease. Patients should be urged not to smoke, lower their lipids, and ensure better control of blood sugars. In addition, enrollment in an exercise program can help lower body weight and help achieve better control of blood pressure.

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