

Right ventricular and left ventricular cardiogenic shock



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Cardiogenic shock is a major and often fatal complication of a variety of acute and chronic disorders whereby the heart muscle fails to effectively pump blood forward and is unable to maintain adequate tissue perfusion. This ongoing clinical problem of cardiogenic shock often results from cardiac failure. Nurses and physicians need to work together to develop a rapid and well-organized treatment approach to this devastating condition. Acute myocardial infarction (AMI) is the most common cause and early recognition of cardiogenic shock is essential to saving the patient and functional organ perfusion. To help discuss the differences between right ventricular and left ventricular cardiogenic shock as a result of AMI, a case study format has been chosen. Priority nursing diagnoses, interventions and outcomes will also be addressed.

Case Study

Mrs. Rudd, a 53-year-old woman, is walking her two golden retrievers in Central Park when she starts to develop a nagging left arm pain radiating up her neck and down to her fingers along with diaphoresis, flushing and shortness of breath. She loses grip of the leashes and the dogs, sensing something wrong, get the attention of a young couple sitting under a tree. They see her in distress and call 911 to get an ambulance. Paramedics arrive on the scene within minutes and transport her to the emergency department at Mt. Sinai Hospital.

On arrival, Mrs. Rudd continues to complain of the pain getting more intense and a new onset of substernal pain. The paramedics note her to be pale and clammy with cool and mottled extremities. Her vital signs in the ED are heart

rate 56 beats/minute, blood pressure 78/53, respiratory rate 24 breaths/min and labored, pain level of 9/10 and temperature 96.9° F orally. The nurse provides supplemental oxygen at 100% via non-rebreather mask and administers 325mg of chewable aspirin, but holds the nitroglycerin because of her already low HR and BP. Then the cardiac monitor leads are placed, which shows sinus bradycardia; 18g IV access is obtained in both arms and blood chemistry, CBC and cardiac enzymes are drawn and sent to the lab. A stat 12-lead electrocardiogram is run which indicates that Mrs. Rudd is having ST elevations in leads II, III and aVF suggesting acute inferior wall myocardial infarction. This explains the bradycardia due to damage to the right ventricle and likely an occluded right coronary artery. Tachycardia is seen in anterior and lateral wall MI where the circumflex and/or LAD coronary arteries are blocked. The nurse may also see ST depression instead of elevation in leads II, III and aVF with either of these infarcts. Her signs and symptoms upon presentation to the hospital suggest that she is in the early stages of cardiogenic shock, a life-threatening complication of AMI associated with high mortality. Early and aggressive treatment is necessary for her to survive this condition (Lenneman, 2011).

Etiology and Pathophysiology

With cardiogenic shock, perfusion is affected and delivery of oxygen to the tissues is markedly decreased. Various conditions can lead to cardiogenic shock besides AMI. It can occur as a complication of open heart surgery, myocarditis, valve failure, severe dysrhythmias or from any disease or injury that leads to mechanical failure of the body's pump. With AMI, the myocardium is starved of oxygen and nutrients and dies (Lenneman, 2011). <https://assignbuster.com/right-ventricular-and-left-ventricular-cardiogenic-shock/>

This leads to diminished contractility, reduced ejection fraction (the percentage of blood present in the ventricle at end-diastole that is pumped out with each heart beat) and disruption of hemodynamic measurements including persistent hypotension, high filling pressures, such as PAWP, PAP, high CVP and SVR, and most importantly reduced stroke volume and cardiac output. As a result of the reduced ventricular emptying, pressure rises within the ventricles, causing dilation of the ventricles, eventually leading the one initially injured to fail and if not corrected both ventricles. In cases of heart failure in both ventricles, the patient will probably require transplantation and an LVAD to keep them alive while on the list awaiting a donor heart (Holcomb, 2002).

Signs and symptoms

Clinical signs and symptoms that are associated with cardiogenic shock depend on the ventricle affected. In right heart failure the nurse may observe jugular vein distension, peripheral edema and weak pulses, altered mental status, elevated ICP. Venous congestion and bradycardia are possible due to the heart slowing to allow adequate blood return and filling before pumping, which can be heard on auscultation as a split second heart beat. With left ventricular failure a pathological S3 or ventricular gallop can be auscultated, and pulmonary edema and congestion will likely be present, evidenced by labored breathing, dyspnea, coarse crackles and wheezing leading to ineffective gas exchange. To improve oxygenation, respiratory rate increases and the patient hyperventilates as manifested by hypocapnia and alkalosis (PaCO₂ less than 35 or pH greater than 7.45) measured by arterial blood gases (Holcomb, 2002). Since the heart is unable to recover <https://assignbuster.com/right-ventricular-and-left-ventricular-cardiogenic-shock/>

and maintain adequate perfusion to the kidneys, they also will fail and subsequent ABGs reveal a shift indicating a worsening condition of both respiratory and metabolic acidosis.

Sustained hypotension (systolic blood pressure less than 90mmHg for longer than 30 minutes) and adequate left ventricular filling pressure with signs and symptoms of tissue hypoperfusion are less common definitions for cardiogenic shock. This hypoperfusion may be exhibited by such signs as cool extremities, altered mental status, oliguria (urine output less than 30mL/hour or less than 0.5mL/kg/hour) or all three. Another helpful measurement utilized in assessing shock is drawing a serum lactic acid level, a diagnostic tool for detecting occult tissue hypoperfusion. Even if a patient does not exhibit low blood pressure at onset, a lactic acid value above 4mmol/L can identify organ dysfunction at the cellular level before the patient becomes hypotensive. This can be assessed by the nurse observing skin becoming cool, pale, and clammy as blood is shunted away from the periphery and skeletal muscles back to the vital organs. As a consequence, wasting and lactic acid buildup occur. The effects of blood being shunted away from the gastrointestinal tract initially lead to decreased bowel sounds and eventually progress to absent bowel sounds or paralytic ileus (Farwell, 2006).

Hemodynamics

In the case of Mrs. Rudd, her initial presentation reveals signs of the early compensatory phase of shock. The physicians and nurses need to collaborate and respond promptly to limit permanent damage to her organs and ensure

her survival. In the early stage of cardiogenic shock the sympathetic nervous system is activated to respond to a failing heart. The renin-angiotensin-aldosterone system (RAAS) is stimulated to cause vasoconstriction and sodium and water retention to maintain blood pressure (Porth, 2006).

To evaluate the effectiveness of organ perfusion cardiac output needs to be monitored and controlled. This is initially sustained with fluid replacement and volume expanders in right heart failure resulting from inferior AMI, to in essence replace oil in the engine to keep it running. In response to heart failure from a lateral or anterior AMI, the patient will exhibit increases in heart rate and/or stroke volume defined as the amount of blood pumped out with each ventricular contraction or the difference between the end-diastolic and end-systolic volumes (Elliott, Aitken, & Chaboyer, 2007). The physician often prescribes vasopressors and inotropic medications such as milrinone or dobutamine to improve contractility and pumping efficiency of the damaged heart as well as reduce afterload.

In cardiogenic shock, the values of cardiac output (normal range of 4-8L/min) and the more accurate measurement, cardiac index (2.5-4.0L/min), can significantly drop in response to heart failure and the pump not being able to adequately circulate blood through the body. In some cases the physician may order a diuretic such as furosemide to be administered to decrease preload by reducing pulmonary (LV) or systemic (RV) congestion and stasis of blood. Another measurement of end organ perfusion that clinicians rely on is mean arterial blood pressure (MAP) which has a normal range of 60 to 110mmHg. When the body is in a state of shock, at first the body attempts to compensate, however as the condition prolongs the values gradually drop

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below 60 mm Hg along with a decreasing cardiac output (Holcomb, 2002). Together these hemodynamic values are used to determine if the patient has inadequate organ perfusion and to evaluate the adequacy of interventions by both the physicians and nurses.

Subsequently, the body attempts to compensate by increasing heart rate, which decreases diastolic filling time. This faster rate increases the oxygen demand of already damaged heart muscle, which negatively impacts cardiac output even further. Unable to keep pace with the increase in volume, hemodynamic values worsen as the heart fails to perfuse the body. This eventually leads to MODS and unfortunately, as a result, death in around 50% of all patients affected by cardiogenic shock (Babaev, Frederick, & Pasta, 2005). Even in cases where the patient is recovered and stabilized, sometimes the damage may be too great for the patient to overcome and within days or weeks they irreversibly deteriorate.

Treatments

The best chance of recovery for Mrs. Rudd relies on rapid percutaneous or surgical revascularization. While awaiting revascularization there are nursing interventions and pharmacologic measures that can be started to optimize her cardiac output. As mentioned earlier, rapid infusion of fluids is the first line of defense to improve cardiac output and stroke volume in right AMI. Diuretics may be used in left heart failure to decrease preload and improve the heart's pumping ability, however it is contraindicated with right AMI as in the case of Mrs. Rudd. Tachycardia is a common compensatory mechanism of cardiogenic shock to improve perfusion, yet beta-blockers, although they

can lower rapid heart rates, are another class of drugs that should be avoided for Mrs. Rudd since they also have a hypotensive effect and could counter the positive effects of fluid resuscitation and further perpetuate severe hypotension, having a bottoming out effect. Therefore beta-blockers should be used carefully and only in early stages of uncomplicated AMI without heart failure (Elliott, Aitken, & Chaboyer, 2007).

Providing inotropic support and improving systemic vascular resistance are important in the management of cardiogenic shock. To increase contractility and consequently cardiac output, inotropes, such as dobutamine, dopamine and milrinone, might be started and gradually increased to obtain adequate perfusion. However, because they can increase myocardial oxygen demand in an already ischemic heart, they must be used cautiously in patients, as they may lead to the incidence of fatal dysrhythmias. To treat life-threatening ventricular dysrhythmias (VT or VF), antiarrhythmic medications such as amiodarone or lidocaine are indicated. Additional considerations to treat dysrhythmias or heart block include defibrillators and transcutaneous pacing, or depending on the damage present, a permanent pacemaker (ECC Committee, American Heart Association, 2005).

Another complication of cardiogenic shock that increases oxygen demand is hypoxemia from pulmonary edema and backflow; common in left AMI and often a secondary complication in right AMI. As the patient deteriorates and stops responding to compensatory mechanisms, mechanical ventilation may be warranted to provide adequate oxygenation. By instituting mechanical ventilation the workload of breathing is decreased as the machine takes over. Also anxiety and metabolic demands are decreased when the patient is <https://assignbuster.com/right-ventricular-and-left-ventricular-cardiogenic-shock/>

sedated and intubated. Unfortunately, sedation may cause a further drop in blood pressure and needs to be closely monitored. Maintenance of adequate MAP is also vital to prevent end-organ damage.

To increase MAP, norepinephrine may be added to the medication regimen, but it may have a negative effect on cardiac output. Through means of combination therapy in severe hypotension, catecholamines are mainly administered in cardiogenic shock along with monitoring urinary output and calculating cardiac output, to sustain functioning of the patient's organs and optimistically to buy time before revascularization and the return of adequate pump function (Elliott, Aitken, & Chaboyer, 2007).

Unless contraindicated, it is protocol to treat any patient admitted with a diagnosis of acute coronary syndromes, including patients in cardiogenic shock, with aspirin and IV anticoagulation (heparin) to slow the progression of the infarct. Fibrinolytics are not recommended in patients requiring percutaneous coronary intervention (PCI) or surgery, however, improvement in hospital mortality with the use of the glycoprotein IIb-IIIa inhibitor abciximab (ReoPro) has recently been shown to reduce mortality from 40% to 50% down to 18% to 26% in cardiogenic shock treated with stent implantation (ECC Committee, American Heart Association, 2005). Hospitals have adopted the PTCA guidelines set forth by The American College of Cardiology and the American Heart Association aiming to provide reperfusion of the infarct artery within 90 minutes after arrival to the hospital. These new guidelines also assist in decision making regarding PCI, a nonsurgical coronary revascularization procedure that relieves the narrowing or obstruction of the coronary artery or arteries to allow more blood and

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oxygen to be delivered to the heart muscle. This ensures patient safety and improves patient quality of care (ECC Committee, American Heart Association, 2005).

Another intervention that is used to prevent or manage cardiogenic shock is an intra-aortic balloon pump (IABP) which improves coronary artery perfusion and reduces afterload. This mechanical device consisting of a 34- to 40-mL balloon catheter, is placed during PCI and operates by using counterpulsation therapy. The IABP inflates during ventricular diastole (increasing coronary artery perfusion) and deflates during ventricular systole (decreasing afterload or the resistance against which the heart has to pump). By increasing coronary artery perfusion with this device, the patient's cardiac output, ejection fraction, and MAP are increased, ultimately improving end-organ perfusion. Heart rate and pulmonary artery pressures, especially pulmonary artery diastolic and wedge pressures are lowered, which essentially decrease the heart's oxygen consumption, blood volume and workload (Holcomb, 2002).

Now an hour and fifteen minutes from onset, Mrs. Rudd's status continues to worsen and the physicians, noting that they are still within the 90 minute timeframe, rush her directly to the cardiac catheterization laboratory and she undergoes PCI with drug-eluting stent placement in the right coronary artery. During the procedure the surgeon places an IABP to increase coronary artery perfusion and decrease workload of the heart. She is transferred to the surgical intensive care unit for further management and monitoring with a pressure dressing to the femoral artery where the catheter was inserted. After recovery of her strength and hemodynamic stabilization, <https://assignbuster.com/right-ventricular-and-left-ventricular-cardiogenic-shock/>

she is transferred to a medical-surgical unit, then discharged four days later on a new medication regimen prescribed to avoid complications and recurrence of an AMI or lethal dysrhythmias.

Nursing Diagnoses & Outcomes

Prioritizing nursing diagnoses and care depends on which side of the heart is affected. With LV failure, respiratory complications are a primary consideration, whereas with RV failure, presentation of systemic signs and symptoms occur early on. Eventually as the patient's condition deteriorates their body's compensation mechanisms fail and cardiogenic shock worsens. Nursing interventions vary based on what stage of shock the person is in, their etiology and presentation, what procedures are planned or have been performed and when care is assumed.

The nursing diagnoses for Mrs. Rudd consist of (in priority):

Risk for decreased cardiac output related to altered cardiac rate and rhythm; reduced preload and increased systemic vascular resistance; infarcted muscle.

Ineffective tissue perfusion related to reduction or interruption of blood flow.

Risk for excess fluid volume related to decreased organ perfusion; increased sodium and water retention; sequestering of fluid in interstitial space and tissues.

Acute pain related to ischemic myocardial tissue.

Anxiety and fear related to change in health status.

Activity intolerance related to imbalance between myocardial oxygen supply and demand; presence of ischemia; cardiac depressant effects of certain drugs, such as beta blockers, antidysrhythmics.

Ineffective protection related to the risk of bleeding secondary to thrombolytic therapy.

Deficient knowledge regarding cause and treatment of condition, self-care, and discharge needs related to lack of information, misunderstanding of medical condition or therapy needs

Some expected outcomes for Mrs. Rudd include:

Maintain an adequate cardiac output during and following reperfusion therapy.

Demonstrate no signs of internal or external bleeding.

Rate chest pain as 2 or lower on a pain scale of 0 to 10.

Verbalize reduced anxiety and fear.

To recap, cardiogenic shock is a life-threatening complication of AMI. It is important to acknowledge that patient survival and recovery rely on early recognition of signs and symptoms of cardiogenic shock and rapid assessment and interventions by the nurse and treatment team.