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# Perioperative Management of Patient with Coronary Artery Disease 3 Vessels Disease, Chronic Total Occlusion in Left Anterior Descending and Right Coronary Artery, History of Hypertension, Extensive Myocardial Infarction and Low Ejection Fraction: A Case Report

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## Abstract

Introduction: Achieving a satisfactory hemodynamic performance is the primary objective in the management of cardiac surgery patient. Optimal cardiac function ensures adequate perfusion and oxygenation of other organ systems (in particular vital organs) and improves the chances for an uneventful recovery from surgery. Case: A 46 year old male diagnosed with Coronary Artery Disease 3 Vessel Disease (CAD 3 VD), CTO in LAD and RCA, history of hypertension, extensive myocard infarction with hypertrophy as well as global function impairment of the left ventricle and low ejection fraction, who underwent Coronary Artery Bypass Graft (CABG). Supportive medications, such as dobutamine and nitroglycerin (NTG) were initiated and maintained intraoperatively, especially after anesthetic induction due to hemodynamic alterations. Patient was able to wean from cardiopulmonary bypass (CPB) machine and transferred to the ICU postoperatively.

**Keywords:** CAD 3VD, Low Ejection Fraction, Myocard Infarct, Perioperative Management

## 1. Introduction

Once a patient is considered a candidate for cardiac surgery, a comprehensive evaluation of the patient's overall medical condition and comorbidities is essential. This includes a detailed history and physical examination, which may identify cardiac and noncardiac problems that might need to be addressed perioperatively to minimize postoperative morbidity (Robert M.B, 2011).

Patients undergoing cardiac surgical procedures are extensively monitored. Hemodynamic alterations and myocardial ischemia that occurs during the induction of anesthesia, in the prebypass period, during CPB, and

following resumption of cardiac activity can have significant adverse effects on myocardial function and recovery. It should be noted that even though both hypertension and tachycardia can increase myocardial oxygen demand, an increase in heart rate results in more myocardial ischemia at an equivalent increase in oxygen demand. Standard monitoring in the operating room consists of a five-lead ECG system, central venous catheter (CVC), a radial arterial line, pulse oximetry, an end-tidal CO<sub>2</sub> measurement, a Swan-Ganz pulmonary artery (PA) catheter, cerebral oximetry, and a urinary Foley catheter to measure urine output, core body temperature and intraoperative transesophageal echocardiography (TEE) should be obtained (Loeb HS, et al., 1978).

Anesthetic management must be individualized, taking into consideration the patient's age, comorbidities, the nature and extent of coronary or valvular disease, the degree of left ventricular dysfunction, and plans for early extubation. These factors will determine which medications should be selected to avoid myocardial depression, tachycardia, or bradycardia, or to counteract changes in vasomotor tone. Generally, a balanced anesthetic technique using a combination of narcotics and potent inhalational agents is used for all open-heart surgery to minimize myocardial depression (Robert M.B, 2011).

## 2. Case

A 46 years old male diagnosed with CAD 3 VD, was admitted electively to the Hasan Sadikin general hospital and scheduled for coronary artery bypass graft (CABG). The patient experienced shortness of breath and chest discomfort with anginal pain. He had a past history of hypertension for over 10 years. He was diagnosed with CAD since 2021 and was on medical therapies NTG retard 2x2,5 mg, ramipril 1x10 mg, bisoprolol 1x2,5 mg, atorvastatin 1x20 mg, clopidogrel 1x75 mg. He was a former smoker. He weighs 65 kg with the height of 170 cm. Physical examination and evaluation at the time of admission did not reveal any other significant findings. Coronary angiography showed chronic total occlusion (CTO) in mid left anterior descending (LAD), CTO in distal right coronary artery (RCA) and 80-90% stenosis in distal left circumflex artery (LCX). Echocardiography revealed failing left ventricular systolic function with an ejection fraction (EF) of 27% and myocard perfusion scan showed extensive myocard infarction (from apical to basal) with hypertrophy as well as global function impairment of the left ventricle. His preoperative blood examination was unremarkable. His renal and liver functions were normal, and the chest radiograph showed cardiomegaly.

In the preparation room, five-lead ECG was placed and we inserted a 16 G peripheral venous catheter. Arterial line was conducted through the left radial artery to provide invasive blood pressure monitoring. The patient's vital signs upon arrival in the operating room were as follows: BP 170/100 mmHg, HR 78x/min, RR 16x/min and oxygen saturation 100%. Anesthesia induction was commenced with 4mcg/kg fentanyl, 0.05 mg/kg midazolam, propofol 20mg, 1.2 mg/kg rocuronium, 1.5 mg/kg lidocaine. After reaching suitable muscle relaxation, the patient was intubated with endotracheal tube number 8. We inserted a left femoral artery cannulation for intra-aortic balloon pump (IABP) access, central venous catheter (CVC) via the left subclavian vein and Swan-Ganz catheter via the right internal jugular vein. Transesophageal echocardiography (TEE) probe was also established. Anesthesia was maintained with oxygen-air mixture and sevoflurane. Prior to incision, fentanyl 100 mcg and rocuronium 20 mg were administered and dobutamine (3- 5mcg/kg/min) and NTG (0.1mcg/kg/min) were initiated through central venous catheter, due to an episodes of hemodynamic alterations and continued intraoperatively, until the end of surgery.

The patient underwent CABG 3 VD with the aid of cardiopulmonary bypass (CPB) where the left internal thoracic artery was anastomosed to LAD, saphenous venous grafts were anastomosed to OM1, and posterior diagonal artery (PDA). Myocardial protection was achieved using cold cardioplegia. CPB time was 132 minutes and aortic cross clamp time was 92 minutes. The total duration of surgery was about 5 hours 15 minutes, with total blood loss 1200 ml, urine output 870 ml, and 3 packs of packed red cell (PRC) were transfused intraoperatively. He was transferred to the ICU with a continuous infusion of dobutamine (10mcg/kg/min), nor epinephrine (0.05mcg/kg/min) and NTG (0.1mcg/kg/min). Patient was gradually weaned off ventilator and extubated on the 1<sup>st</sup> post-operative day. He transferred to ward on 2<sup>nd</sup> post-operative day.

### 3. Discussion

Myocardial ischemia occurs when the oxygen supply to the heart is insufficient to meet metabolic needs. This mismatch can result from a decrease in oxygen supply, a rise in demand, or both. The most common underlying cause of myocardial ischemia is obstruction of coronary arteries by atherosclerosis. In the presence of such obstruction, transient ischemic episodes are usually precipitated by an increase in oxygen demand as a result of physical exertion. Ventricular hypertrophy due to hypertension can predispose the myocardium to ischemia because of impaired penetration of blood flow from epicardial coronary arteries to the endocardium (Thomas HL, 2013).

The achievement of satisfactory hemodynamic performance is the primary objective of postoperative cardiac surgical management. Optimal cardiac function ensures adequate perfusion and oxygenation of other organ systems and improves the chances of an uneventful recovery from surgery. Even brief periods of cardiac dysfunction can lead to impairment of organ system function, leading to potentially life-threatening complications. The important concepts of perioperative cardiac care are those of cardiac output, tissue oxygenation, and the ratio of myocardial oxygen supply and demand (Robert M.B, 2011).

Contractility is the intrinsic strength of myocardial contraction at constant preload and afterload. However, it can be improved by increasing preload or heart rate, decreasing the afterload, or using inotropic medications. Contractility generally reflects systolic function as assessed by the ejection fraction, but is only indirectly related to the cardiac output. Conversely, a low cardiac output does not necessarily imply that ventricular function is impaired. It may be noted with slow heart rates, with hypovolemia, and with a small, hypertrophied ventricle. Nonetheless, the state of contractility is usually inferred from an analysis of the cardiac output and filling pressures, based upon which steps can be taken to optimize hemodynamic performance. In cardiac surgery patients, the cardiac output is usually obtained by thermodilution technology using a Swan-Ganz catheter and bedside computer.

**Myocardial O<sub>2</sub> demand (mvO<sub>2</sub>)** is influenced by factors similar to those that determine the cardiac output (afterload, preload, heart rate, and contractility). Reducing afterload will generally improve cardiac output with a decrease in mvO<sub>2</sub>, whereas an increase in any of the other three factors will improve cardiac output at the expense of an increase in mvO<sub>2</sub>. Preoperative management of the patient with ischemic heart disease is primarily directed towards minimizing O<sub>2</sub> demand. **Myocardial O<sub>2</sub> supply** is determined by coronary blood flow, the duration of diastole, the coronary perfusion pressure, the Hb level, and the arterial oxygen saturation. When complete revascularization has been achieved, postoperative management is directed towards optimizing factors that improve O<sub>2</sub> supply and, to a lesser degree, minimize an increase in O<sub>2</sub> demand.

A low cardiac output state in patients with a history of CAD may result from abnormal preload, contractility, heart rate, or afterload. It may also be noted in patients with satisfactory systolic function but marked left ventricular hypertrophy and diastolic dysfunction. Management therapy : 1. **Ensure satisfactory oxygenation and ventilation** ; 2. **Treat ischemia or coronary spasm** if suspected to be present. Myocardial ischemia often responds to intravenous nitroglycerin (NTG) but may require further investigation if it persists; 3. **Optimize preload** by raising filling pressure with volume infusion; 4. **Stabilize the Heart rate and Rhythm**; 5. **Improve contractility** with inotropic agents. This should be based on an understanding of the  $\alpha$ ,  $\beta$  or nonadrenergic hemodynamic effects of vasoactive medications and their anticipated effects on preload, afterload, heart rate, and contractility; 6. **Reduce afterload**; 7. **Maintain blood pressure** (Robert M.B, 2011).

If the cardiac output remains low despite pharmacologic support, physiologic support with an **intra-aortic balloon pump (IABP)** should be strongly considered. If the patient cannot be weaned from bypass or has hemodynamic evidence of severe ventricular dysfunction despite maximal medical therapy and the IABP, use of a circulatory assist device should be considered.

The main perioperative concern in our patient was his past history of hypertension for over 10 years, CAD (CTO in LAD and RCA, 89-90% stenosis in LCX), low EF (27%), extensive myocardial infarction (from apical to basal)

with hypertrophy as well as global function impairment of the left ventricle. Providing safe anesthesia to these patient who are posted for CABG has always been challenging. Hemodynamic alterations and myocardial ischemia that occur during the induction of anesthesia, in the prebypass period, during CPB, and following resumption of cardiac activity can have significant adverse effects on myocardial function and recovery. It should be noted that even though both hypertension and tachycardia can increase myocardial oxygen demand, an increase in heart rate results in more myocardial ischemia at an equivalent increase in oxygen demand (Robert M.B, 2011).

We believe that commencement of Dobutamine (3-5mcg/kg/min) and NTG (0.1mcg/kg/min) after anesthesia induction and continued intraoperatively until the end of surgery might have prevented some of the possible adverse outcome in our patient. **Dobutamine is predominantly  $\beta$ -1-adrenergic agent**, making it a potent inotrope useful in stimulating the rate and the force of cardiac contraction with less arrhythmogenic potential. It has been utilized in the treatment of acute myocardial and congestive heart failure as it causes marked improvement in cardiac outuput and stroke volume without increase in heart rate. **Nitroglycerin is a direct vasodilator**, producing greater venous than arterial dilatation, this effect usually reduces Mvo<sub>2</sub> and increases diastolic coronary blood flow and relieves coronary spasm. It provides more flow to ischemic myocardium and increases endocardial-to-epicardial flow ratio and improved inotropy of myocardium (Westfall TC & Westfall DP, 2011) (Nirvik & John, 2019).

#### 4. Conclusion

Although excellence in pre and postoperative care can often make the difference between an uneventful and a complicated recovery, the care provided in the operating room usually has the most significant impact on patient outcome. Performing a technically proficient, complete, and expeditious operation is only one component of this phase. Refinements in anesthetic techniques and monitoring, cardiopulmonary bypass (CPB), and myocardial protection have enabled surgeons to operate successfully on extremely ill patients with far advanced cardiac disease and multiple comorbidities (Robert M.B, 2011).

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