Case Report

Anesthesia Management in a Hypertrophic Cardiomyopathy Patient with Brain Tumor: A Case Report

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Abstract

Background: Brain tumour surgeries are high-risk because of their potential for postoperative fetal complications. This risk is exaggerated in the presence of hypertrophic cardiomyopathy (HCM). Hypertrophic cardiomyopathy includes asymmetric left ventricular hypertrophy with mitral valve dysfunction that obstructs the left ventricular outflow tract. Various postoperative factors may accelerate this obstruction and lead to life-threatening consequences. Hypertrophic obstructive cardiomyopathy (HOCM) is a type of HCM that obstructs the left ventricular outflow tract. The most important parameters are preload, afterload, and ventricular contraction, resulting from the surgical method, anesthesia factors, and changes in intravascular volume that are prone to fluctuations in HOCM patients in the postoperative period. These cases increase the risk of arrhythmias and myocardial ischemia and significantly increase patients' morbidity and mortality after surgery. It should avoid lowering blood pressure, increasing heart rate, and reducing preload and afterload in these patients.

Cases Report: In this study, we reported a 59 years old man who complained of a speech disorder that had worsened three months ago and mentioned a history of hypertrophic cardiomyopathy. The patient underwent brain tumor surgery and was discharged from the hospital with proper management of anesthesia.

Conclusion: Loading and maintaining sinus rhythm are recommended to prevent complications. We presented the intraoperative management of a patient with HCM undergoing a brain tumour resection and discussed its complications.

Keywords: Anesthesia management, Hypertrophic cardiomyopathy, Brain tumour, Surgery

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Introduction

Anesthesia management is challenging for intracranial tumour surgery due to bleeding, cerebral ischemia, and heart failure complications. During intracranial tumour resection, the anesthesiologist is expected to maintain the patient and the surgeon's desired condition after surgery by maintaining proper brain perfusion, preventing cerebral edema, and increasing intracranial pressure, ensuring hemodynamic stability and rapid comfortable recovery with Providing early neurological evaluation. The most important approach is maintaining hemodynamic stability during surgery and minimizing side effects on intracranial pressure[1].

The risks of these surgeries increase in the presence of

concomitant heart diseases such as HCM, which may accelerate life-threatening complications such as arrhythmia, congestive heart failure (CHF), and even death. Usually, anesthesia management of these high-risk patients is challenging because of limited resources[2].

HCM means an abnormal thickening of the myocardium characterized by left ventricular (LV) or right ventricular (RV) hypertrophy without any cardiac or systemic cause and asymmetric ventricular septal hypertrophy[3]. With an incidence of 0.2%, HCM is the most common genetic heart disease and the most common cause of sudden cardiac death in young adults and athletes[4]. The hereditary pattern of HCM is autosomal dominant. HCM is characterized by a mutation in one of the genes encoding myocardial sarcomere proteins, leading to an abnormal myocyte growth pattern[5]. HOCM is a type of HCM that causes LV output obstruction (LVOT)[5]. The increased pressure gradient across LVOT in patients with HOCM can lead to circulatory collapse due to anterior mitral valve systolic movement and mitral regurgitation (MR)[5,6]. The most important parameters are preload, afterload, and ventricular contraction, which are prone to fluctuations in HOCM patients in the postoperative period due to surgical procedures, anesthesia factors, and changes in intravascular volume. These changes in blood circulation may increase the pressure gradient at the LVOT level, which increases the likelihood of myocardial arrhythmia and ischemia and may significantly increase morbidity and mortality in HCM patients[3].

Case Report

A 59 years old man complained of a speech disorder that had worsened three months ago. The patient had no movement disorders, headache, dizziness, nausea, and vomiting. He mentioned a history of diabetes from 8 years ago, which was being treated with metformin. He also mentioned a history of HCM. Apical HCM mild MR, mild Tricuspid regurgitation (TR), and (Ejection Fraction (EF) = 55% were reported in cardiac counseling and echocardiography. Our anesthetic goals included avoiding factors that worsen LVOT obstruction, such as tachycardia, dehydration, and peripheral vasodilators, maintaining appropriate preload and afterload, and having adequate intraoperative and postoperative pain control.

The patient received Nothing by mouth (NPO) for 8 hours before anesthesia and had received 300 ml of clear fluid 2 hours before anesthesia to minimize hypovolemia. It is also recommended that DC Shock be available to treat Ventricular tachycardia (VT) and Ventricular Fibrillation (VF) arrhythmias because the patient is prone to ventricular tachyarrhythmias. MRI showed a temporoparietal glioma (Figure 1: A, B). Blood tests and chest x-rays were normal. (Hb = 15.8) When assessing the patient, he was awake, but he had speech problems. At the onset of vital signs, BP = 120 / 65mmHg, SPO2 = 95%, HR = 45 / min. ECG changes in ST-T and ST depression were seen. First, in the awake state under local anesthesia, an arterial line was implanted, and 500cc of normal saline 0.9% was given and pre-oxygenated.

Vasopressors, phenylephrine and noradrenaline, blood products, defibrillators, and Osmolols were available in the operating room. General anesthesia was performed with 1 mg midazolam, 200 mic Fentanyl, 10 mg Etomidite, 20 mg Propofol, and 40 mg Atracurium. The patient was intubated with tube number 8. After intubation the monitor showed BP = 110/60 mmHg, HR = 43 min, SPO2 = 100%. Anesthesia maintained with propofol 100 micrograms per minute and Atracurium 10 mg every 30 minutes and intravenous fluids to maintain target hemodynamics (BP = 100-120 / 60-80 mmHg, HR = 40-60, U / O = 0.5cc / kg) Was preserved. The surgery lasted about 6 hours. One hour after the start of anesthesia, systolic blood pressure dropped to (75 mmHg), and bradycardia / 30 min happened after incision. Atropine 0.6 mg was administered twice, and the heart rate reached 48 / min. Also, after administration of 100 macro phenylephrine, systolic blood pressure was 100 mmHg. After that, the patient's vital signs did not change until the end of the surgery, and there was no need to prescribe vasopressor and inotropic medicines. He also has no ECG changes until the end of the surgery. The amount of bleeding during the operation was about 450cc, which was compensated by administering fluids. The total fluid received during the operation was 2000 cc normal saline and 2000cc Ringer. In total, patient's ABG sample was sent every hour. 1 g Acetaminophen

Table 1: Vital signs during surgery (Every hour).

HR beats/min	45	30	48	45	43	45
BP mmHg	120/65	75/30	100/50	100/50	95/45	110/60

was prescribed to control the pain after the surgery, and he was transferred to recovery. He was monitored for one hour in recovery, and when the vital signs were stable (BP =100/50mmHg, HR = 45 / min, SPO2 = 98%), he was transferred to ICU.

Anesthetic Management: Hypertrophic cardiomyopathy is a sign of disease rather than a specific diagnosis, and there is any medication or incident that reduces myocardial contractility or increases preload or reduces afterload on left ventricular outflow. Conversely, sympathetic stimulation and vasodilation make LVOT-O worse[3]. During surgery, these patients may develop severe hypotension, myocardial ischemia, acute heart filler, and supraventricular or ventricular tachycardia[2]. The first manifestation during surgery may be unexplained hypotension or systolic marrow following acute bleeding or vasodilation caused by the drug. In patients treated with beta-blockers or Calcium Chanel Blockers (CCB), the drug should be continued in the near-term period[7]. Defibrillators should be available during surgery. Anti-anxiety medications should be prescribed to reduce sympathetic stimulation. They were given anesthesia and beta antagonist before laryngoscopy reduces sympathetic stimulation by intubation. Positive pressure ventilation reduces preload and exposes patients to LVOT obstruction. So, it is better to adjust the high respiratory rate and low current volume and avoid giving PEEP. For muscle relaxation, one should choose drugs that have little effect on systemic circulation. Pancuronium, which increases sympathetic activity, is not a good choice[7]. The anesthetic used should have little effect on preload and afterload. Medium-dose anesthetic gases are a good choice for this purpose. Hypotension should be treated with alpha-adrenergic agonists such as phenylephrine[8]. The use of beta-adrenergic agonists such as Ephedrine, Dopamine, and Dobutamine is contraindicated because they increase heart contractility and heart rate. Immediate replacement of lost blood and titration of venous fluids is essential to maintain preload and

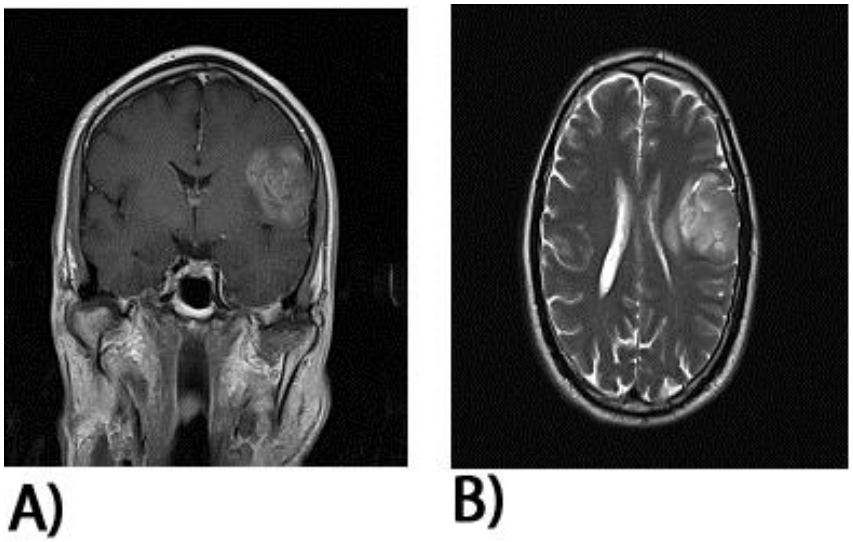

Figure 1. Magnetic resonance imaging shows Temporoparital glioma.

blood pressure. Vasodilators should not lower blood pressure as they reduce systemic vascular resistance and exacerbate left ventricular outflow obstruction. It is crucial to maintain a sinus rhythm. The best medicine for dysrhythmia is amiodarone[8].

Various postoperative factors such as tachycardia, Valsalva maneuver, laryngoscopy, intubation, preload and afterload reduction, and inotropic or chronotropic factors may exacerbate LVOTO in HCM, leading to hemodynamic complications. Therefore, maintain a sinus rhythm with a target HR of 60 to 70 bpm, accurate drug titration, and targeted fluid therapy. Vasopressors that do not make LVOTO worse, such as phenylephrine and noradrenaline, are recommended in HCM, while inotropes such as Dopamine, Dobutamine, Epinephrine, and Isoproterolone, which make LVOTO worse, should be avoided. Rapid monitoring with Atrial Blood Pressure (ABP), Central Venous Pressure (CVP), Pulmonary Venous Pressure (PVP), blood loss, and urinary excretion should be performed to lower blood pressure and other side effects. However, their availability and skill are still limited among anesthesiologists. An essential consideration in HCM is the prevention of sympathetic stimulation due to laryngoscopy, intubation, or surgical incision. The choice of suitable anesthetic drugs to reduce this stress response in HCM is limited[3,5].

Beta-blockers and calcium channel blockers are the preferred drugs for the treatment of HCM. Beta-blockers are beneficial because they reduce heart rate and prolong diastole, increasing ventricular filling and reducing myocardial oxygen demand. Ca2 + channel blockers such as Verapamil and Diltiazem are also helpful for relaxing and filling the ventricles. Fluids are recommended in HOCM cases—this helps to maintain intravascular volume while minimizing the side effects of positive pressure ventilation. In a patient with HCM for non-cardiac surgery, anesthesia management aims to prevent LVOT obstruction, arrhythmia, and diastolic dysfunction through the following strategies6: maintain sinus rhythm, decrease sympathetic activity, and maintain left ventricular filling, and maintain systemic vascular resistance.

The use of anti-anxiety drugs as prodrugs during surgery plays a vital role in reducing sympathetic activity and thus reducing heart rate. Invasive monitoring for ABP and CVP should be performed in HCM patients undergoing non-cardiac surgery.

In such patients, efforts to prevent tachycardia, hypotension, sympathetic stimulation, increased heart rate, and reduced preload and afterload are significant.

Discussion

Cardiomyopathies are a heterogeneous group of myocardial diseases with mechanical or electrical dysfunction that are usually disproportionately associated with hypertrophy or ventricular dilatation and have several causes in which genetic factors are common. Hypertrophic cardiomyopathy is a cardiovascular genetic disease that is autosomal dominant. In this disease, left ventricular hypertrophy is seen without the presence of any other disease that causes ventricular hypertrophy (such as hypertension or aortic stenosis). The most common form is septal hypertrophy and the free anterolateral wall. Pathophysiology of HCM Left ventricular (LVOT) is diastolic dysfunction, myocardial ischemia, and dysrhythmias. Sudden death is one of the complications of HCM and is mainly seen in patients aged 10-30 years[2,8].

Cardiomyopathy is a genetic heart disease with a prevalence of 0.2%. However, only 0.03% of patients are symptomatic, and the majority are diagnosed accidentally[4]. This includes asymmetric left ventricular (LV) hypertrophy with or without mitral valve leading to LVOT[5]. Hypertrophic LV leads to diastolic dysfunction, atrial fibrillation, Cardiac heart failure (CHF), and death. Patients are often treated with negative inotropic agents such as beta-blockers, calcium channel blockers, and Disopyramide. The goal of treatment is to reduce or eliminate the LVOT slope and ultimately reduce left ventricular outflow obstruction and fatal dysrhythmias[1,2]. Hypertrophic cardiomyopathy is a complex condition. The pathophysiologic determinants of clinical course and disease progression in HCM include dynamic obstruction to LV outflow, diastolic dysfunction, impaired coronary vasodilator reserve, myocardial ischemia, and supraventricular or ventricular tachyarrhythmias[5]. The symptoms include dyspnea with exertion, angina, and dizziness varying from

lightheadedness, presyncope, syncope, and sudden death. Medical therapy of HCM consists of beta-blockers and calcium channel blockers. Disopyramide may benefit in patients of hypertrophic obstructive cardiomyopathy with atrial fibrillation. The rationale for using beta-blockers is their ability for negative inotropic. Beta-blocker prolongs diastolic filling time by decreasing heart rate, decreases myocardial oxygen requirement (increase in left ventricular end-diastolic volume and a resultant decrease in LVOT gradient), and improves exercise tolerance. Diltiazem provides benefits due to ventricular relaxation and filling and control of ventricular rate. It also shortens intraventricular relaxation time and enhances early diastolic filling in patients with HCM. Atrial fibrillation and flutter are the most common dysrhythmias in patients with HCM and are associated with an increased risk of thromboembolism. Our patient was concerned about HCM, diabetes, brain tumours, and MR and TR. Also, the patient was not receiving cardiac medication. Although a patient with HCM may not show LVOT under initial conditions, its occurrence has been described during stimulation.

Take measures to prevent conditions that increase myocardial contraction (e.g., tachycardia, stress) or reduce preload and ventricular volume (e.g., vasodilators, hypovolemia, hypotension) because of the slope of the LVOT increases with decreasing volume. Maintaining sinus rhythm is very important in these patients due to the dependence of preload on atrial contraction. Prodrugs play an important role in reducing sympathetic activity and thus reducing heart rate. Preload before induction helps maintain stroke volume and possibly minimize the side effects of positive pressure ventilation. Invasive monitoring for BP and LV filling pressures should be performed in HCM patients undergoing non-cardiac surgery. HCM affects all ages and has unique and complex pathophysiology that includes dynamic blockage for LV outflow, diastolic dysfunction, coronary artery bypass grafting, myocardial ischemia, and supraventricular or ventricular tachyarrhythmias[4]. HCM can be classified as hypertrophic non-obstructive cardiomyopathy, apical HCM, and obstructive ventricular HCM. In addition, patients with the following criteria are diagnosed as having a severe illness: Moderate to severe physical activity limit (NYHA Class III / IV), a history of ventricular tachycardia or ventricular fibrillation, and hospitalization[5].

Conclusion

HCM is a group of myocardial diseases characterized by ventricular hypertrophy, left ventricular outflow obstruction, myocardial ischemia, diastolic dysfunction, dysrhythmia, and sudden death. In these patients, sympathetic stimulation, preload reduction, afterload, and consequent exacerbation of ventricular outflow obstruction should be avoided. In case of hypotension, the use of phenylephrine is beneficial, and also, by giving intravenous fluids, preload can be maintained. In the case of dysrhythmia, Amiodarone is the best treatment. Maintaining sinus rhythm and avoiding systemic vascular resistance are recommended.

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