

Anesthetic Management of a Patient with Hypertrophic Obstructive Cardiomyopathy in a Place with Limited Resources

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Abstract

Hypertrophic Obstructive Cardiomyopathy (HOCM) is a rare genetic disorder of heart characterized by Left Ventricular Outflow Tract (LVOT) obstruction. Clinical presentation ranges from absence of symptoms to sudden death. A 62 year old patient presented with left carcinoma breast was scheduled for modified radical mastectomy. The patient was being treated for hypertension but was diagnosed as HOCM just two weeks prior. Anesthetic management of these patients presents considerable challenges and requires maintenance of desired hemodynamic parameters and management of specific complications. Factors like tachycardia, hypovolemia, vasodilation and increased cardiac contractility leads to exacerbation of the obstruction. We managed to successfully maintain the desired hemodynamics throughout the surgery and the patient was discharged home on the postoperative day.

Keywords: Anesthesia; Case management; Hypertrophic obstructive cardiomyopathy

Introduction

Hypertrophic Obstructive Cardiomyopathy (HOCM) is a rare genetic disorder of the heart inherited as an autosomal dominant trait attributed to mutation of the gene that encodes for the sarcomere proteins. It is characterized by massive asymmetric hypertrophy of myocardium resulting in Left Ventricular Outflow Tract (LVOT) obstruction [1]. Clinical presentation ranges from absence of symptoms to sudden death [2]. Decrease in venous return and systemic vascular resistance or increase in myocardial contractility worsens the LVOT obstruction [3]. These patients are highly prone to arrhythmias like atrial fibrillation and Ventricular Tachycardia (VT) [2]. Management of anesthesia in these patients poses considerable challenges for the anesthesiologist. We report successful conduct of anesthesia in a patient with HOCM undergoing Modified Radical Mastectomy (MRM).

Case Report

A 62 year old female was scheduled for Modified Radical Mastectomy (MRM) for carcinoma of the left breast. She complained of gradually increasing painless mass in left breast and breathlessness on climbing a flight of stairs and had METS<4. She was a known case of Hypertension for ten years. She also has history of two episodes of TIA, 6 years and 4 years back. She was taking amlodipine and atorvastatin. Her examinations were normal except for hypertension, left shifting of apical impulse and ejection systolic murmur in the mitral area. The routine laboratory investigations were within normal limits. Her ECG showed Left ventricular hypertrophy with normal sinus rhythm. Her echocardiogram showed severe concentric left ventricular hypertrophy, a Grade II diastolic dysfunction, thickened Interventricular Septum (IVS) with LVOT obstruction of 60 mmHg and moderate Mitral Regurgitation (MR) with normal Systolic function (EF=60%). She was started on metoprolol after echocardiography findings.

She was premedicated with tablet diazepam 10 mg at 10 pm the day before surgery and 2 hours prior to surgery. After monitoring with ECG lead II, NIBP, SpO₂ and peripheral venous line insertion, an arterial catheter was placed in the left radial artery under local anesthesia. External defibrillator was kept standby. General anesthesia was induced with fentanyl (2 µg/kg), propofol (titrated to 80 mg) and vecuronium (0.1 mg/kg). Intubation reflex was blunted with esmolol and lidocaine

(1.5 mg/kg) and a 7.5 mm ID endotracheal tube was inserted smoothly. Intermittent positive pressure ventilation with oxygen and isoflurane adjusted to 1.2 MAC with fentanyl and vecuronium boluses were used for maintenance of anesthesia. After induction a 7Fr double lumen central line was inserted in right subclavian vein for monitoring of Central Venous Pressure (CVP) and drugs infusion if needed. She was infused with gelatin 500 ml to achieve CVP of 12-15 cm H₂O.

The surgery lasted for 80 minutes during which there was one episode of hypotension fifteen minutes after induction, which responded to a 100 ml bolus of normal saline and 50 µg phenylephrine. Thereafter she remained hemodynamically stable and her CVP was maintained at 12-13 cm H₂O. At the end of the procedure reversal of the neuromuscular block was achieved by neostigmine and glycopyrrolate. Extubation reflex was blocked with esmolol and lidocaine. She was extubated and was transferred to the coronary intensive care unit. Patient remained stable hemodynamically. She was discharged home on the seventh postoperative day.

Discussion

Diagnosis of HOCM is established with noninvasive cardiac imaging, including echocardiography and/or Cardiac Magnetic Resonance Imaging (cardiac MRI). Findings on transthoracic echocardiography include asymmetric myocardial hypertrophy and Systolic Anterior Motion (SAM) of the mitral valve with associated left ventricular outflow tract obstruction and mitral regurgitation. Histopathologic features include myocardial fibrosis and myocyte disarray. Systolic anterior motion of the mitral valve leads to LVOT obstruction and often precipitates mitral regurgitation [3,4]. Diastolic dysfunction occurs due to impaired ventricular compliance [2]. All

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these features were present in our patient. Factors such as tachycardia, hypovolemia, vasodilatation and increased cardiac contractility exacerbate the obstruction [3]. Anesthetic management entails maintenance of desired hemodynamic parameters, and management of specific complications like hypotension, dysrhythmias and congestive heart failure [5,6]. Sinus rhythm is crucial in these patients because of the dependence of preload on atrial contraction.

Our patient had a severe LVOT obstruction with peak gradient of 60 mmHg. Negative inotropic drugs like beta blockers and calcium channel blockers are used to decrease the degree of outflow tract obstruction [4,7]. Our patient was on amlodipine which was continued perioperatively, while intraoperatively the dose dependent myocardial depression caused by inhalation anesthetics might have helped in this regard. In our patient dose selection of anesthetic and ancillary medication based on clinical experience, convenience, cost, pharmacokinetic and pharmacodynamic properties of the drugs [8]. In addition to these approaches, combine clinical pharmacokinetics with pharmacogenetics based approaches are also emerging for dose selection of medications [9]. These patients should be given adequate premedication to reduce stress. Avoidance of vasodilators and agents that increase contractility is essential during the anesthetic management [2,7]. Episodes of hypotension can be treated with volume replacement and/or vasoconstrictors like norepinephrine or phenylephrine. Our patient responded well to fluids and phenylephrine. We used IV fluids judiciously and kept the CVP at 10-15 cm of H₂O as adequate preload is necessary to maintain optimal cardiac output and avoid undue increase in contractility because of hypovolemia [7]. Although CVP may be an inaccurate guide to filling pressure due to abnormalities in left ventricular compliance, we decided to keep CVP for accurate fluid management and drug infusion during an MRM procedure.

Conclusion

Management of anesthesia in a patient with HOCM is challenging for anesthesiologists which is even more complicated by the absence of advanced cardiac monitoring system. Such case can be safely managed

in a place with limited resources if the pathophysiology of the disease is clearly understood and the hemodynamic goals are maintained. Key points in anesthetic management include a) maintenance of sinus rhythm, b) adequate preloading, c) avoidance of vasodilatation, d) increase cardiac contractility, e) management of hypotension with vasopressors, f) immediate management of dysrhythmias and congestive heart failure.

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