

Anaesthetic management of a patient with hypertrophic obstructive cardiomyopathy undergoing laparoscopic cholecystectomy

Gautam B¹, Bhochhibhoya A²

¹Binod Gautam, Assistant Professor, ²Anisha Bhochhibhoya, Third Year Resident, Department of Anaesthesiology and Intensive Care, Kathmandu Medical College Public Limited

Abstract

Hypertrophic cardiomyopathy is a genetic disorder characterized by asymmetric left ventricular hypertrophy with or without left ventricular outflow tract obstruction. From asymptomatic state to sudden death comprise its varied clinical range. Owing to complex patho-physiology and very high risk of cardiac events, its peri-operative management demands meticulous maintenance of hemodynamic parameters. Prevention and prompt correction of factors such as hypovolemia, tachycardia, and increased contractility that can exacerbate the left ventricular outflow obstruction, arrhythmias and myocardial ischemia are essential for improving patient outcome. Laparoscopic surgeries in these patients can further amplify anaesthetic concerns and add to the challenges. A 58 year female was scheduled for surgery having suffered frequent complications of cholelithiasis. As there is paucity of reports evaluating laparoscopic surgery, we report this patient diagnosed with hypertrophic obstructive cardiomyopathy, who received general anaesthesia to undergo laparoscopic cholecystectomy successfully without suffering complications during surgery and hospital stay.

Key words: Anaesthetic management; Hypertrophic obstructive cardiomyopathy; Laparoscopic cholecystectomy; Left ventricular outflow tract obstruction.

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INTRODUCTION

Hypertrophic obstructive cardiomyopathy (HOCM) is a mutational disorder of myocardial sarcomere proteins transmitted as an autosomal dominant trait with genotypic and phenotypic variability. Echocardiographic diagnosis is established by asymmetric left ventricular hypertrophy with non-dilated ventricular chamber¹. With prevalence of one in 500, its clinical presentation ranges from being asymptomatic to heart failure and sudden unexpected death².

Up to 60% patients with HOCM undergoing non cardiac surgery suffer myocardial infarction, congestive heart failure or both^{3,4}. Acute precipitation or exacerbation of left ventricular outflow tract (LVOT) obstruction, diastolic

dysfunction, myocardial ischemia and dysrhythmias increase peri-operative risks⁵. Neuro-endocrine stress, carbon dioxide pneumoperitoneum, and positioning to facilitate laparoscopic surgery can adversely affect hemodynamics adding to anaesthesiologists' challenges.

There are no published guidelines for anaesthetic management of HOCM patients and there is paucity of reports evaluating laparoscopic surgery. We present a patient with HOCM who underwent laparoscopic cholecystectomy successfully without suffering complications.

CASE REPORT

A 58 year old female, weighing 60 kg, was scheduled to surgery for cholelithiasis, which was complicated to biliary pancreatitis needing Intensive Care stays twice within the previous nine months. She was receiving metoprolol and diltiazem twice a day for nine months since she was diagnosed having HOCM. She would experience dyspnea and palpitations during moderate

Address for correspondence

Binod Gautam
Assistant Professor, Department of Anaesthesiology and Intensive Care
Kathmandu Medical College Public Limited, Baburam Acharya Sadak, Sinamangal, Kathmandu, Nepal
E-mail: gautambinod@hotmail.com

exertion. She denied of any sudden death in her family members.

On examination, heart rate (HR) was 68/min and blood pressure was 100/70 mmHg. Cardiac auscultation revealed apical systolic murmur. Airway and respiratory system examinations and routine blood investigations were within normal limits. Chest X-ray showed cardiomegaly. Electrocardiography revealed sinus rhythm at 70/min, left ventricular hypertrophy, and inverted T-waves in leads II, V4, V5, V6, aVF and aVL. Trans-thoracic echocardiography confirmed HOCM with asymmetric septal hypertrophy, LVOT peak gradient of 144 mmHg, moderate diastolic dysfunction, systolic anterior motion, moderate mitral regurgitation, dilated left atrium and ejection fraction of 60%.

Cardiac medications were continued and clear oral fluids were allowed till two hours before surgery. In operation room, standard American Society of Anesthesiologists' monitors were established. A 16 Gauge IV cannula was opened to infuse 500 ml Ringer's lactate. Ondansetron and Dexamethasone were administered for anti-emetic prophylaxis. Fentanyl 75 Mcg. was administered in increments. Analgesia was supplemented with Paracetamol one gram and Diclofenac 75 mg IV. An arterial line was opened in left brachial artery, under local anaesthesia, to monitor beat to beat blood pressure. Lignocaine 60 Mg. was administered five minutes before laryngoscopy.

Induction of general anaesthesia was done with sleep dose of Propofol (90 mg) and Vecuronium. She was ventilated with Halothane up to two percent for three minutes before laryngoscopy. Immediately after achieving smooth tracheal intubation and its confirmation, monitors read blood pressure at 80/50 mmHg with HR of 50/min. Glycopyrrolate 0.2 mg and a rapid fluid bolus of 300 ml were given. Hemodynamic parameters then after remained stable throughout procedure with HR between 65 and 75/min and blood pressure at 100-120/60-80 mm Hg. Anaesthesia was maintained with Halothane at one to 1.5% concentration in 100% Oxygen. Ventilation was adjusted to keep end-tidal carbon dioxide tension between 30 and 35 mmHg. Bupivacaine 0.25% in 30 ml was infiltrated at surgical port sites, diaphragmatic surface of liver and Callot's triangle before surgical incision and dissection.

Laparoscopic cholecystectomy was performed using three surgical ports. Pneumoperitoneum was created

with carbon dioxide at a flow rate of one litre/minute to maintain intra-abdominal pressure at eight mmHg. Patient was kept in reverse Trendelenberg position (15 degrees) with table tilted to left. Nasogastric tube and sub-hepatic drain placements were not needed. Procedure lasted for 45 minutes and she received 1400 ml Ringer's lactate.

At the end, neuromuscular block was reversed with Neostigmine and Glycopyrrolate. Esmolol 30 mg was administered to blunt extubation reflex. Patient was monitored electively in Intensive Care Unit for 24 hours which was uneventful. After four hours, she was started on oral fluids and her pain was well managed with regular Paracetamol and Ketorolac. Diltiazem was continued as pre-operatively, whereas single dose of Metoprolol was omitted. She did not have nausea-vomiting, chest pain, dyspnea and palpitation. Patient was discharged from hospital on the fourth post-operative day.

DISCUSSION

There are a few case reports published about anaesthetic management of HOCM patients undergoing laparoscopic surgery. HOCM is the most common genetic cardiac condition comprising a heterogenous clinical course determined by dynamic obstruction to LVOT, diastolic dysfunction, impaired coronary vasodilator reserve, myocardial ischemia, and arrhythmias. The major underlying structural abnormalities include myocardial cell disarray, coronary microvascular dysfunction with increased wall/lumen ratio, remodeling changes and mitral valve pathologies⁶. It is unique in its potential for presenting during any phase of life from infancy to the elderly. Sudden unexpected death, ischemic heart disease, heart failure and stroke are the major concerns in HOCM patients^{1,2,5}.

Pharmacological treatment of HOCM is based on balancing supply and demand of Oxygen to the heart. The goal is to control limiting heart failure and anginal symptoms, LVOT obstruction and arrhythmias⁷. Beta blockers remain the mainstay in treatment with the benefits attributed to decreased HR with prolonged diastolic relaxation and increased passive ventricular filling^{1,8}. Lessened cardiac contractility reduces incidence of LVOT obstruction, myocardial Oxygen requirement and possibly the ischemia. Non-dihydropyridine calcium channel blockers including Verapamil and Diltiazem are used similarly to reduce cardiac chronotropy and inotropy, leading to improved diastolic filling, reduced outflow gradient, and improved sub-endocardial perfusion⁸.

The goal of anaesthetic management in these patients appearing for non-cardiac surgery is to prevent occurrence of the most dreaded complication of LVOT obstruction. Similarly, prevention and aggressive correction of arrhythmias and controlling the consequences of diastolic dysfunction to avoid refractory hypotension and hypoxemia secondary to heart failure must be given a top priority³. Strategies to establish and maintain normal sinus rhythm, reduced sympathetic activity, filled left ventricle, and adequate systemic vascular resistance comprise the cornerstone of peri-operative care.

Pre-medication may decrease anxiety and sympathetic stimulation. However, our well-informed patient with two prior Intensive Care stays, having no difficulty in sleep, and well controlled HR made us not to opt for sedative medications. Hypovolemia with resultant tachycardia and increased contractility may precipitate LVOT obstruction emphasizing the role of proper peri-operative hydration⁵. Adequate fluid infusion is crucial to counteract vasodilation during anaesthesia induction. Cardiac loading also helps to minimize the adverse effects of positive pressure ventilation and pneumoperitoneum both of which may impair venous return. Acute hypervolemia, on the other hand, may complicate the diastolic dysfunction into heart failure and thus must be avoided. As we were not expecting major fluid shifts, and since central venous pressure monitoring may be an inaccurate guide to filling pressures in patients with diastolic dysfunction we decided not to monitor it.

Although both general and neuraxial anaesthesia can be used in HOCM patients, it is important to have a clear understanding of associated hemodynamic effects⁶. To avoid rapid reduction in systemic vascular resistance associated with spinal anaesthesia, we opted for general anaesthesia with the goal of maintaining afterload and preload and avoiding sympathetic stimulation. For induction and maintenance of anaesthesia, Halothane was used in our patient. Halothane has got least effects on systemic vascular resistance, decreases HR and it has got some myocardial depressant effect, all of these properties are beneficial in HOCM patients.

Refractory hypotension during surgery in patients with HOCM is a well described complication occurring

due to systolic anterior motion of mitral valve leaflet and consequent LVOT obstruction. Apart from addressing fluid management, its correction might need vasopressors, preferably the direct acting agents which are devoid of inotropic and chronotropic actions⁵. The overall hemodynamic goal includes maintenance of mean arterial pressure to preserve coronary perfusion pressure to the sub-endocardium of the hypertrophied heart. Hypotension and bradycardia occurring immediately after intubation in our patient must have been due to the effects of pre-operative cardiac medications and the agents used for anaesthesia induction. However, it was transient, responded well with anti-cholinergic, rapid fluid bolus, and reduced Halothane concentration.

Measures taken to avoid sympathetic surge, hypovolemia, excessive venodilation, hypothermia, hypercarbia, and pain prevented deterioration of our patient. To reduce possible negative impact of carbon dioxide pneumoperitoneum on cardiovascular system, slower flow rate, minimal intra-abdominal pressure and augmented alveolar ventilation for achieving eucapnia were utilized. In addition, reverse Trendelenberg position was allowed only for minimal degrees so as to avoid decrement in cardiac preload.

CONCLUSION

Hypertrophic obstructive cardiomyopathy, a complex cardiovascular entity might deteriorate the cardiac function of patients during the times of stress, including surgery and peri-operative period. The involved care givers must have a thorough understanding of its patho-physiology and implement strategies to prevent the predisposition or exaggeration of left ventricular outflow tract obstruction. The anaesthesiologist must have a meticulous planning and implementation of anaesthesia conduct to prevent and correct any of the sympathetic surge, arrhythmias, excessive venodilation, and hypovolemia for attaining an uncomplicated peri-operative course. Laparoscopic cholecystectomy may be safely conducted in patients with hypertrophic obstructive cardiomyopathy when hemodynamic goal of normal sinus heart rate/rhythm, adequate preload, maintained afterload, and reduced cardiac contractility is achieved.

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