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Anaesthetic Management of a Patient with Hypertrophic Obstructive Cardiomyopathy Undergoing Septal Myectomy

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Abstract

Hypertrophic Obstructive Cardio-Myopathy (HOCM) is a relatively common disorder that anesthesiologists encounter among patients in the perioperative period. It is autosomal dominant disorder having familial nature. Aim of this case report is to highlight the problems related to anaesthetic management of such patients. Patient admitted was a 50 years old male (weight 72 kilogram) with complaints of chest pain on exertion. Two dimensional Ecocardiography (2D Echo) showed Systolic Anterior Motion (SAM) of mitral valve, dynamic left ventricle outflow tract obstruction, severe constrictive left ventricular hypertrophy and interventricular septal thickness was 20mm. Optimal anaesthetic management in patients with HOCM involves monitoring adequate preload, optimal systemic vascular resistance and minimal outflow obstruction during intra operative period. Vigilant Intensive Care Unit (ICU) management and post operative care is mandatory for an uneventful recovery.

Keywords: Hypertrophic Obstructive Cardiomyopathy, Left Ventricle Outflow Tract Obstruction, Septal Myectomy

1. Introduction

Hypertrophic obstructive cardiomyopathy is relative common autosomal dominant disorder having familial nature with a prevalence of 1:500 in united states[1]. Male to female ratio is 2:1. The cardiac sarcomere proteins that causes asymmetric hypertrophy of segments of the ventricle and septum. When basal septum of left ventricle is affected Left Ventricle Outflow Tract Obstruction (LVOTO) can occur. Due to venturi effect SAM of mitral valve occurs. In these patient use of positive inotropic drugs, decrease in venous return or Systemic Vascular Resistance (SVR) can worsens LVOTO and serious problems might occur during anaesthesia.

2. Case Report

50 years old male patient was admitted with complain of chest pain on exertion for four to five years (NYHA class II). Patient was known case of hypothyroidism and was on Levothyroxine 100 µg .There was no history of any other chronic illness and surgery in the past. There was no family history of HOCM. Patient's blood pressure was 100/86, pulse rate 74/minute, Spo2 97%. Blood investigations were within normal limit .Viral marker tests were negative. Angiography showed normal coronaries with right coronary dominant. Echocardiography showed SAM, dynamic LVOT, severe constrictive left ventricular hypertrophy, LV ejection fraction and interventricular septal thickness was 60% and 20 mm respectively. Patient

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was premedicated with Injection morphine 0.1 mg/kg and inj promethazine 25 mg intramuscular 45 minutes prior to induction.

Patient was taken in OT after written informed consent. Through peripheral venous cannula Ringer lactate was started at the rate of 5ml/kg/hr standard monitoring included 12 leads ECG, pulse oximetry, temperature, End Tidal Carbon Dioxide ($ETCO_2$), CVP, intra arterial blood pressure and urine output. After adequate preoxygenation induction was done with inj Midazolam 0.05 mg/kg, inj fentanyl 3 µg /kg, inj. Etomidate 0.3 mg/kg and inj. Rocuronium 0.9mg/kg. IPPV done for 90 seconds, under direct laryngoscopy patient was intubated with 8.5mm cuffed ET. Haemodynamics were maintained to keep heart rate between 70-90 beats per minute, mean artery pressure 60-70 mmHg, $ETCO_2$ was maintained between 35-40 mmHg and CVP was kept higher (7-8 cm H_2O) in pre and post bypass period[2]. For maintenance of anaesthesia O_2, sevoflurane (0.6- 1 vol %) Inj. vecouronium 0.01 mg/kg, Inj fentanyl 20 µg and inj midazolam 0.4 mg was given at every 30 minutes. Sternotomy was done with electric saw and pericardium was incised. Before aortic cannulation inj heparin 4 mg/kg was given through central line. ACT was 520 sec.

Before going on bypass cardiac chamber pressures were measured with the help of surgeons. LV pressure 202/28 (108) mmHg, RV pressure 63/26 (46) mmHg, femoral pressure 103/64 (76) mmHg, aortic pressure 103/80(91) mmHg was assessed. Aortic and venous cannulation was done and patient was taken on Cardio Pulmonary Bypass (CPB). Surgery planned was septal myectomy (morrow's procedure). Anomalous muscle bundle causing LVOT was planned to be excised. After coming on CPB full flows were attained and ventilation was stopped. Del Nido cardioplagia was used. Mean arterial pressure was maintained between 50 to 70 mm Hg and temp 32° C. Flows were kept 3.5l/min on CPB. Septal myectomy was done and after rewarming patient was slowly weaned off CPB, heart rate of the patient was less than 50 beats per minute, so we started inj. isoprenaline in the dose of 0.05 µg/Kg/minute rate increased to 80 beats per minute. The rest gradients before myectomy were 99 mmHg and after myectomy were 13 mmHg. After isoproterenol infusion peak provoked gradient was measured which was not significantly increased. CVP was kept between 7-8 cm H_2O. Now LV pressure was 80/26 (52) mmHg, femoral pressure was 86/38(54) mmHg. Injection protamine 450 miligrams was given from peripheral line after test dose. ACT was 115 seconds. Systolic pressure was maintained between 100 to 110 mmHg and heart rate was maintained between 70 to 90 per minute. Patient was shifted to ICU and day after surgery patient was extubated with pressure 110/70, HR 86 per minute, saturation 95%. The ICU stage was uneventful. Patient was discharged from hospital on 10th post operative day.

3. Discussion

HOCM is characterized by LVOT obstruction caused by asymmetric ventricular septal hypertrophy and anterior displacement of papillary muscles and mitral leaflets. A two fold increase in wall to lumen ratio, predisposing patients to silent myocardial ischemia, ongoing myocardial injury and fibrosis[3]. Patients with HOCM can present early in life with symptoms or can live for decades asymptomatically. In our case patient present late with symptom of chest pain on exertion for 5 years as patient was not diagnosed till he came to tertiary level hospital. So he was not taking any medicines except tablet Thyroxine for hypothyroidism. After diagnosis of HOCM patient was taken on calcium channel blocker and β blocker. Modality of treatment varies from pharmacological management, alcohol septal ablation and surgical treatment. The gold standard for first consideration is still surgical septal myectomy, when performed in experienced centers in patients who are drug refractory and severely symptomatic[4,5]. Our goal for management was to maintain sinus rhythm, left ventricular diastolic filling phase, reduce secretion of catecholamines, systemic vascular resistance and venous return.

To avoid anxiety patient was pre medicated adequately. Injection Esmolol 0.5 mg/Kg given intravenously to blunt intubation response Preload was kept adequate to prevent hypovolemia.

Patient was induced with injection Etomidate to avoid vasodilation and decrease venous return. There was no rhythm abnormality in our case but arrhythmias may be problematic in such patients. Atrial fibrillation and junctional rhythm are poorly tolerated in HOCM patient

due to loss of atrial kick. Arrhythmias, particularly atrial fibrillation require prompt treatment[6]. β blockers and amiodarone are drugs to control arrhythmias. Del Nido Cardioplegia solution was used for myocardial protection. Monitoring of ABG and ACT was done regularly to maintain pH, pCO2, pO2, ACT (>480 sec) during cardiopulmonary bypass. Serum potassium was maintained between 3.5 to 4.5. Intraoperative temperature and urine output was monitored. Although Trans-Esophageal Echocardiography (TEE) is essential to guide surgical myectomy, assess the myectomy, MV repair and to prevent potential complication. TEE provides important information about residual gradients, obstructive SAM, septal anatomy and the need for immediate post myectomy revision. But it is essential to attempt provocation of LVOTO to evaluate adequate resection. For this purpose we used injection Isoprenaline to enhance β_1 β_2 agonist properties leading to LVOTO by stimulating exercise. An adequate response (starting from 1μg/Kg/min reduced to 0.05 μg/Kg/min) would result in increase in heart rate to more than 125/min. So in patient with resting gradient 25 mm Hg or post isoprenaline administered gradient more than 50 mm Hg indicates prompt revision of myectomy[7,8]. In our case there was no need of revision of myectomy. Intra operative direct measurement of gradient with the help of surgeons can give more accurate information. So we relied on this method as intra operative TEE monitoring was not available in our operation theatre. Echocardiography guides and demonstrates the degree of hypertrophy and may show systolic anterior movement of the mitral valve, but this is often a dynamic situation and its absence before operation does not mean that it will not occur intraoperatively[9]. Minimal dose of inotropes was required to wean off from CPB. Systolic blood pressure was maintained between 100 to 110 mmHg and heart rate was maintained between70 to 90 per minute. Adequacy of preload was judged by CVP monitoring (7-8cm H_2O). Hypotension if occurs should be treated with judicious volume resuscitation and alpha-agonists. The use of agents with inotropic or chronotropic actions can increase myocardial oxygen demand. We did not encountered hypotension in perioperative period.

4. Conclusion

Optimal anaesthetic management in patients with HOCM involves adequate monitoring of preload by optimizing systemic vascular resistance and minimizing outflow obstruction during intra operative period. Vigilant ICU management and post operative care is mandatory for an uneventful recovery.

5. References

1. Towbin JA. Hypertrophic cardiomyopathy. Pacing Clin Electrophysiol, 2009; 32 Suppl 2:S23-31. https://doi.org/10.1111/j.1540-8159.2009.02381.x. PMid:19602159.
2. Agarwal N, Kapoor PM, Chaudhary S, Kiran U. Anaesthesia management of a patient with hypertrophic obstructive cardiomyopathy undergoing morrow's septal myectomy. Indian J. Anaesth., 2007; 51:134-136.
3. Camici PG, Olivotto I, Rimoldi OE. The coronary circulation and blood flow in left ventricular hypertrophy. J. Mol. Cell. Cardiol., 2012; 52:857-864. https://doi.org/10.1016/j.yjmcc.2011.08.028. PMid:21924273.
4. Gersh BJ, Maron BJ, Bonow RO, Dearani JA, Fifer MA, Link MS, et al. 2011 ACCF/ AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy: Executive summary: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation, 2011; 124:2761-2796.
5. Maron BJ. Commentary and re-appraisal: Surgical septal myectomy vs. alcohol ablation: After a decade of controversy and mismatch between clinical practice and guidelines. Prog. Cardiovasc. Dis., 2012; 54:523-528. https://doi.org/10.1016/j.pcad.2012.04.008. PMid:22687595.
6. Oliver WC, de Castro MA, Strickland RA. Uncommon Diseases and Cardiac Anaesthesia. Kaplan JA, Editor. Fourth edition. Cardiac Anaesthesia. Philadelphia: Saunders; 1999. p. 909-915.
7. Sherrid MV, Chaudhry FA, Swistel DG. Obstructive hypertrophic cardiomyopathy: Echocardiography, pathophysiology, and the continuing evolution of surgery for obstruction. The Annals of Thoracic Surgery, 2003; 75:620-632. https://doi.org/10.1016/S0003-4975(02)04546-0.
8. Marwick TH, Stewart WJ, Lever HM, Lytle BW, Rosenkranz ER, Duffy CI, et al. Benefits of

intraoperative echocardiography in the surgical management of hypertrophic cardiomyopathy. J. Am. Coll. Cardiol., 1992; 20:1066-72911011. https://doi.org/10.1016/0735-1097(92)90359-U.

9. Davies MR, Cousins J. Cardiomyopathy and anaesthesia. Continuing Education in Anaesthesia, Critical Care and Pain, 2009; 9(6). https://doi.org/10.1093/bjaceaccp/mkp032.