Anaesthetic Management Of A Case Of Hypertrophic Obstructive Cardiomyopathy For Non Cardiac Surgery
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Citation

Abstract
Hypertrophic obstructive cardiomyopathy (HOCM) or asymmetrical septal hypertrophy (ASH) is a challenge to the anesthesiologist due to the complex pathophysiology involved and various intraoperative complications associated with it. Here we report successful anaesthesia management of a case of HOCM posted for functional endoscopic sinus surgery (FESS).

INTRODUCTION
Hypertrophic obstructive cardiomyopathy (HOCM) has an incidence of 1 in 500, is an autosomal dominant lesion, and is the most common genetic cardiac disease\(^3\). Intraoperative anaesthetic management includes understanding of the complex pathophysiology involved, maintaining desired hemodynamic parameters, and managing specific complications like congestive heart failure, myocardial infarction, dysrhythmias and hypotension\(^8\)\(^,\)\(^9\)\(^,\)\(^10\)\(^,\)\(^11\)\(^,\)\(^12\) associated with this condition. We report successful conductance of anaesthesia in a case of HOCM posted for Functional Endoscopic Sinus Surgery (FESS) in view of CSF rhinorrhoea.

CASE REPORT
A fifty year old female presented with complaint of CSF rhinorrhoea since 1 year. She was posted for functional endoscopic sinus surgery (FESS). Past medical history revealed hypertension and dyspnea on exertion grade I since 10 years. She was taking tab. Atenolol 5 mg bd, Tab. Sorbitrate 5 mg bd, Tab. Acetzolamide 50 mg bd. Clinically, airway was MPC I and cardiovascular and respiratory examination was within normal limits. All routine investigations including serum electrolytes were normal. CXR showed cardiomegaly with clear lung fields. During thorough cardiac work up, baseline findings were: HR-72 bpm regular, Blood pressure-128/76 mmHg, ECG-severe left ventricular hypertrophy and T wave inversion in lead I, II, aVL, V\(_2\)-V\(_6\) 2-D Echo- basal LA size increased, LV size normal, asymmetrical septal hypertrophy (ASH) and sigmoid shaped septum, systolic anterior motion of mitral valve(SAM), LVOT gradient at rest-23, mild to moderate AR, no RWMA and LVEF being 50%. Dobutamine stress was performed. During peak of test findings were: ECG-sinus tachycardia and ST depression in V\(_1\)-V\(_6\) - Blood pressure 70/0mmHg. 2-D Echo- no new RWMA, obstruction of LV cavity in systole, LVOT gradient increased to 177 mmHg and LVEF-50%. Stress test was terminated due to severe heaviness in chest and marked increase in LVOT gradient, which determines the severity of obstruction. After the test all parameters returned nearly to same as base line values. Patient was asked to stop Tab. Aspirin 7 days prior to surgery and Tab. enalapril 72 hrs prior to surgery. After securing i.v. lines preinduction parameters were monitored with cardio scope, pulse oxymeter and NIBP. Right sided IJV was cannulated to monitor CVP. After premedicating with Inj. Midazolam 0.03 mg/kg and Inj. Fentanyl 2mcg/kg, Induction was done with Inj. Propofol 2mg/kg and Inj Vecuronium 0.1mg/kg/hr, Induction was done with Inj. Propofol 2mg/kg and Inj Vecuronium 0.1mg/kg/hr and patient intubated with 7.0 no. cuffed ETT. Intraoperative patient was maintained on N\(_2\)0:O\(_2\)::60:40, Inj. Vecuronium 0.1mg/kg/hr, Inj midazolam 0.05 mg/kg/hr and Inj. Fentanyl 2 mcg/kg/hr. Anaesthesia was reversed with Inj. Glycopyrrolate 8 mcg/kg and Inj. Neostigmine 0.05 mg/kg and patient was extubated. After 24 hrs of uneventful observation patient was discharged from anaesthesia care unit.

DISCUSSION
Hypertrophic obstructive cardiomyopathy, or HOCM or asymmetrical septal hypertrophy (ASH), is a genetically determined disease characterized by histologically abnormal myocytes and myocardial hypertrophy developing in the absence of pressure or volume overload\(^1\). More than 140 mutations on nine different genes coding for sarcomere
proteins and two genes coding for cardiac mitochondria have been associated with it. Obstruction in HOCM is dynamic outflow obstruction, because the degree of obstruction is variable and is dependent on the amount of blood in the ventricle immediately before ventricle systole. Dynamic outflow obstruction is usually due to systolic anterior motion (SAM) of the anterior leaflet of the mitral valve leading to its apposition with hypertrophied septum. Systolic anterior motion of the mitral valve (SAM) was initially thought to be due to the septal sub aortic bulge, narrowing the outflow tract, causing high velocity flow and a Venturi effect. Recent echocardiographic evidence indicates that drag, the pushing force of flow and not ventricle effect is the dominant hydrodynamic force on the mitral leaflets. The symptoms of HCM include dyspnea, chest pain, palpitation, dizziness, fainting and sudden cardiac death. Medical therapy with beta-blockade, calcium channel blockade and diuretics may improve symptoms. Surgical interventions include surgical septal myectomy, alcohol septal ablation or dual chamber cardiac pacing.

Anaesthetic implications of management, of case of HOCM for noncardiac surgery include intensive cardiovascular monitoring and managing intraoperative complications. The dynamic obstruction in HOCM depends on the timing and duration of hypertrophied septal-mitral leaflet contact. Severity of obstruction is accentuated by any intervention that reduces ventricular size, and hence, increases the septal-leaflet contact. Therefore, hemodynamic goals include maintaining adequate preload and afterload and preventing excessive increase in contractility and tachycardia. Review of literature reveals various Intraoperative complications in patients of HOCM for non-cardiac surgery like congestive heart failure, myocardial infarction, dysrhythmias and hypotension. A case report states intraoperative pulmonary edema in a parturient during caesarean section in a patient of HOCM. In one study increased risk of cardiac complications has been associated with major surgery and increased surgical time. Sinus rhythm is crucial in these patients because of the dependence of preload on atrial contraction. So, atrial pacing may be required in these patients, in case junctional rhythm develops.

In our patient, smooth induction and intubation was done using propofol and vecuronium. Propofol, is generally avoided in cardiac induction due to its cardiodepressent effect, however in HOCM we need mild cardiac depression, so propofol can be useful. Laryngeal mask airway has been used in patients of HOCM, however FESS being a nasal procedure with risk of post nasal bleed we did endotracheal intubation with throat packing. During peak of dobutamine stress test, done preoperatively, in this patient, blood pressure dropped from 128/76 mmHg to 70/0 mmHg and LVOT gradient increased from basal 23 mm Hg to 177 mmHg. So we avoided stress response during intubation with Inj. Esmolol 0.5 mg/kg and maintained heart rate and blood pressure to near preinduction values. Pulmonary artery catheter is the ideal guide to determine fluid intake in these patients as CVP in these patients does not determine true volume status of LV. Considering FESS being a short procedure without much hemodynamic shifts and the complications being associated with pulmonary artery catheter, we put IJV and maintained CVP at 8-12 cm H2O cm H2O, thus maintaining adequate preload. Blood pressure was maintained at around 120-130/70-80 mmHg keeping after load in high-normal range. Midazolam-Fentanyl infusion provided intraoperative cardiac stability. Intraoperative ECG lead II showed ST depression and peaked T waves, but sinus rhythm was maintained throughout. We, however kept cardiac drugs, defibrillator and temporary pacemaker standby for any emergency. Intraoperative course, extubation and post operative observation were uneventful.

We conclude that management of HOCM includes thorough understanding of the pathophysiology of condition and maintaining specific hemodynamic goals to prevent the intraoperative complications associated with it.

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