# Anaesthethic Management of a Case of HOCM Posted for Rt. # IT Femur (DHS Plating)

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***Case Report***

***Abstract:*** Hypertrophic Cardiomyopathy is most common genetic cardiovascular disease with a prevalence of 1:500 and is transmitted as autosomal dominant condition with variable penetrance (1, 2). It can affect patients of all ages. The disease is characterised by LV hypertrophy most commonly septal and anterolateral free wall hypertrophy. Surgery poses a very high risk for these patients because of several complications (1, 2) that can occur in the perioperative and post operative period. We present a case of 67yr old male a diagnosed case of HOCM posted for #Rt. IT Femur(DHS plating) under Epidural anaesthesia.

***Keywords***: Hypertrophic cardiomyopathy, epidural anaesthesia, etc.

## Introduction

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant condition. It is an important cause of sudden death in young adults ,affects significant number of population who are unaware that they have the condition .The progression of an individual's disease cannot be predicted, with many patients having a normal life span being relatively unaffected. Diagnosis is based upon unexplained left ventricular hypertrophy which may be concentric or asymmetrical, diffuse or focal.

Pathophysiology of HCM is related to following issues:

A:Myocardial hypertrophy

B:Dynamic Left Ventricular outflow tract obstruction

C:systolic anterior movement of mitral valve and mitral regurgitation

D:Diastolic dysfunction

E:Myocardial ischemia and dysrhythmias

Hypertrophied myocardium exhibits prolonged relaxation time and decreased compliance leading to increased left ventricular end diastolic pressure and impaired filling.

The left ventricular outflow tract obstruction increases due to increased myocardial contractility, decrease in preload (hypovolemia, vasodilators, tachycardia, positive pressure ventilation), decrease in afterload (hypotension, vasodilators). (3)

Dysrhythmias are the cause of sudden death in young adults with HCM. (1, 2)

These risks are increased several fold in perioperative period and any surgical intervention in these patients should be undertaken only after careful contemplation and weighing the balance between risks and benefits of the surgery. (2)

## Case Summary

A 67yr old male diagnosed case of HOCM came with history of fall referred from a peripheral hospital with # Rt. IT femur. Patient was accidently diagnosed on echocardiography as having HOCM one year back when he had an episode of retention of urine associated with nausea and vomiting and was admitted for the same. Patient was asymptomatic with no history of dyspnoea on exertion, chest pain, fatigue, palpitations and syncope.

There was no history of admission in hospital for heart failure, tachyarrhythmias in the past. No family history suggestive of sudden cardiac death. Patient was not on any medication and there was no past surgical history.

On examination patient was conscious and oriented,afebrile, pale with pulse rate 84/min, blood pressure 160/90mmhg with no rise in jvp/pedal edema/clubbing or cyanosis. On auscultation chest was clear and s1 s2 were normal with no murmur

Laboratory investigations were as follows:

Hb: 8.7g/dl TLC: 7200/cumm PLT:281000/cumm

Bld Grp: A+ve

BT: 2min 10sec CT: 3min 30 sec

Sr.creatinine: 1.70

Sr.urea: 29

Total bilirubin: 0.43 (direct-0.12)

SGOT:28 SGPT:26 Alkaline phosphatise: 88

Total proteins: 7.3 (albumin- 3.5)

Urine: Albumin-nil

Sugar-nil

2D Echo: Hypertrophic Obstructive Cardiomyopathy with ASH

Basal IVS akinetic, No other RWMA

Good LV/RV systolic function(LVEF-69%)

Mild LV Diastolic dysfunction

Patient was newly diagnosed as hypertensive and started on metpure XL 50mg BD and tab Dytor 10mg BD. After 3 days of optimisation patient was posted for surgery i.e Rt. DHS plating of femur. On day of surgery,Patient was

haemodynamically stable with heart rate of 58/min regular, normovolumic and Blood pressure of 156/74mmhg with tab. Metpure XL 50mg and Dytor 10mg continued on day of surgery. Fasting status was confirmed, high risk written informed consent was taken.
We preferred regional (epidural) anaesthesia for the patient.
Patient was taken in operation theatre and electrocardiogram, pulse oximeter, non invasive blood pressure were attached. Continuous ECG monitoring was done on an external defibrillator. Patient received i.v Ranitidine 50mg and i.v metoclopramide 10mg before shifting in the operation theatre.
With 20G i.v line in situ(Rt. Forearm) 1Ө RL started and inj.tranexamic acid 500mg in drip started.
Under all aseptic precautions; Pt. in sitting position, Epidural catheter (18 G) inserted at L2-L3 interspinal space and fixed at 8cm.
Then inj. Ropivacaine(0.5%) 10ml + inj. Lignocaine(2%) 4ml + 25µg Fentanyl given through epidural catheter in titrated doses each time confirming negative aspiration for blood and CSF.
Adequate level of anaesthesia was achieved after 15minutes.

**INTRA-OPT**

| Time | Pulse | BP | SPO2 | Urine Output | I.V fluid | Drugs |
|---|---|---|---|---|---|---|
| 1.45pm | 54 | 136/70 | 99% | 25ml | 1ӨRL | Inj.Emset 4mg i.v |
| 2.00pm | 52 | 140/72 | 100% | ------ | ↓ | --------- |
| 2.15pm | 52 | 138/70 | 100% | 50ml | 2ӨRL | --------- |
| 2.30pm | 54 | 138/76 | 100% | 80ml | ↓ | --------- |
| 2.45pm | 52 | 144/80 | 100% | 110ml | ↓ | --------- |
| 3.00pm | 52 | 142/76 | 99% | | 3ӨRL | --------- |

Procedure was uneventful.
Following surgery patient was shifted to surgical intensive care unit for monitoring and further management. In ICU patient was judiciously monitored for haemodynamic parameters (pulse, Blood pressure and Spo2). Post operative pain was managed by giving epidural top up at interval of 8hrs. (inj.Tramadol 50mg + 9cc Normal saline given through epidural catheter confirming negative aspiration for blood). Metpure XL and Dytor were continued post operatively and i.v fluid was given as maintenance of 2ml/kg/hr to cover 6hrs of NBM period.
Epidural catheter was removed two days after surgery and patient was shifted toward ($3^{rd}$ post operative day). Patient got discharged on post operative day $15^{th}$ after receiving daily physiotherapy.

## Discussion

HOCM is an autosomal dominant disease characterised by asymmetrical septal hypertrophy with dynamic left ventricular outflow tract obstruction due to systolic anterior motion of mitral valve. There is impaired diastolic filling and is mainly dependent on atrial contraction. Decreased compliance of left ventricle leads to increase in left ventricular end diastolic pressure which may lead to pulmonary edema on further increase and cause impaired coronary perfusion leading to myocardial ischaemia. Dysrhythmias are common cause of sudden death in young patients with HOCM.(1, 2)
This poses a major threat to the patient requiring surgical intervention. The risks and benefits of surgery must be weighed carefully prior to making decision to perform surgical procedure in these patients. In order to avoid serious complications, adequate optimization is essential before proceeding with any elective surgery. The advice of an experienced cardiologist should be sought.
Most patients are usually asymptomatic throughout life just like in our case but few may present with symptoms of severe heart failure and often sudden death. Principle symptoms of HCM are angina pectoris,fatigue or syncope, tachyarrhythmias and heart failure (1). Cardiac physical examination may reveal double apical impulse, gallop rhythm and cardiac murmurs and thrills. The intensity of these murmurs change with certain maneuvers eg. Valsalva maneuver , standing, nitroglycerin increases loudness of these murmurs.
Medical therapy is used for patients with symptoms of heart failure and includes β-blockade combined with or without disopyramide or verapamil. These should be continued in the perioperative period as they are thought to improve symptoms by reducing heart rate and by exerting a negative inotropic effect leading to improved coronary perfusion pressures. Diuretics administration should be done cautiously.
Management of anesthesia: Any drug or event that increases myocardial contractility or increases preload and afterload reduces LVOT obstruction. Conversely, sympathetic stimulation, vasodilation and hypovolemia worsen LVOT obstruction.(1,2)
Diagnosed cases of HOCM (like in our case) must be preoperatively evaluated and should include a 12 lead ECG and echocardiographic examination. Beta blockers or calcium channel blockers continued throughout the perioperative period. Those with an ICD should have the unit turned off in the immediate pre operative period and have an external defibrillator immediately available in the operating room.
Though many studies advocated general anaesthesia for cases of HOCM many studies are there were surgeries were conducted successfully under Epidural anaesthesia (4, 5, 6). We have chosen regional over general

anaesthesia in our case due to several disadvantages posed by general anaesthesia.

A: Sudden decrease in systemic vascular resistance due to i.v induction agent.

B: Activation of sympathetic nervous system during intubation and extubation.

C: Positive pressure ventilation leading to decrease in preload in hypovolemic patients.

D: Need of invasive blood pressure monitoring and Transesophageal echocardiography. Neither central venous pressure monitoring nor pulmonary artery pressure monitoring can diagnose LVOT obstruction.

E: Post operative pain management to prevent sympathetic nervous system activity and avoid hypoxia and hypercarbia.

Central neuroaxial blockade has the disadvantage of sudden decrease in preload leading to hypotension and tachycardia which leads to increase in LVOT obstruction and decreased ventricular filling.(5)

But considering the fact we have chosen epidural anesthesia and achieved the desired level by injecting drugs in titrated doses taking care to avoid sudden decrease in preload. We kept phenylephrine (α – adrenergic agonist) ready to deal with decrease in preload and afterload (5,8). Prompt replacement of blood loss and titration of intravenous fluids is important for maintaining preload and blood pressure. However, because of diastolic dysfunction aggressive fliud therapy may result in pulmonary oedema (7). We have kept esmolol and metoprolol (8) ready to slow persistently elevated heart rates. Defebrillator /cardiovertor was kept attached to the patient. We shifted the patient to post operative room giving inj.tramadol 50mg+9ml normal saline through epidural catheter to reduce post operative pain which may trigger sympathetic nervous system and increase LVOT obstruction. Hypothermia and shivering prevented by giving warm blanket and keeping radiant warmer near the patient. Post operative pain managed effectively by giving 8hrly epidural top ups of inj. tramadol 50mg +9ml normal saline for two days post operatively. Patient was monitored judiciously in surgical ICU for maintenance of euvolemia.

## Conclusion

In the patient with known HCM, the overall aim is to prevent or treat LVOT obstruction, arrhythmia, and ischaemia should they occur.

The strategy involves:

- maintenance of sinus rhythm;
- reduction in sympathetic activity to reduce chronotropy and inotropy;
- maintenance of left ventricular filling;
- the maintenance of systemic vascular resistance.

Any surgical intervention and induction of anaesthesia may lead to intraoperative hypotension, myocardial ischaemia, acute heart failure and supraventricular or ventricular tachydysrhythmias. So surgery should be undertaken only after preoperative optimization and calculation of risk benefit ratio. Effective co-ordination between surgeon, anesthetist and cardiologist is essential for better outcome of any surgical intervention in these patients.

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