Case Report

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Pseudopancreatic cyst in a patient with hypertrophic cardiomyopathy

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ABSTRACT

Hypertrophic cardiomyopathy (HCM) is a rare genetic disorder characterised by massive myocardial hypertrophy leading to left ventricular outflow tract obstruction. Clinical presentation ranges from absence of symptoms to sudden death. A 17 yr old male patient, known case of hypertrophic cardiomyopathy was posted for excision of pseudopancreatic cyst. Anaesthetic management of these patients poses considerable challenges. Factors like tachycardia, hypovolemia, vasodilatation and increased cardiac contractility lead to exacerbation of the obstruction.

Key words: Anaesthesia, hypertrophic cardiomyopathy, pseudo-pancreatic cyst

INTRODUCTION

Hypertrophic Cardiomyopathy is a rare genetic disorder (autosomal dominant) characterised by massive asymmetric myocardial hypertrophy usually involving interventricular septum leading to Left Ventricular Outflow Tract obstruction (LVOT) and diastolic dysfunction^{1,2}. It is the most common of genetic cardiovascular disease caused by mutation in genes encoding proteins of cardiac sarcomere³.Prevalence of HCM is 1:500 individuals. It is the most common cause of Sudden Cardiac Death (SCD) in less than 40 years age group & in competitive athletes. Clinical presentation ranges from absence of symptoms to sudden unexpected death. Decrease in venous return and systemic vascular resistance or increase in myocardial contractility worsens the LVOT obstruction. These patients are highly prone to arrhythmias like atrial fibrillation and ventricular tachycardia⁵. Management of anaesthesia in these patients poses considerable challenges for the anaesthesiologist. We report successful conduct of anaesthesia in a patient with HCM undergoing pseudopancreatic cyst excision.

CASE REPORT

A 17 yrs young male patient weighing 49kg, a known case of hypertrophic cardiomyopathy was scheduled for pseudopancreatic cyst excision. He had complaint of chest pain & breathlessness on exertion since 8 yrs. He was on Metoprolol 50 mg OD orally since 5yrs. The routine laboratory investigations were within normal limits. His

electrocardiogram showed ST-T changes with signs of LVH with LV strain & Lt atrial overload with Rt atrial overload. His 2-D echocardiogram showed no left ventricle outflow tract obstruction & his ejection raction was 67%. Systolic anterior motion (SAM) of mitral valve leaflet was present, but without evidence of mitral regurgitation.

We planned to conduct surgery under General Anaesthesia combined with epidural block. Standard monitors including SpO2, NIBP & ECG were attached. An intravenous line was secured with18 G cannula. Preloading was done with 500 ml Ringer lactate. The patient was premedicated with Inj Midazolam 1mg iv. A 18 G Epidural catheter was inserted via 18 G Tuohy's epidural needle at T11 - T12 interspace via median approach in sitting position under full aseptic precautions. 12 ml of 0.125% Bupivacaine was given as loading dose. After positioning patient supine, preoxygenation was done with 100% O2 for 5 min. Inj Fentanyl 100 mcg was given iv as analgesic. InjEsmolol 25mg was given 3min before intubation. Induction was done using InjPropofol, given slowly in titrated doses. After bag and mask ventilation, endotracheal intubation was done with ETT 8.5 mm facilitated by InjVecuronium

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4mg. After induction a 7 French Swansheath was inserted in right internal jugular vein for monitoring of Central Venous Pressure (CVP) and arterial catheter was placed in the right radial artery for invasive blood pressure monitoring. Anaesthesia was maintained with 50:50 oxygen &nitrous oxide with Sevoflurane 1%-2% (intermittent)+ InjVecuronium 6mg in divided doses. Surgery was started.

Patient had an episode of hypotension (90/40 mmHg) intraoperatively. It was managed with administration of 200 ml of colloid (Hestar 6%). The hemodynamic parameters remained stable with heart rate from 60 to 76/min & BP from 90/60 to 126/84 mmHg. Duration of surgery was 2hrs. At the end of surgery, neuromuscular blockade was reversed with Inj Neostigmine 2.5 mg + InjGlycopyrrolate 0.5mg. Before shifting patient to Surgical Intensive Care Unit, epidural supplement was repeated with InjBupreonorphine 100 mcg diluted upto 10 ml for postoperative pain relief & thereafter 8 hourly for 3 days.

DISCUSSION

Hypertrophic Cardiomyopathy pathophysiology involves four inter-related processes including left ventricular outflow tract obstruction, diastolic dysfunction, myocardial ischemia& systolic anterior motion of anterior mitral valve leaflet against hypertrophied septum. Systolic anterior motion of the mitral valve leads to LVOT obstruction and often precipitates mitralregurgitation^{5,6}. Factors exacerbating obstruction are tachycardia, hypovolemia, vasodilation and increased cardiac contractility7. Anaesthetic management entails maintenance of desired hemodynamic parameters & management of specific complications like hypotension, dysrhythmias (AF) & CHF.⁸ Preloading before induction helps in maintaining troke volume & minimize adverse events of positive pressure ventilation. Premedication with midazolam alleviates anxiety and blunts sympathetic stimulation. Administration of beta blockers suppresses sympathetic stimulation & hence decreases degree of outflow tract obstruction. Our patient was on beta blocker which was continued perioperatively, while intra operatively the dose dependent myocardial depression caused by inhalation anaesthetics might have helped in this regards. Episodes of intraoperative hypotension are treated with fluids & phenylephrine. Phenylephrine is preferred over ephedrine & dopamine as it increases SVR without significant effect on myocardial contractility & heart rate. Our patient responded well to fluids. We used IV fluids judiciously and kept the CVP at 10cm of H2O as adequate reload is necessary to maintain optimal cardiac output and avoid undue increase in contractility because of hypovolemia. Maintenance of sinus rhythm is important due to dependence of preload on atrial contraction. Defibrillators should be kept ready always. IPPV was done using small tidal volume at rapid frequency in order to minimize reduction in venous return. In this patient, smooth induction was done using propofol. Propofol, is generally avoided for induction due to its cardiodepressant effect, however in HCM we need mild cardiac depression. so propofol can be used. We used it in titrated doses to avoid precipitous hypotension. Vecuronium as muscle relaxant was used as it is a cardio-stable drug and thus avoids LVOT obstuction. Epidural catheter was put in this patient for reducing intraoperative analgesic requirements and for postoperative analgesia.

CONCLUSION

Hypertrophic Cardiomyopathyis a rare disorder imposing challenging conditions for the anaesthesiologist. A thorough understanding of the pathophysiology of the disease, good pre-operative preparation, and meticulous intra-operative management along with vigilant postoperative care is necessary for safe outcome.

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