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ORTHOTOPIC LIVER TRANSPLANT IN A PATIENT WITH HYPERTROPHIC CARDIOMYOPATHY

Anaesthesiology

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ABSTRACT

Live Donor Liver Transplantation (LDLT), in patients with end stage liver disease is a condition associated with a high cardiac output, low systemic vascular resistance, coagulopathy and a potential for significant blood loss. The main feature of HOCM is left ventricular outflow tract obstruction which is aggravated by increased heart rate, decreased LV preload, decreased afterload, increased LV contractility, all conditions common during orthotopic liver transplantation (OLT). Minimum data is available regarding the number and outcome of patients with LVOT obstruction present either at rest or dynamic in nature who have undergone OLT. We present a case of chronic liver disease with HOCM who successfully underwent OLT. Our anaesthetic goals included maintenance of strict euvolemia, avoidance of tachycardia, avoidance of inotropes, use of vasopressors for hypotension. Intraoperative TEE was utilised to detect early signs of LVOT obstruction and provide rationale guide for fluid and hemodynamic management.

KEYWORDS

Live Donor Related Liver Transplantation, Hypertrophic cardiomyopathy, Left ventricular outflow tract obstruction, TEE, Phenylephrine.

INTRODUCTION

Hypertrophic Obstructive Cardiomyopathy (HOCM) is a heterogenous disorder, affecting patients of all ages. It has a diverse clinical course varying from being asymptomatic to symptoms of heart failure, angina or syncope and a minority may present with sudden cardiac death. Features of HOCM are hypercontractile LV, diastolic dysfunction, increased Ejection Fraction, SAM (systolic anterior motion of mitral leaflet), with or without MR. Left ventricular outflow tract (LVOT) obstruction is dynamic, aggravated by hypovolemia, positive inotropy and vasodilatation[2]. Patients with end stage liver disease manifest a hyperdynamic circulation characterized by a decrease in systemic vascular resistance, decreased arterial pressures, increase in heart rate and cardiac output which can aggravate the dynamic left ventricular outflow tract obstruction[3]. This is worsened during liver transplantation due to coagulopathy, potential for massive haemorrhage and hypovolemia, need for inotropes at the time of reperfusion. All these factors exacerbate hemodynamic disturbances of HOCM.

CASE REPORT

We present here 50 year old male with alcoholic liver disease for live donor related liver transplantation. Pt had renal dysfunction, serum creatinine 1.8mg/dl, creatinine clearance of 30ml/min/1.73m^2, with normal electrolytes, coagulopathy with INR 1.9. Preoperative Transthoracic Echo showed normal ventricular function, moderate concentric LVH, SAM with EF65%, LVOT resting gradient of 30 mm Hg and mild MR.

Our anaesthetic goals included maintenance of adequate preload, afterload, absolute avoidance of tachycardia with use of beta blockers as needed, avoidance of inotropic agents and use of pure vasopressors like phenylephrine.

In OT, after placement of routine preinduction monitors ECG, PULSE OXIMETRY, NIBP, 16G iv cannula was secured. Radial artery was cannulated with 20G arterial cannula. RSI was done with iv injection Fentanyl 3mcg/kg, iv injection Propofol 60mg, iv injection Succinylcholine 100mg. Trachea was intubated with 8.5 mm cuffed ETT. Right IJV was cannulated with 9F 3L catheter (AVA advanced venous access) and Pulmonary arterial catheter was inserted. Continuous cardiac output monitoring was done using Flo-Trac/Vigileo device which uses arterial pressure waveform analysis to calculate the derived variables of Stroke Volume Variation (SVV), Systemic vascular resistance (SVR) and Cardiac Index(CI). Vitals remained stable post induction of Anaesthesia. Additional monitoring of Etco2, temperature, urine output, ABG and TEG was done. A Transesophageal Echocardiography (TEE) probe was placed for monitoring of pressure gradient across LVOT in order to detect increased SAM and for guidance of fluid and pressor therapy. TEE performed prior to incision confirmed normal ventricular function, moderate LVH with prominent septum. No signs of LVOT obstruction were detected.

The aim of anaesthesia was to maintain adequate preload, afterload, intravascular volume and to avoid inotropy and chronotropy (tachycardia). Anaesthesia was maintained using isoflurane at a MAC of 0.8-0.9, fentanyl at 150 mcg/hr and atracurium at 30 mg/hr. All emergency drugs, vasopressors were kept ready for use as and when needed.

The prehepatic phase demonstrated a low SVR with an increased cardiac output. During the dissection 2l blood loss was encountered and replaced with iv fluids and 2 units PRBC. Haemodynamic variables at various stages are shown in table 2.TEE showed mild-moderate SAM, moderate MR, LVOT gradient of 18. Patient was haemodynamically stable, use of vasopressors was not required.

The anhepatic period(time from the physical removal of the liver from recipient to recirculation of graft) lasted for 3hours and 45 minutes, the patient lost around 3.5 L of blood. Volume replacement with 4 units of packed cells, 2 units of fresh frozen plasma was done. Portocaval shunt was performed to reduce mesenteric pooling. The total duration of the clamp was 40 minutes which was associated with a drop in cardiac output and decrease in SVRI to 500 dyne sec m^2 cm^{-5}. This was managed with fluid boluses of colloid gelofusin and intermittent boluses of phenylephrine.

Prior to reperfusion of the allograft, phenylephrine infusion was started to avoid systemic hypotension related to PRIS (post reperfusion syndrome). LVOT was continuously monitored by TEE for the evidence of dynamic obstruction.

Immediately following reperfusion there was decrease in cardiac index to 5.8 (litre min^{-1} m^{-2}), drop in SVRI to 400 dyne sec m^2 cm^{-5} and increased stroke volume variation. There was modest increase in dynamic LVOT obstruction. HR increased to 120/min, BP dropped from 160/90 to 100/50 mmHg, with concomitant decrease in left ventricular EDV. It was treated with iv fluid bolus, transfusion of colloid Haemaccel, 1 PRBC.

Phenylephrine infusion rate was increased which resulted in increase in Afterload and decrease in SAM and pressure gradient across the mitral valve. The rate of phenylephrine infusion varied from 50mcg/min in anhepatic period to 200 mcg/min immediately post reperfusion. The final TEE evaluation of LVOT showed no evidence of dynamic obstruction or SAM. The further intraoperative period was uneventful and the patient was electively ventilated and shifted to the transplant ICU.

Post operatively the Patient was electively ventilated overnight extubated next morning when all hemodynamic parameters were normal, ABG acceptable and adequate UO. He was started on beta blockers to maintain a target HR of 60-70 bpm.

DISCUSSION

To our knowledge three case reports of patients with Hypertrophic obstructive cardiomyopathy (HOCM) for OLT have been described[10]. As such there is scant information regarding preoperative evaluation or anaesthetic management of such patients with severe dynamic LVOT obstruction presenting for Orthotopic liver transplant (OLT). Till date no guidelines have been described regarding the severity of LVOT obstruction that can pose significant risk for OLT.

HOCM is a subset of HCM in which hypertrophy causes LVOT obstruction with an autosomal dominant inheritance.[1] It is diagnosed principally with transthoracic ECHO showing LVH, wall thickness of atleast15mm in adults, which is typically asymmetric in the absence of cardiac conditions[11].

The features of HOCM are a hypercontractile left ventricle, increased ejection fraction, diastolic dysfunction and SAM. The obstruction is dynamic as in it depends on preload, after load, sympathetic stimulation and posture. The symptoms of HOCM range from none to chest pain, palpitations, syncope and even sudden cardiac death. Signs include a jerky pulse, 4th heart sound and a mid systolic murmur exacerbated by Valsalva. Treatment options include administration of beta blockers, calcium channel blockers, placement of ICD, alcohol septal ablation[1] and surgical septal myomectomy[14].

Patients with end stage liver disease (ESLD) usually have a hyperdynamic circulation characterised by increased CO, decreased SVR. In addition to this OLT is associated with changes in left ventricular preload secondary to intra operative bleeding and inferior venae cava obstruction (during prehepatic and anhepatic periods) and further decrease in SVR post reperfusion of the donor liver.[4] The use of inotropes with beta1 activity like epinephrine can worsen LVOT obstruction and further increase the gradient. It is believed that a patient with HOCM with LVOT obstruction has multiple risk factors for the development of dynamic LVOT obstruction.[15,16]

Anaesthetic goals include maintenance of preload, after load, euvolemia, avoid marked increase in ventricular contractility. Avoiding decrease in afterload is very crucial to prevent reflux tachycardia and subsequent increase in contractility. Increase in SVR can be achieved with pure alpha agonist Phenylephrine infusion. Reduction in HR and contractility can be achieved with user of short acting selective beta adrenergic blocker(Esmolol). Epinephrine should be avoided as it causes tachycardia and increased contractility which worsens LVOT obstruction.

We did a predominantly fentanyl based induction to obtund tachycardia, maintained with a low MAC of isoflurane, fentanyl at 2-3 mcg/kg/hr and atracurium at 0.25-0.30 mcg/kg/hr. Phenylephrine infusion was used to increase SVR. Inotropes were completely avoided. A portocaval shunt was created early in the anhepatic period to reduce mesenteric pooling and optimize preload. TEE has better correlation with ventricular filling than PCWP[8,12].It helped in detecting early signs of dynamic LVOT obstruction and provided rational guide for fluid and haemodynamic management during reperfusion.[7,8,15] TEE aided management of LDLT in two patients with HOCM has been reported.[9,13]

It is debatable whether the presence of dynamic LVOT obstruction is contraindication for undergoing OLT. Each patient should be carefully evaluated with intraoperative anaesthetic management guided by TEE.[15]

CONCLUSION

In conclusion, we believe patients diagnosed with LVOT obstruction should be carefully managed with intraoperative TEE. Maintaining haemodynamic goals can lead to an acceptable outcome. We advocate the use of TEE as an intraoperative monitor of dynamic obstruction, appropriate therapy can be initiated based on real time observation.

Table 1: Showing Preoperative Blood Investigations

Haemoglobin	8.2 gm/dl
Total Leucocyte count	8600 cells/mm3
Platelet Count	1,45,000 cells/mm3
INR	1.9
Serum Urea	69 mg/dl
Serum Creatinine	1.8 mg/dl
Sodium	133 mEq/L
Potassium	4.3 mEq/L
Magnesium	1.4 mEq/L
Total Bilirubin	1.3 mg/dl
SGOT	36
SGPT	44
ALP	102
Albumin	2.8

Table 2: Intraoperative Haemodynamic Variables.

Stage	HR	Sys BP	Dia BP	CI	SVRI	SVV	PCWP
Baseline	90	142	64	8.0	550	7	14
X-30	115	110	60	7.4	500	20	12
X+5	120	100	50	6.8	420	24	16
X+60	110	124	65	8.0	490	14	16
X+120	98	122	60	7.8	520	12	16

X-30 = 30 minutes prior to reperfusion, x+5 = 5 minutes after reperfusion, ‡x+60 = 1 hour after reperfusion, § x+120 = 2 hours after reperfusion, CI= cardiac index, SVRI = Systemic Vascular Resistance Index, SVV = Stroke Volume Variation, PCWP = Pulmonary Capillary Wedge Pressure

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