Case Report

Unexpected refractory intra-operative hypotension during non-cardiac surgery: Diagnosis and management guided by trans-oesophageal echocardiography

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ABSTRACT

We present a case of severe refractory hypotension in a patient undergoing de-bulking liver resection for massive polycystic liver disease. Emergent trans-oesophageal echocardiography (TOE) revealed dynamic left ventricular outflow tract (LVOT) obstruction with systolic anterior motion (SAM) of the anterior mitral leaflet (AML). Notably, he had a structurally normal heart on pre-operative trans-thoracic echocardiography (TTE). Diagnosis of SAM by TOE, possible mechanisms and specific management of refractory hypotension in this context are discussed.

Key words: Anaesthesia, hypotension, intraoperative monitoring, trans-oesophageal echocardiography

INTRODUCTION

Systolic anterior motion (SAM) of the mitral valve has been well described in hypertrophic obstructive Cardiomyopathy (HOCM) due to the enlarged ventricular septum coming into contact with one of the mitral valve leaflets, usually the anterior leaflet.[1] Refractory intraoperative hypotension with left ventricular outflow tract (LVOT) obstruction during general anaesthesia is well described in patients with hypertrophic obstructive cardiomyopathy (HOCM) who have concentric or asymmetric left ventricular hypertrophy. To our knowledge systolic anterior motion (SAM) in patients with structurally normal hearts is a rare occurrence and is not a well-known entity in non-cardiac anaesthesia setting. This case demonstrates the utility of trans-oesophageal echocardiography (TOE) in non-cardiac cases as a diagnostic tool and as a guide to therapy in managing refractory hypotension in the operating room setting and highlights the necessity to consider SAM of the mitral valve leaflets in the differential diagnosis of unexpected refractory intraoperative hypotension.

In this report we present a case of severe refractory hypotension in a patient with an anatomically normal heart on preoperative echocardiography undergoing non-cardiac surgery. Dynamic LVOT obstruction with SAM of the anterior mitral leaflet (AML) was diagnosed on TOE. Management of hypotension associated with SAM is unique and inappropriate management may even exacerbate hypotension. In cases of refractory intraoperative hypotension, TOE can be helpful in diagnosis and management of uncommon problems such as SAM.

CASE REPORT

A 48-year-old, dialysis dependent male (ASA physical

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status III, body weight 82 kg) with polycystic kidney and liver disease and post bilateral nephrectomies status presented with persistent massive ascites, mild pericardial and bilateral pleural effusions. His ascites and effusions were secondary to a combination of obstruction of hepatic venous outflow and compression of the right heart by cysts in the liver and chronic renal failure. He was scheduled for a debulking liver resection and deroofing of cysts to relieve compression. Other pertinent medical history included diabetes and hypertension. He was also on the waiting list for a combined liver and kidney transplant. His pre-transplant cardiac workup included a trans-thoracic echocardiography (TTE) that showed mild left ventricular dysfunction (EF 45-50%), normal right ventricular function, mild pericardial effusion, no LVOT gradient and no valvular or wall motion abnormalities. Nuclear myocardial perfusion scan demonstrated a small reversible septal defect consistent with myocardial ischemia and a follow-up coronary angiography showed normal coronary circulation.

General anaesthesia was induced after establishing standard ASA monitors and central venous pressure (CVP) was monitored from a pre-existing right internal jugular dialysis catheter. At baseline his blood pressure (BP) was 107/56 mmHg, heart rate (HR) 102 bpm, CVP 16. Intravenous bolus induction drugs include fentanyl 150 mcg, midazolam 2 mg, propofol 150 mg, and rocuronium 40 mg. Anaesthesia was maintained on O_2/N_2O (50:50), sevoflurane at 0.5-1 MAC and fentanyl boluses as required. His ventilation parameters included a tidal volume of 8 ml/kg, respiratory rate of 10/min and PEEP of 8 cm H_2O. A 20-gauge radial arterial line and a 14-gauge intravenous cannula were inserted in the left arm post induction. He became progressively hypotensive, prompting volume replacement, several bolus doses of phenylephrine (100-200 μg aliquots) and initiation of IV norepinephrine infusion. Hypotension and tachycardia worsened following drainage of a large ascites, despite aggressive volume replacement with colloid and crystalloid guided by trends in CVP (range 9-14 cm H_2O), systolic pressure variation (SPV) on the arterial line tracing (7-14 mmHg) and increasing norepinephrine infusion (0.02-0.15 mcg/kg/min). Given his continued hypotension, IV epinephrine infusion (0.02-0.07 mcg/kg/min) was initiated, which resulted in further deterioration of haemodynamics. (Please see Figure 1 for haemodynamic trends).

Emergent TOE demonstrated an under filled and hyper dynamic LV with SAM of the AML leading to functional LVOT obstruction and moderate mitral regurgitation (MR) [Figures 2 and 3]. Hypotension resolved after cessation of epinephrine, further volume replacement, increasing norepinephrine infusion (0.3 mcg/kg/min), addition of IV vasopressin infusion (0.04 units/min) and reduction in heart rate with beta blocker (10 mg of IV metoprolol in increments).

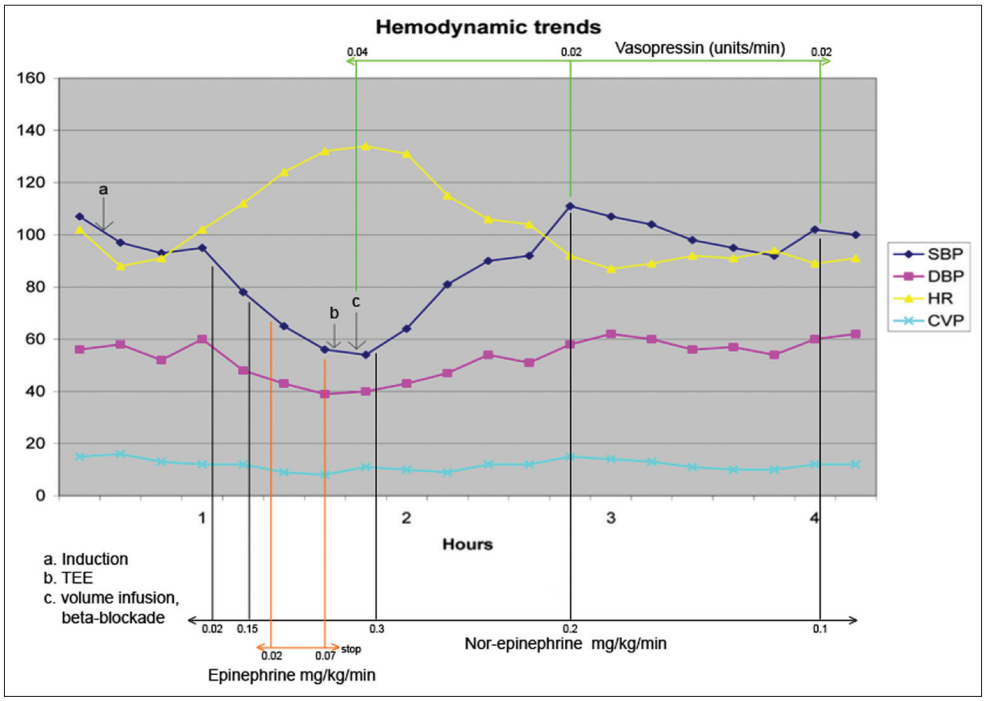

Figure 1: Hemodynamic trends

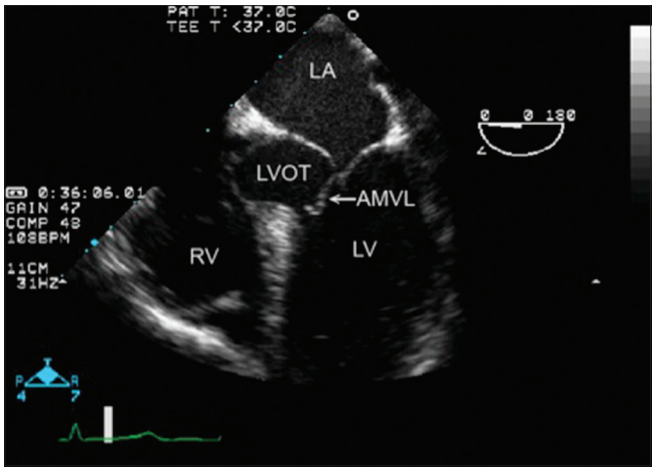

Figure 2: TOE: Mid-oesophageal short axis view demonstrating SAM. LA – Left atrium, LV – Left ventricle, AML – Anterior mitral leaflet, LVOT – Left ventricular outflow tract, RV – Right ventricle

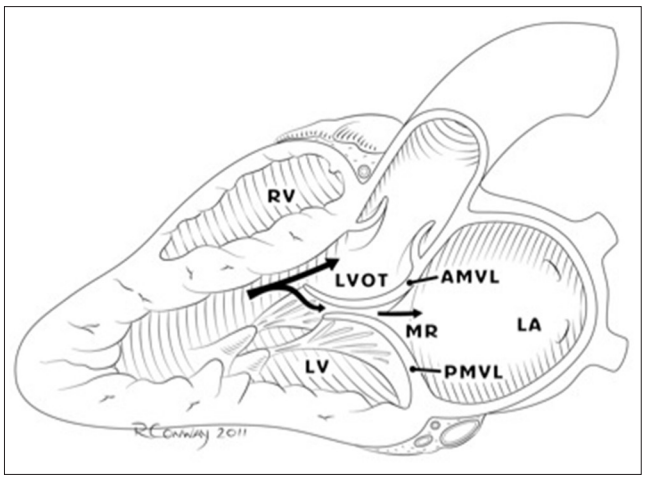

Figure 3: Illustration of systolic anterior motion (SAM) of mitral valve. RV – Right ventricle, LV – Left ventricle, LA – Left atrium, MR – Mitral regurgitation, LVOT – Left ventricular outflow tract, AML – Anterior mitral leaflet, PML – Posterior mitral leaflet

The procedure lasted 4.5 hours and was remarkable for a large blood loss of 1500 cc requiring blood and blood product transfusion. He was transferred to ICU postoperatively, extubated on day 3 and weaned off pressors on day 5. He was discharged from ICU on day 6 and from hospital on day 17. He returned back after 6 months to receive a combined liver and kidney transplantation successfully without any complications.

DISCUSSION

Hypotension is one of the most common complications during general and regional anaesthesia, but hypotension refractory to treatment is uncommon.

In this case we tried conventional management with volume replacement, vasopressors, and an inotrope without significant improvement in hypotension. Once we placed the TOE we found: (1) The LV was under filled despite what we thought was adequate preload based on CVP and SPV. (2) LV was hyper contractile with no wall motion abnormalities. (3) The patient had LVOT obstruction secondary to SAM of the AML.

In retrospect, the learning points from this case were as follows: (1) We should have reached for TOE sooner, before starting epinephrine as this intervention clearly worsened the hypotension. (2) CVP readings may have been abnormal due to right heart compression from surgical manipulation, pericardial effusion and hepatic cysts.

SAM of the mitral valve has been well described in HOCM due to the enlarged ventricular septum coming into contact with one of the mitral valve leaflets, usually the anterior leaflet.[1] However, SAM and dynamic outflow tract obstruction can even occur in patients without HOCM.[2-5] SAM was thought to occur as the result of venturi forces created by the rapid ejection of blood through the outflow tract that is narrowed by upper septal hypertrophy, drawing the mitral leaflet(s) anteriorly. However, more recent investigations indicate that drag, the pushing force of flow, is the dominant force that initiates SAM, pushing the protruding mitral leaflet into the septum.[6] Many factors have been shown to predispose patients to SAM including prior mitral valve surgery, ischemic heart disease that causes septal wall motion abnormalities, a small and hyper dynamic left ventricle, and congenital heart disease such as transposition of the great vessels.[6] In predisposed patients, aggravating factors include hypovolaemia, vasodilation from general or regional anaesthesia, and a hyper dynamic ventricle often secondary to increased catecholamine concentrations leading to a hyper contractile, under-filled ventricle (stress, catecholamine infusion, hyper dynamic circulation due to systemic inflammatory response or sepsis). Additionally, relative decreases in ventricular preload depend largely on pre-existent patient factors such as ventricular compliance making patients with HOCM and diastolic dysfunction more susceptible to develop SAM.[2,7]

CONCLUSION

This case demonstrates the growing utility of echocardiography (TTE and TOE) in non-cardiac surgery as a diagnostic tool and as a guide to therapy

in managing refractory hypotension in the operating room setting. The case report also highlights the necessity to consider SAM in the differential diagnosis of unexpected refractory intraoperative hypotension and brings focus on the need for anaesthesiologists to become proficient with echocardiography.

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