Anaesthetic management in a case of concurrent hypertrophic cardiomyopathy and constrictive pericarditis: Are there special concerns?

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is an obstructive cardiac disorder which has autosomal inheritance and constrictive pericarditis (CP) is acquired in nature. Because of varied pathophysiology, these two diseases have divergent anaesthetic goals. We describe the special concerns in anaesthetic management of a patient with concomitant HCM and CP undergoing decortication and pericardiectomy. Consent was obtained from the patient for reporting this case.

CASE REPORT

A 43-year old male weighing 53kg, without any comorbidity presented to the hospital with gradually increasing breathlessness of 3 months duration, one episode of haemoptysis, and oedema of legs. Examination revealed a baseline heart rate of 106/min and blood pressure was 114/70 mmHg, grossly reduced breath sounds on left lung fields and a grade 3/6 ejection systolic murmur. Chest X-ray showed heterogeneous opacity in left lower lung fields [Figure 1 (Panel A)]. Transthoracic echocardiography revealed asymmetric hypertrophy of interventricular septum (IVS) (19 mm) and IVS to posterior wall thickness ratio of 1.3 with mid-cavitary gradient of 60 mmHg. There was also mitral inflow variation of more than 25%, tricuspid inflow variation of 40%, expiratory flow reversal of hepatic veins and inferior vena cava plethora consistent with CP. Computed tomographic scan showed pleural and pericardial calcification [Figure 1 (Panel B)]. A catheterisation study demonstrated normal coronaries and confirmed presence of pericardial calcification [Figure 2]; however myocardial biopsy for confirmation of HCM was not taken. The patient was already on anti-tubercular treatment (ATT) for previous 2 months and sputum examination was negative for acid-fast bacillus. The patient was started on tablet metoprolol 25 mg twice daily in view of the HCM.

As the patient refused any surgical intervention for HCM, he was scheduled for decortication and pericardiectomy in single session surgery via anterolateral thoracotomy. ATT and beta blockers were continued up to the day of surgery. In the operation theatre standard monitoring were applied. Defibrillation pads were applied in anteroposterior position. The patient was premedicated with IV fentanyl 50 μg and midazolam 1 mg. Arterial and central venous lines were placed in the right radial artery and right internal jugular vein, respectively. Thoracic epidural anaesthesia was avoided because of the potential adverse effects of sympathetic block and vasodilation on an HCM patient. The patient was induced with fentanyl 200 μg, midazolam 3 mg and thiopentone 150 mg in incremental doses and tracheal intubation with a 37 Fr left sided double lumen tube was facilitated with rocuronium. The patient was ventilated with low tidal volume (5 ml/kg) and higher rate (16–20) to minimise compromise of venous return. Anaesthesia was maintained with 50% O2 in air with

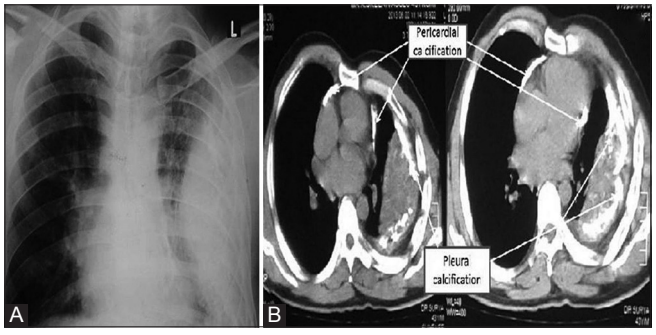

Figure 1: Panel A-Chest X-ray posteroanterior view showing heterogeneous opacity due to pleural thickening and calcification. Panel B-Computed tomography images demonstrating pericardial and pleural calcification (White arrows)

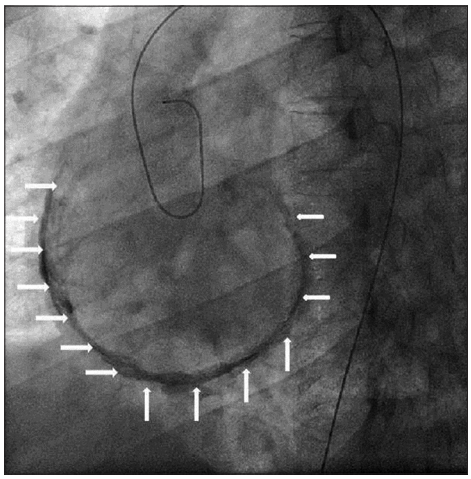

Figure 2: Left Anterior Oblique view in catheterisation study showing pericardial calcification (White arrows)

titrated doses of sevoflurane, vecuronium and fentanyl infusion to maintain heart rate of 75–85/min and blood pressure within 20% of baseline. Thoracotomy was performed and one lung ventilation(OLV) was initiated. During OLV pulmonary gas exchange was well-maintained and there were no significant changes in airway mechanics. An episode of paroxysmal supraventricular tachycardia which occurred during pericardial dissection resolved spontaneously when stimulation was stopped. Intraoperatively there was one episode of hypotension associated with blood loss. This was treated with phenylephrine (50 μg) and blood transfusion. Total blood loss during surgery was approximately 1L and patient was transfused one unit of packed cells and a total of 1.5 L of crystalloids. Urine output was normal during surgery.

For postoperative analgesia intercostal block was administered under vision by the surgeon before closure of chest. The patient was ventilated post operatively as the hospital protocol mandates elective ventilation in all pericardiectomy patients. In the intensive care unit analgesia was maintained with fentanyl infusion 1.5 μg/kg/h. The patient was weaned and extubated after 12 hours. Rest of the patient's hospital stay was uneventful. Post-operative Echocardiography showed HCM gradient of 45 mmHg and absence of CP features. The patient was discharged with the advice to continue β blockers and follow up for HCM.

DISCUSSION

HCM and CP are potentially life-threatening diseases rarely seen in combination. Given the fact that HCM has an incidence of 1 in 500[1] and CP of tuberculous origin is still common in developing and underdeveloped countries, anaesthesiologists in these areas are more likely to come across this combination than expected.

In general, both HCM and CP are diastolic dysfunctions of the heart. HCM is a genetic disorder with autosomal dominant transmission.[1] The genetic mutation in HCM causes asymmetric hypertrophy of the ventricular wall leading to either left ventricular outflow tract obstruction, mid-cavitary ventricular obstruction (MVO), or apical hypertrophy. MVO, as seen in our case, is a relatively rare type of HCM with an incidence of 9.4–12.9%.[2,3] The clinical course of the disease may vary from absolutely asymptomatic life to sudden death. The most common non-surgical treatment for HCM is use of β blockers/calcium channel blockers to reduce the heart rate, contractility and thus the obstruction.[4] On the contrary CP is mostly an acquired condition resulting from chronic infection or inflammation of the pericardium or mediastinal irradiation. Due to the fibrotic constriction in CP, the diastolic filling is impaired. This results in a relatively fixed stroke volume, making maintenance of cardiac output dependent on increases in heart rate. Thus, tachycardia is a predominant feature of CP.[5] High heart rate desirous in CP can be deleterious in HCM patients. We targeted a heart rate of 80 ± 5 for our patient to balance the requirements of both conditions. Maintenance of adequate volume status is important in both CP and HCM. Both central venous pressure (CVP) and pulmonary capillary wedge pressure are unreliable markers in these patients as they overestimate volume status. Pulmonary artery catheter has an added risk of arrythmogenicity and CVP monitoring becomes more dependable after adequate pericardiectomy. Hence, we used serial trends in CVP to guide fluid therapy. Transoesophageal echocardiography (TOE) can be a very useful tool to determine the filling conditions in such a patient but in our institution TOE was not available.

Pericardiectomy can influence the HCM gradient. Pericardiectomy may improve diastolic filling of the ventricle leading to reduction in HCM gradient. However, pericardiectomy often results in low cardiac output syndrome.[6] The low cardiac output may underestimate the HCM gradient. On the contrary, the use of perioperative inotropes to manage low output syndrome may increase the heart rate and contractility leading to increasing of gradients in an HCM patient. In our patient, transient periods of hypotension were treated with phenylephrine, which is a pure alpha 2 agonist and does not increase heart rate and contractility.

Post-operative analgesia is of immense importance in patients with HCM to reduce sympathetic stimulation. The neuraxial blockade can be successfully used in patients with HCM but its haemodynamic effects may affect HCM gradients adversely.[7] Thus we avoided thoracic epidural in our patient. Instead we used intercostal block and intravenous opioids which are safer and reliable methods of pain relief.[8]

In a patient of concomitant HCM and CP, maintenance of optimal heart rate, filling pressures and adequate analgesia leads to favourable outcome. Although HCM gradient improved after pericardiectomy in our patient, more data are required to support or refute this finding.

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Conflicts of interest

There are no conflicts of interest.

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