Predicting intraoperative cardiovascular complication in patients with anterior mediastinal mass—role of central venous pressure monitoring

INTRODUCTION

The risk of life threatening cardiovascular collapse or airway compromise is well recognized in patients with large anterior mediastinal mass.[1-3] The peri-operative risk predictors like site, volume of the mass, extent on computed tomography (CT) scan of the mass are well known.[4] We report a case of dynamic subclinical superior vena caval (SVC) obstruction evident with continuous CVP monitoring in the peri-operative period and to utilize CVP changes as a risk predictor.

CASE REPORT

A 30-year-old male presented to the pre-anaesthetic clinic with history of dry cough for 9 months and recent onset of weight loss. There was no history of dyspnoea at any position. Dysphagia, muscular weakness, fatigue and drooping of eye lids were not elicited. On examination, he was conscious, comfortable without clinical evidence of either tracheal or SVC obstruction. The Pemberton’s sign was negative. The chest X-ray showed mediastinal widening [Figure 1]. The CT scan [Figure 2] revealed a large, $10.2 \times 9.3 \times 6.3$ cm well-defined heterogeneous mixed density mass lesion lying over the brachiocephalic confluence, SVC, right atrium (RA) and right ventricular (RV) outflow tract without any pericardial or pleural effusion. The fat plane above pericardium was preserved and separate from the mass. The normal airway caliber was maintained. The CT guided biopsy was non-informative with respect to the pathology of the lesion. Hence, this asymptomatic young, fit patient was posted for therapeutic excision of the anterior mediastinal mass with an uncertain chance of SVC and RV outflow obstruction during anaesthetic intervention.

A sequential plan of thoracic epidural and inhalational induction was planned. An epidural catheter was inserted at T4-T5 interspace. Invasive monitoring which included left radial artery for systemic pressure monitoring, right internal jugular vein (IJV) for CVP and left femoral vein for inferior vena cava (IVC) access were established under local anaesthesia with titrated dose of intravenous midazolam and morphine. After recording the baseline parameters, segmental epidural anaesthesia (T1-T6) was established with incremental dose from 6-10 ml of 1% preservative free lignocaine. Increasing amount of Sevoflurane with gas oxygen mixture was used for induction of anaesthesia. During this period, the CVP progressively increased from 15 mmHg to 24 mmHg with stable systemic pressures, oxygen saturation and heart rate. The airway was secured with size 4 Proseal laryngeal mask airway (LMA) without any relaxant and the surgeon was requested to proceed with sternotomy and take control of the mass. But the CVP dropped and stabilized at around 20 mmHg following application of sternal retractor. Still, the systemic pressures were maintained around 100 mmHg systolic without vasopressors. The mass was predominantly a bag of necrotic material arising from thymus which was removed in toto with
the accompanying pericardial and pleural window. The patient was breathing spontaneously throughout the procedure and as the pleural window was opened, we started assisted ventilation with the proseal LMA. With this, CVP stabilized at around 8 mmHg till the end of the procedure. The Proseal LMA was removed at end of the surgery and the patient was shifted to the postoperative ward with intercostal drain (ICD) in situ. The patient was asymptomatic and the ICD was removed after 2 days. The analgesia was maintained with epidural bupivacaine infusion.

**DISCUSSION**

Mediastinal masses can present with predominant venous obstructive, low cardiac output or respiratory symptoms according to the site of obstruction. When the patient has definite signs, the peri-operative management strategy and the procedural risks are self explanatory. But in an asymptomatic patient like ours, with potential cardiovascular complication posted for major therapeutic procedure, the preoperative risk stratification and the management plan is crucial for safer outcome. Hence, we did literature search to identify the preoperative factors that predict the risk of developing haemodynamic compromise on induction in an asymptomatic patient. Bechard et al., correlated the preoperative findings with the peri-operative complication in adult patients with anterior mediastinal mass. They analyzed independent variables like preoperative signs and symptoms, pulmonary function test, tracheal compression more than 50% on CT, mass volume more than 130 cm³, pleural effusion, pericardial effusion, compression of SVC and the pathology of the lesion with peri-operative complications. They found significant correlation exist between the volume of the mass on CT scan more than 130 cm³ and the occurrence of perioperative complications. This patient had mass volume of 150 cm³ on CT scan. Hence, we placed our patient in a high risk category of developing haemodynamic compromise and planned for an inhalational induction with maintenance of spontaneous respiration till the surgeon takes control of the mass. During inhalational induction as the patient losing consciousness, the CVP progressively increased probably because of the SVC compression by the mass due to the progressive loss of intercostal muscle tone and reduction in the ribcage volume. The fall in functional residual capacity (FRC) due to the decrease in thoracic cage volume on induction of anaesthesia is a well-known phenomenon. High thoracic epidural anaesthesia reduced the ribcage contribution to thoracic volume expansion from 27% to 10% in awake spontaneously breathing volunteers. Although we used 1% lidocaine to avoid intercostal muscle weakness and chosen inhalational induction to preserve the inspiratory intercostal muscle activity, the relative contribution of both factors should be considered in the reduction of thoracic cage volume in our patient. In spite of increasing CVP, stable systemic arterial pressure and saturation indicate the pressure effect was more on the SVC than on the RV out flow tract. This was the prime reason for which we avoided neuromuscular blockers and controlled ventilation. Johnson et al., demonstrated that the decrease in cardiac index associated with a mediastinal mass results from an increase in right ventricular afterload, causing right ventricular enlargement. Subsequently, there is impingement on the left ventricle volume because of interventricular interdependence. The fall in CVP to 20 mmHg following sternal retraction and 8 mmHg after the removal of the mass further indicate the trend in CVP was due to the mass effect on the SVC.

The avascular mass was a bag of necrotic material and the estimated blood loss was about 200 ml. Hence, we could not attribute the fluid shift as a cause for the trend in CVP. The contents of the mass, even though big was fluid, which may be the possible reason for its dynamic and incomplete obstruction. The base line CVP in our patient was 15 mmHg before induction which was considerably higher for his young age and clinically normal heart. We didn’t attach much significance to the high base line CVP and did not measure CVP in either the lateral or prone position which would have further suggested any dynamic SVC compression.

**CONCLUSION**

In an asymptomatic patient with large anterior mediastinal mass, continuous CVP monitoring especially in different positions may help in identifying subclinical SVC obstruction and this may be a potential predictor of haemodynamic compromise on induction. When in doubt, it’s better to use inhalational induction and preserve spontaneous breathing till the surgeon takes control over the mass. Our finding needs further confirmation with larger samples and comparative evaluation with other known risk factors.

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