Case Report:

Anaesthetic considerations in a patient with an anterior mediastinal mass

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ABSTRACT

We report a 35-year-old gentleman who presented to us with respiratory distress. He was diagnosed to have a large anterior mediastinal mass and was and posted for debulking of mediastinal mass. Immediately after intubation, airway collapsed and there was difficulty in ventilating the lungs, followed by drop in oxygen saturation. After sternotomy, oxygen saturation and airway pressures improved. In this report we discussed in detail about the successful anaesthetic considerations for patients with large anterior mediastinal tumour including the management of airway obstruction.

Key words: *Anterior mediastinal mass, Airway collapse, Sternotomy*

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INTRODUCTION

Patients presenting with a large anterior mediastinal mass pose challenge to the anaesthesiologist due to its complications ranging from dynamic airway collapse to haemodynamic collapse on induction of anaesthesia.[1] At the extreme end of the disease spectrum, however, anterior mediastinal tumours can be very difficult to manage in the perioperative period, and the literature provides many examples of cardiorespiratory disasters and even death.[2,3] Many of these publications relate to paediatric practice. Here we report the anaesthetic difficulties encountered in an adult patient who presented to us with respiratory distress and was posted for debulking of mediastinal mass.

CASE REPORT

A 35-year-old male presented with a one month history of breathlessness and chest pain which was more in supine position. He was comfortable while in the sitting and lateral position. After thorough clinical and radiological evaluation, he was diagnosed to have large anterior mediastinal tumour and was posted for emergency sternotomy and debulking of the tumour. Review of the pre-operative CT imaging (Figure 1) revealed a 13 $\times$12 cm large mass in anterior mediastinum extending into the superior and middle mediastinum displacing the superior vena cava and right atrium and compressing trachea and right main bronchus. Physical examination revealed that he was in respiratory distress. Prominent non-pulsatile jugular venous pulse was evident. Patient was shifted to

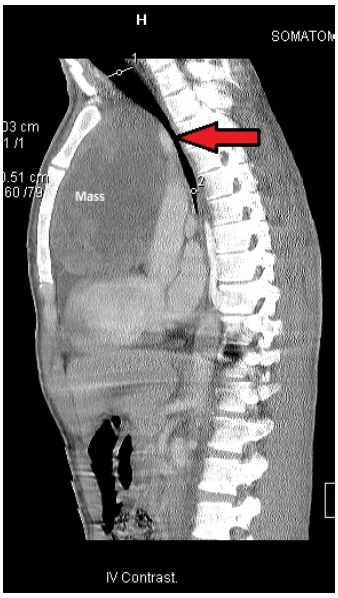

Figure 1: CECT chest (saggital reconstruction) showing a soft-tissue mass measuring 12 $\times$ 13 cm showing minimal enhancement on intravenous contrast administration in the anterior mediastinum compressing the distal part of trachea

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the operating room and was positioned in the lateral position. Apart from the routine American Society of Anaesthesiologist (ASA) standard monitoring, left radial artery was cannulated with 20 gauge cannula for invasive arterial pressure monitoring and a 16 gauge peripheral intravenous cannula was inserted in the lower limb under local anaesthesia. After 3 minites of pre oxygenation with 100% oxygen, general anaesthesia was induced slowly by inhalational administration of sevoflurane up to 8%. After check ventilation with face mask, endotracheal intubation (ETI) was facilitated with injection vecuronium 0.1 mg/kg. Trachea was intubated with 7.5 mm plain endotracheal tube (ETT). We encountered severe resistance to manual bag ventilation immediately after intubation. We thought that it could be due to tracheal or right main stem bronchial compression by large anterior mediastinal mass. We could not negotiate the tracheal compression with a single lumen plain ETT, so we did not think of using a double-lumen ETT. The lungs could not be ventilated effectively to maintain arterial oxygen saturation by pulse oximetry (SpO_2) of more than 90%. The patient had one episode of bradycardia (heart rate <30 beats per minute) and was successfully treated with intravenous injection of 0.6 mg atropine. But, the oxygen saturation did not improve. Considering the possibility of tracheal collapse, the surgeon decided to go ahead with emergency sternotomy. Immediately after sternotomy, the entire mediastinum was filled with the tumour mass (Figure 2). There was a decrease in airway pressure and improvement in oxygen saturation. The surgeon proceeded for debulking of the tumour and only part of the tumour weighing around 350 mg was debulked and submitted for histopathology which was suggestive of primary mediastinal mixed malignant germ cell turmour. Patient lost around 2.5 liters of blood during the procedure and developed hypotension which was managed with intravenous fluids, blood transfusion and adrenaline infusion. In view of short period of hypoxia for about 5 min in the post ETI period and unstable haemodynamic status, it was decided to ventilate the patient electively in the post-anaesthesia care unit (PACU). Patient was extubated and was administered oxygen through a face mask during the first post-operative day. He had uneventful recovery, became haemodynamically stable after that and was discharged from the PACU in the second post-operative day.

DISCUSSION

Airway and vascular collapse is a well-recognized complication when patients with an anterior mediastinal mass are managed surgically.[1] The risk of life-threatening or fatal airway obstruction or cardio-vascular collapse during induction of anesthesia in patients with central airway obstruction has been recognized since the 1970s.[4] Over the subsequent three decades, anaesthesiologists have been aware of the high risk associated with general anaesthesia in these patients. When possible, in symptomatic patients with large anterior mediastinal masses, children with orthopnea and in more than 50% of tracheal compression general anaesthesia is avoided.[5] If administered, it is

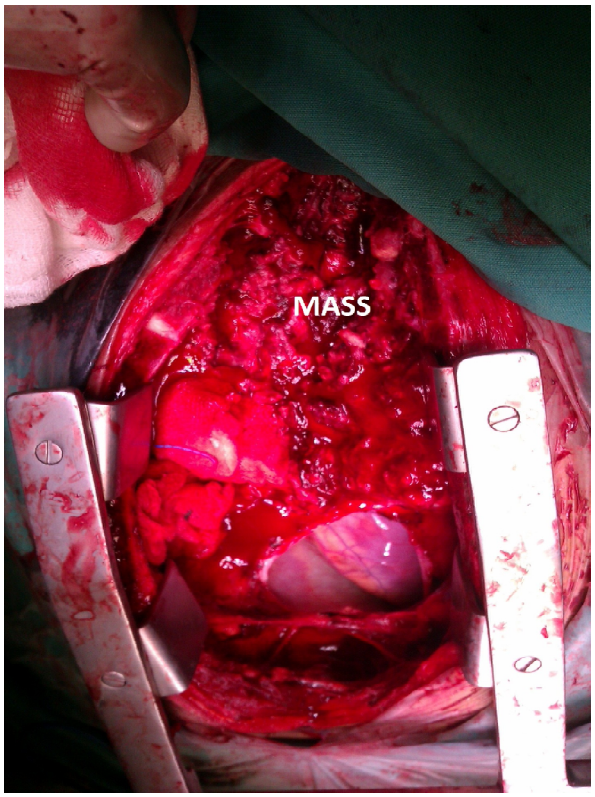

Figure 2: Intraoperative photograph showing mediastinal mass

titrated stepwise induction with maintenance of spontaneous ventilation and avoidance of neuromuscular blockers is strongly advised. Only after a successful trial of manual ventilation, the use of neuromuscular blocking drugs should be considered.

During anaesthesia lung volumes are reduced and bronchial smooth muscle relaxes, thereby increasing the compressibility of large airways.[6] Partially obstructed respiration, which can occur during an inhalational induction, generates large negative pressures that tend to flatten further, a trachea weakened by extrinsic compression. Muscular relaxation, on the other hand, causes loss of chest wall tone and disrupts the forces of active airway inspiration, thereby, further reducing external support of the narrowed airway. Abnormal patterns of spontaneous breathing on emergence also can cause partial obstruction of breathing on inspiration.

General anaesthesia also results in reduction of functional residual capacity (FRC) and alters respiratory mechanics in a manner that favors airway collapse. In the supine position, the abdominal contents tend to push the diaphragm cephalad in to the chest, which reduces negative intrapleural pressure and FRC. The compliance of the thoracic wall is reduced in the anaesthetized state, further decreasing FRC by approximately 20%.[7] Consequently, the chest wall expansion/lung-elastic recoil equilibrium favors smaller lung volumes and closure of susceptible airways especially in dependent areas of the lung. In the pathologic state, such as in the presence of an intrathoracic mass, both anatomic and physiologic perturbations may occur. Mediastinal tumour invasion may compromise the cartilaginous structural integrity, making the airway more susceptible to direct tumour compression. With tumour invasion, intrathoracic collapse with forced expiration and reduced or lack of airway dilatation during inspiration may occur. Similarly, partial upper airway obstruction in the rapidly spontaneously breathing patient may generate sufficiently negative intraluminal pressure to collapse the compromised segment.

In our case, the patient was posted for sternotomy and debulking of the tumour. Sternotomy is a very painful procedure which requires adequate analgesia, deeper planes of anaesthesia and good muscle relaxation to spread the sternum. We thought that spontaneous ventilation is difficult to maintain, hence we decided to secure the airway with the help of neuromuscular blocking agents. We could maintain the ventilation with face mask before the tracheal intubation but were unable to ventilate after ETI probably because of collapse of the trachea or bronchi distal to the ETT. The only option available to us at this stage *in lieu* of falling arterial oxygen saturation by pulse oximetry (SpO_2) is to release this airway pressure by doing an emergency sternotomy.

The other life saving maneuver at this stage could be a direct rigid bronchoscopy[8] or to institute cardiopulmonary bypass (CPB).[9,10] We could not exercise first option because of unavailability of rigid bronchoscope. In experienced hands a rigid bronchoscope therefore can be advanced to stent the airway.[11] After initial assessment of the anatomy/pathology, it may be possible to stent[11] the airway for resection surgery with an ETT or a double-lumen endobronchial tube placed under direct vision into the most patent main bronchus. If this technique is not possible, ventilation can be maintained down the rigid bronchoscope via a Venturi injector, in the usual way, and anaesthesia can be maintained intravenously. Once surgery has commenced and the tumour has been lifted anteriorly, the degree of airway obstruction tends to lessen, and it may be possible to replace the bronchoscope with an endotracheal tube at some stage.

Though the second option was feasible in our setup, it could not be implemented, as the case was taken up on emergency basis and both the available heart lung machines were occupied at that particular time for other open heart surgeries. The establishment of CPB by femoral cannulations prior to induction of anaesthesia has been safely

performed in adult patients. However, once airway or cardiovascular collapse has occurred, it will require at least 5-10 min to cannulate and establish adequate circulation and oxygenation,[10] even with a primed pump and a prepared team. In such a scenario, it is probable that a young patient can be resuscitated but may suffer neurological injury.[12]

This case report is an example of successful anaesthetic management of patients with airway obstruction. In patients at-risk of airway obstruction, whenever possible try to avoid general anaesthesia and if at all general anaesthesia is required, spontaneous ventilation should be maintained as long as possible during and after induction of anaesthesia. The decision to use neuromuscular blockers after induction and intubation may be dangerous in the high-risk patient with disease in the distal trachea or main bronchus. Use of these drugs is strongly discouraged if the planned procedure is solely diagnostic in nature and thus devoid of any meaningful tumour debulking. Repositioning adult patients lateral or prone in comparison with children may be less effective owing to a more ossified and developed chest wall. Should airway collapse occur, rigid bronchoscopy should be immediately available to facilitate lifesaving ventilation. Lastly, if a high-risk patient requires a general anaesthesia, strong consideration should be given to placement of the femoral cannulae with CPB machine stand-by before induction.

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