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Letter to Editor

Anaesthesia Section

Navigating Airway Obstruction during Resection of Anterior Mediastinal Mass: An Anaesthetic Perspective

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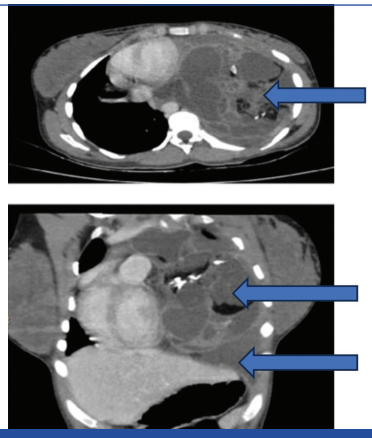
Dear Editor,

An Anterior Mediastinal Mass (AMM) poses significant challenges for the anaesthesiologist due to its impact on surrounding mediastinal structures [1]. They can cause airway and circulatory compression under general anaesthesia, leading to dreaded complications [2]. Effective management of AMM resection requires understanding its relationship with cardiorespiratory structures, thorough preoperative evaluation and anticipation of intraoperative complications. Strategies such as awake fiberoptic intubation, spontaneous ventilation, judicious use of muscle relaxants, positional adjustments, access to rigid bronchoscopy and in severe cases, elective cardiopulmonary bypass if available, enhances a safer outcome. This case explores the anaesthetic management and complexities faced in managing the resection of a large AMM.

A 19-year-old female presented with cough, dyspnoea which worsened on supine position, chest pain which was gradual in onset, non-progressive in nature, dull and non-radiating, blurring of vision, headache and intermittent low-grade fever for eight months. Computed Tomography (CT) revealed a 15×14.4×12.3 cm thymic mass displacing mediastinal structures and abutting left brachiocephalic vein, aorta, pulmonary trunk, left atrium, and ventricle, with mild left pleural effusion [Table/Fig-1]. General examination showed tachypnoea- 32 breaths/min, 94% oxygen saturation, reduced air entry on the left side and bilateral wheeze. She was diagnosed with thymic hyperplasia and scheduled for sternotomy and debulking under American Society of Anaesthesiologists (ASA) III. Preoperative treatment included nebulisations with levosalbutamol, ipratropium bromide, and budesonide twice daily, intravenous hydrocortisone 100 mg twice daily, chest physiotherapy, and incentive spirometry.

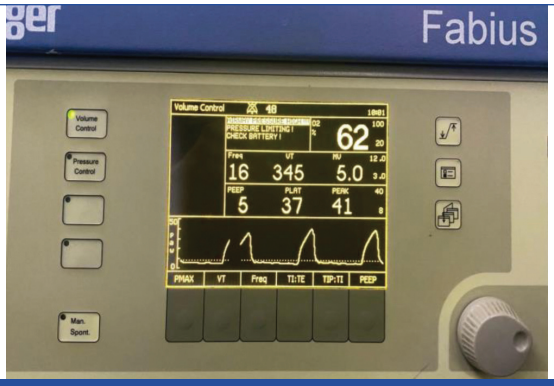


[Table/Fig-1]: CT thorax (axial and coronal view) depicting intrathoracic mediastinal mass compressing cardiopulmonary structures and left sided pleural effusion.

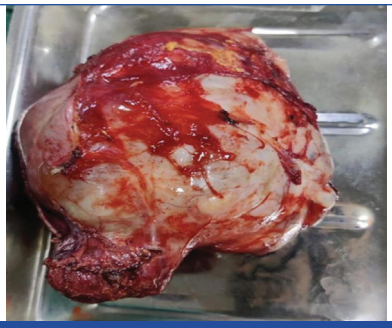
A comprehensive preoperative evaluation indicated a significant risk of airway compression during general anaesthesia. Consequently, the anaesthetic plan included thoracic epidural anaesthesia and awake fiberoptic intubation with wire-reinforced Endotracheal Tube (ETT) due to anticipated difficult intubation. Strategies included maintaining spontaneous ventilation until airway was secured, and standby rigid bronchoscopy. Difficult airway trolley, inotropes, vasodilators and defibrillator were prepared. Preoperative preparation for awake intubation included nebulisation with 3 mL of 4% lignocaine, oxymethazoline nasal drops in both nostrils and nasal packing with gauze piece soaked with 5 mL of 2% lignocaine with adrenaline and 5 mL normal saline for three minutes. Intramuscular glycopyrrolate 0.004 mg/kg and intravenous ondansetron 0.1 mg/kg were given 20 minutes prior to procedure.

In the operating room, standard ASA monitors were connected and epidural catheter was fixed at T6-T7 level. Airway anaesthesia was achieved with bilateral glossopharyngeal nerve block with 2 mL of 2% lignocaine on each side, along with transtracheal injection with 3 mL of 2% lignocaine. The patient was preoxygenated and premedicated with intravenous midazolam 0.01 mg/kg and fentanyl 2 mcg/kg. Nasal intubation was performed with 6.5 mm wire-reinforced cuffed ETT, guided by fiberoptic bronchoscope, the tube was secured at 25 cm and bilateral air entry was confirmed. A 9 Fr Arndt bronchial blocker was subsequently introduced into the right bronchus and the balloon was inflated under fiberoptic guidance after confirming proper ETT placement and achieving effective lung isolation. General anaesthesia was induced with inj. propofol 1 mg/kg and inj. ketamine 2 mg/kg. One-lung ventilation was confirmed, and neuromuscular blockade was achieved with inj. vecuronium 0.1 mg/kg. General anaesthesia was maintained with oxygen, nitrous oxide, and sevoflurane, tidal volume at 6 mL/kg, Positive End Expiratory Pressure (PEEP) of 5 cm H_2O. Post induction, her blood pressure was 124/78 mmHg, heart rate of 74 beats/min, oxygen saturation of 96% and peak airway pressure of 29 cm H_2O. Central venous access was achieved through the right femoral vein, and left radial artery was cannulated for invasive blood pressure monitoring. Dexmedetomidine infusion was started at 0.3 mcg/kg/hour prophylactically, to attenuate sympathetic response, to prevent tachycardia and Ventilation-Perfusion (V/Q) mismatch, and to prevent exacerbation of Superior Vena Cava (SVC) syndrome.

Approximately, five minutes after administering muscle relaxant, plateau pressure and peak pressure increased significantly, 38 cm H_2O and 41 cm H_2O, respectively, and patient desaturated to 76%, with sudden onset of hypotension [Table/Fig-2]. The bronchial blocker was deflated temporarily to allow both lung ventilation and improve oxygenation. Measures including increasing FiO_2 to 100%, applying PEEP to the ventilated lung, suctioning of the airway was done and dexmedetomidine and inhalational agents including nitrous oxide were discontinued. Manual ventilation was initiated and for haemodynamic stability, phenylephrine boluses and noradrenaline at 0.05 mcg/kg/min were administered, and the patient was positioned to the right lateral decubitus to relieve compression of mass on major vessels and airway and to improve V/Q mismatch. Hydrocortisone 2 mg/kg was administered, along with salbutamol and budesonide metered dose inhalers through the ETT. Manual



[Table/Fig-2]: Ventilator changes post induction.



[Table/Fig-3]: Resected Anterior Mediastinal Mass (AMM) weighing 1.5 kg following median sternotomy.

ventilation was continued till saturation plateaued to 91%, and the airway pressures improved. Repeat auscultation revealed air entry in right upper zones compared to right basal area. Check fiberoptic bronchoscopy revealed that the ETT had been misplaced into the right upper bronchus. The tube was withdrawn by 2 cm guided by fiberoptic bronchoscopy and secured, and recruitment manoeuvre was initiated until saturation improved to 96%.

The patient was repositioned to supine and the surgeons immediately preceded with sternotomy and lifting the mass. The surgery lasted approximately 4.5 hours and the tumour weighing 1.5 kg was resected through a median sternotomy [Table/Fig-3], and sent for histopathology. The airway pressure decreased and oxygen saturation improved to 99%. For intraoperative analgesia, 0.125% bupivacaine and 2 mg morphine were administered via the epidural catheter before skin incision. Intraoperative Arterial Blood Gas (ABG) monitoring was done every hourly to assess oxygenation, ventilation status and haemoglobin monitoring. To minimise postoperative airway oedema, intravenous hydrocortisone 2 mg/kg and dexamethasone 0.1 mg/kg were administered. Prior to extubation, cuff leak test was done and volume was noted to be >110 mL, indicating adequate airway patency. After confirming adequate ventilation, oxygen saturation of >98% and protective airway reflexes, patient was reversed with inj. sugammadex 4 mg/kg and was extubated and subsequently transferred to the surgical Intensive Care Unit (ICU) for further monitoring. Emergency reintubation equipments and difficult airway cart were kept ready at the bedside. Post-extubation, the patient received nebulisation with budesonide and salbutamol to reduce airway inflammation. No signs of respiratory distress or airway compromise were observed post-extubation. Recovery period was uneventful in Surgical Intensive Care Unit (SICU) with stable haemodynamics, normal respiratory function, and no signs of airway compromise.

Managing intraoperative SVC syndrome during mediastinal mass resection requires optimising venous return and fluid resuscitation, maintaining airway patency and ensuring haemodynamic stability. Induction during mediastinal mass resection may require a semi-erect or sitting position to avoid airway compression and to optimise airway management, as in the index case, where intubation was done in semi-Fowler's position [3,4]. Awake fiberoptic intubation is the gold standard for securing the airway, as general anaesthesia can cause bronchial smooth muscle relaxation, worsening tracheal compression, elevate airway pressures and difficult ventilation [3]. Inhalational induction should be avoided or used cautiously, as partial airway obstruction could also create negative pressures, potentially collapsing a compressed trachea and causing obstruction [5]. If ventilation is impaired, steps such as increasing oxygen concentration, both lung ventilation and maintaining functional residual capacity are vital to reduce hypoxaemia [1]. Repositioning the patient can help relieve airway obstruction, but care must be taken to avoid or identify ETT malposition. Muscle relaxants should be used only after confirming airway patency and adequate ventilation. Rigid bronchoscopy with a skilled surgeon on standby is essential for airway assessment and management. Large-bore intravenous access and femoral central lines are recommended [4]. Post-extubation, ICU monitoring is critical due to risk of emergent reintubation.

Constant vigilance and preparedness, and close monitoring of ventilatory and haemodynamic parameters are key to avoiding catastrophic airway or circulatory collapse. Extreme care and vigilance are needed to prevent apnoea, as there exists a significant risk of failing to intubate or establish a patent airway in such patients. In situations of emergency, immediate assistance should be called upon, including notifying the surgeon and arranging equipments for the management of such difficult and challenging airway.

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