General Anaesthesia with Muscle Paralysis in Patients with Anterior Mediastinal Mass - an audit of 15 cases and review of literature

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ABSTRACT

This study was undertaken to evaluate the safety of positive pressure ventilation (PPV) in patients with Anterior Mediastinal Mass (AMM) and to identify the preoperative features which may guide safe anaesthesia.

General Anaesthesia (GA) with PPV can lead to cardiorespiratory collapse in patients with AMM. However, adequate relaxation facilitates major surgery and it is important to be able to predict when it is safe to use muscle paralysis. The medical notes of patients who had undergone resection of AMM were studied to look for complications developed due to PPV. The clinical features, lung functions, echocardiography and computed tomography (CT) findings were recorded to see if any of them could predict adverse events.

Of the fifteen surgeries evaluated, six patients had symptoms suggesting airway compression but CT scan showed patent airways. In nine patients the mass was abutting pulmonary vessels, one patient had pericardial effusion and one had mediastinal shift with left lung collapse. None of these patients developed major cardiorespiratory compromise with PPV.

Clinical symptoms and CT images together are useful in planning safe anaesthesia. In doubtful cases ‘manually ventilating test’ can predict the safety of using PPV. This is especially useful when the AMM is compressing the pulmonary vessels.
INTRODUCTION

Patients with anterior mediastinal mass (AMM) can develop life threatening cardiorespiratory complications after the induction of General Anaesthesia (GA) and the institution of positive pressure ventilation (PPV), due to the compression of underlying major airways or vascular structures. This was described by Dr Bittar in 1970s as Mediastinal Mass Syndrome (MMS). MMS is defined as acute right ventricular failure secondary to vascular compression after the initiation of PPV.\(^1\)

Supine position leads to a reduction in thoracic volume and an increase in central blood volume leading to a relative increase in the volume of the AMM. Induction of anaesthesia further reduces the lung volumes and relaxes the bronchial smooth muscles. Muscle paralysis and PPV reduce the normal transpleural pressure gradient which dilate the airways. All these factors make the airways more compressible by the overlying mass under GA.\(^2\) Compression of the main pulmonary artery (PA) by the AMM is usually rare as it is protected by the overlying high pressure vessel, aorta. But compression of the pulmonary trunk or one of its branches can cause sudden hypoxaemia, hypotension and even cardiac arrest.\(^3\) Institution of positive pressure ventilation (PPV) impairs venous return and increases the pulmonary vascular resistance which can further compromise the pulmonary perfusion. These patients may be asymptomatic when awake or have subtle features like dyspnoea on exertion, arrhythmias, cardiac murmur or syncope during forced Valsalva manoeuvre.\(^2\) It is important to ascertain the relation of AMM with all these structures on Computed Tomography (CT) scans.

The standard recommendation in patients with AMM is to avoid GA if possible and maintain spontaneous respiration when GA is administered. Clinical evidence to support this recommendation comes from case reports in which cardiorespiratory compromise occurred following the use of muscle paralysis and PPV or from those cases which were safely managed on spontaneous respiration.\(^4\) However adequate muscle relaxation is helpful in cases like thoracotomy or rigid bronchoscopy for tracheal stenting. Anaesthetists do frequently administer muscle relaxants in adult patients with AMM without untoward consequences. The intraoperative complication rate in adults is much lower than in the paediatric age group.

There are few case series describing the safe use of muscle relaxants and PPV in patients with AMM. Most of them focus on airway compromise and airway maintenance rather than compression of pulmonary vessels. Even the large series by Bechard et al have not specifically looked at the use of muscle relaxants in the conduct of GA.\(^5\) The purpose of this audit was to evaluate the complications that have occurred in our patients with AMM when muscle paralysis and PPV were used, to evaluate the preoperative findings that can guide safe decision making and review the current literature regarding anaesthesia for patients with AMM. This will help the anesthesiologists who encounter patients with AMM in making a rational choice of anaesthesia technique.

METHODS

The medical notes of adult patients who underwent surgery for the excision of AMM during the period from October 2011 to June 2016 under the thoracic surgical unit were evaluated with the approval of Institutional Scientific & Ethics Board.

The following points were recorded from the patients’ notes:

- clinical features when the patient presented for surgery.
• Pulmonary functions tests (PFT) were graded as restrictive, obstructive or mixed using the methodology adopted by Bechard et al. [5]

  - Obstructive if forced expiratory volume at one second (FEV1) was less than 80% of predicted with a FEV1/FVC ratio less than 70%
  - Restrictive if forced vital capacity (FVC) was less than 80% of predicted with an FEV1/FVC ratio of 70% or more.
  - Mixed when there was a combination of both obstructive and restrictive features.

• Findings on echocardiography report

• Impact of the AMM on the surrounding vital structures as reported on the Computed Tomography (CT) scan report.

• Mass volume of the AMM was estimated using the three diameters, anteroposterior, cephalocaudad and transverse, given on the CT scan report \((d_1,d_2,d_3)\) with the formula \(-\frac{4}{3} \times \pi \times d_1/2 \times d_2/2 \times d_3/2 (cm^3)\). [5]

The following when recorded on anaesthesia chart were recorded as adverse events:

• Inability to ventilate the lungs or peak airway pressure greater than 40cm H2O
• Oxygen saturation of less than 95% at an FiO2 (fraction of inspired oxygen) of 1.0
• Drop in systolic blood pressure below 20% of baseline for 5 minutes and necessitating aggressive treatment.

Anaesthesiologists conducting the case were also interviewed if any clarification was required.

**RESULTS**

Fifteen surgeries for the excision of AMM were carried out in this period. Table 1 gives the details recorded from patients’ notes. Six patients had no postural symptoms at the time they were taken up for surgery, six patients had clinical features like dry cough, dyspnoea, decreased air entry on one side, hoarseness of voice and chest pain indicating some involvement of the airways. Twelve patients had a mass volume above 100cm3 with the largest mass being above 4000 cm3. Eight patients showed a restrictive pattern on lung function tests indicating some compression of lung parenchyma. In nine patients, the mass was abutting the pulmonary artery (PA) or one of its branches and one had pericardial effusion. All patients received muscle relaxants at induction and during maintenance of anaesthesia. Attempt was made to maintain spontaneous respiration in the patient no 15 (Table 1) as he had myasthenia gravis. However, this patient needed double lumen tube (DLT) for surgical access. Insertion of DLT was not possible because the vocal cords kept adducting, hence a short acting muscle relaxant was administered for intubation. As the patient tolerated muscle paralysis and PPV without any complications, a long acting relaxant and controlled ventilation was continued throughout the surgery. Patient no 9 showed moderate pericardial effusion on the preoperative echocardiography, with no postural symptoms. Anaesthesia was induced using titrated doses of propofol and sevoflurane. After confirming that patient tolerated PPV, short acting muscle relaxant was administered, with measures for pericardial tapping kept ready. The blood pressure (BP) dropped minimally and responded immediately to vasopressors. Pericardial fluid tapping was not needed at induction. Patient no 15 who had a very large mass, had very poor lung functions with collapse of one lung. Using controlled ventilation was considered the safest option in this patient as induction of anaesthesia would have compromised his lung functions further. Here too, a trial of PPV was given after induction of anaesthesia with propofol and sevoflurane followed by a short acting muscle relaxant. The blood pressure which dropped after induction of
anaesthesia responded immediately to a change in position and the case was conducted subsequently using muscle paralysis and PPV.

### Table 1: Results

<table>
<thead>
<tr>
<th>Sex/Age in years</th>
<th>Histological Diagnosis</th>
<th>Clinical features at the time of surgery</th>
<th>Lung Function Tests</th>
<th>CT Scan features</th>
<th>2D Echo findings</th>
<th>Adverse Events</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>volume of AMM [cm³]</td>
<td>Impact on surrounding structures</td>
<td></td>
</tr>
<tr>
<td>1 F/58</td>
<td>Hurthle cell Ca of ectopic thyroid</td>
<td>Nil</td>
<td>Normal</td>
<td>34</td>
<td>Nil</td>
<td>Normal</td>
</tr>
<tr>
<td>2 F/45</td>
<td>Teratoma</td>
<td>Nil</td>
<td>Normal</td>
<td>122.12</td>
<td>Indentation of thoracic aorta and main PA</td>
<td>Normal</td>
</tr>
<tr>
<td>3 M/74</td>
<td>Thymoma</td>
<td>Nil</td>
<td>Normal</td>
<td>133</td>
<td>Nil</td>
<td>Normal</td>
</tr>
<tr>
<td>4 M/24</td>
<td>Germ cell tumour</td>
<td>Nil</td>
<td>Normal</td>
<td>197.92</td>
<td>Mild compression of left subclavian artery. Abuts main PA</td>
<td>Normal</td>
</tr>
<tr>
<td>5 F/33</td>
<td>Pleomorphic Liposarcoma</td>
<td>Nil</td>
<td>Obstructive</td>
<td>299</td>
<td>Partially compressing SVC, RV, and IVC. Abuts ascending aorta and right PA. Mass extends from right lung apex to diaphragm</td>
<td>Mass seen at junction of RV and IVC</td>
</tr>
<tr>
<td>6 F/33</td>
<td>Pleomorphic Liposarcoma</td>
<td>Nil</td>
<td>Restrictive</td>
<td>863.94</td>
<td>Compression of right lung, abutting mediastinum and great vessels</td>
<td>Normal</td>
</tr>
<tr>
<td>7 M/36</td>
<td>Teratoma</td>
<td>Dyspnoea on exertion, decreased air entry on left</td>
<td>Normal</td>
<td>138</td>
<td>Compression of main PA, left PA and left main bronchus</td>
<td>Normal</td>
</tr>
<tr>
<td>8 M/67</td>
<td>Thymoma</td>
<td>Cough, hoarseness of voice, dyspnoea on exertion</td>
<td>Restrictive</td>
<td>142.66</td>
<td>Abuts arch of aorta &amp; main PA. Minimal atelectasis of right upper lobe</td>
<td>Significant diastolic dysfunction</td>
</tr>
<tr>
<td>9 M/16</td>
<td>Thymic Neoplasm</td>
<td>Dry Cough</td>
<td>Restrictive</td>
<td>251.43</td>
<td>Mediastinal shift to right</td>
<td>Moderate pericardial Effusion</td>
</tr>
<tr>
<td>10 M/40</td>
<td>Thymoma</td>
<td>Pleural effusion - drained preoperatively</td>
<td>Restrictive</td>
<td>454.78</td>
<td>Mass infiltrates pericardium and lingual lobe of left lung</td>
<td>Normal</td>
</tr>
<tr>
<td>11 M/19</td>
<td>Germ cell tumour</td>
<td>Cough, decreased air entry on right, pain</td>
<td>Restrictive</td>
<td>465</td>
<td>Mass closely apposed to ascending thoracic aorta, SVC, distal aspect of right PA, ventral aspect of right upper lobe bronchus, pericardium overlying the RA</td>
<td>Normal</td>
</tr>
<tr>
<td>12 M/43</td>
<td>Thymoma</td>
<td>Dyspnoea on exertion, decreased air entry on left, chest pain</td>
<td>Restrictive</td>
<td>1649.34</td>
<td>Mediastinal Shift to right. Mass abutting left PA, pulmonary trunk, upper lobe bronchus</td>
<td>Normal</td>
</tr>
<tr>
<td>13 M/21</td>
<td>Teratoma</td>
<td>No air entry on left, comfortable in left lateral position. Can lie supine for short duration</td>
<td>Restrictive</td>
<td>4432.5</td>
<td>Displacement of mediastinum to right</td>
<td>Normal</td>
</tr>
<tr>
<td>14 F/67</td>
<td>Thymoma</td>
<td>Atrial Fibrillation (rate controlled)</td>
<td>Normal</td>
<td>91.13</td>
<td>Mass abutting PA</td>
<td>Anterosetal hypokinesia with prominent left atrial chamber</td>
</tr>
<tr>
<td>15 M/37</td>
<td>Thymic Carcinoma</td>
<td>Mynaesthesia - oculocutaneous and limb weakness</td>
<td>Restrictive</td>
<td>93.52</td>
<td>Dextroposition of heart, mass abuts PA</td>
<td>Poor window. Invasive thymoma abutting the PA</td>
</tr>
</tbody>
</table>

Abbreviations: M – Male, F - Female, PA – Pulmonary artery, RV – Right Ventricle, IVC – Inferior Vena Cava
It was observed that BP dropped by more than 20% of baseline in two patients, of which one responded to change of position and the other responded to phenylephrine. None of the patients developed airway obstruction, hypoxaemia or cardiovascular collapse.

In patients where uncertainty existed, manual ventilation test was reliable in predicting the safety of PPV.

**DISCUSSION**

Induction of anaesthesia in patients with AMM can cause life threatening compression of major airways and cardiovascular structures. Hence the standard recommendation in patients with AMM is to do a procedure under local anaesthesia when possible or to maintain spontaneous respiration when GA is administered. But in practical situations, only short procedures like imaging studies or biopsies can be conducted under local anaesthesia or without muscle relaxation. Major surgeries like median sternotomy, thoracotomy or even rigid bronchoscopy for tracheal stenting need adequate muscle relaxation. It is therefore important to be able to make a preoperative assessment to decide which patients can be safely administered muscle relaxants.

Fifteen surgeries were done for the excision of AMM and muscle relaxants were used in each of these patients. None of the patients developed significant cardiorespiratory decompensation with the use of muscle paralysis and PPV. In this series, none of the patients had compression of trachea, but the mass was abutting the bronchus, PA or its branch or the heart in nine patients. Four of these patients had symptoms like dyspnoea on exertion and arrhythmia which can indicate compression of right heart or pulmonary vessels. But none of the patients in this series who had involvement of pulmonary vessels, developed cardiovascular collapse on the initiation of PPV.

The size of the AMM had no influence on the development of complications with GA as 12 of the patients had AMM of volume greater than 100cm³ and the largest was 4432cm³. Clinical features present at the time of surgery were correlated with the CT scan images to look for compression of airways or pulmonary vessels. If there was no evidence of compression, muscle relaxants were administered. In uncertain cases, a combination of propofol and sevoflurane was used for the induction of anaesthesia in patients when there were concerns of decompensation at induction. These agents cause a loss of muscle tone and allow the trial of PPV, but are short acting and allow the patient to be immediately woken up in case of cardiorespiratory compromise. This helped in deciding if muscle paralysis and PPV would be tolerated by the patient and if not, then patient could be quickly woken up. This was fairly accurate in predicting the safety of using PPV.

Blank and de Souza stratify the anaesthetic risk based on the presence of postural symptoms, pericardial effusion, superior vena cava syndrome and radiological evidence of airway compression. Erdos and Tzanova use dynamic evaluation of airway (awake fibreoptic bronchoscopy or flow volume loops) in addition to the clinical symptoms and CT scan pictures, to plan the anaesthetic in patients with AMM. However, Slinger and Karsli consider patient’s history and chest imaging to be of greatest value in guiding the anaesthetic management with flow volume loops not offering any clinical benefits. A retrospective analysis of 105 anaesthetics in patients with AMM by Bechard et al is the largest case series of AMM. They found that patients at high risk of perioperative complications could be predicted by the presence of cardiorespiratory symptoms at presentation, CT scan evidence of tracheal compression of more than 50%, pericardial effusion and mixed abnormality on pulmonary function tests. Two patients in their series who had pericardial effusion experienced cardiovascular collapse after the initiation of PPV which could be treated. In the present series, one patient had pericardial effusion, but he tolerated PPV. The patients with tracheal compression of more than 50% also underwent GA without intraoperative complications in Bechard’s series. They observed that though there are reports of asymptomatic children who develop airway obstruction under anaesthesia, it is safe to administer GA in asymptomatic adults
after radiological evaluation. In this series six patients had symptoms pointing towards airway involvement, but the CT scan images did not show significant narrowing of the large airways. These patients did not develop any airway compromise with the induction of GA with PPV.

The trial of PPV was found to be a reliable guide to decide the use of muscle relaxants. Gharavifard et al have formally evaluated the “Manually ventilating test” as a predictive test to decide about paralysing children with AMM who need tracheal intubation. Out of the 20 symptomatic children, 19 could be ventilated manually and they could be paralyzed and intubated successfully. The one patient who could not be ventilated manually, was maintained on spontaneous respiration through a laryngeal mask airway along with a change in surgical plan. They state that CT scan is a static evaluation of an airway obstruction which is a dynamic event. Patency of narrowed airways is maintained by the negative pressure of surrounding parenchyma. By giving PPV, intrapleural negative pressure is lost which mimics the situation when muscle relaxants are given. Therefore, if PPV is possible during the manual ventilation after deepening anaesthesia, then it is safe to administer muscle relaxants.

Difficulty in bag and mask ventilation in patients with AMM is usually attributed to airway compression by the mass after the loss of muscle tone. But Sarkiss et al suggests that this occurs partly due to air trapping and consequent hyperinflation. They observed during rigid bronchoscopy of two patients with extrinsic airway compression that the airway diameter actually increased when PPV was administered. This diameter was even larger in paralyzed patients as muscle relaxants enhance airway and lung expansion. They could safely conduct rigid bronchoscopy and airway stenting in patients with AMM which had caused airway compression. The authors consider that airway obstruction occurs during expiration leading to unrecognised air trapping resulting in hyperinflation, which in turn increases the intrathoracic pressure leading to cardiovascular collapse. They therefore recommend an increase in expiratory time to avoid air trapping.

Though this study is limited due to the small number of cases, it is important because there are few case reports describing the safe use of muscle relaxants when AMM compresses the pulmonary vessels. More cases will need to be evaluated.

CONCLUSION

Muscle relaxants and PPV can be used safely in patients with AMM after judicious interpretation of the patients' symptoms at the time of surgery and correlating them with the CT scan images. If there is uncertainty after interpretation of the two, then stepwise induction of anaesthesia using short acting agents and test of manual ventilation should be given. If the patient tolerates the test ventilation, a short acting muscle relaxant can be administered followed by a long acting relaxant. Anaesthesia related morbidity and mortality in adult patients with AMM is much lower than in the paediatric age group.

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