Case report

Anaesthetic management of a huge mediastinal tumour with tracheobronchial compression

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Abstract

A 20 year old man diagnosed to have multiple neurofibromatosis presented with 2 months history of orthopnoea and cough with moderate expectoration since 1 month. Chest Xray and CT scan showed a huge mediastinal mass with compression of the trachea and superior vena cava. In view of the anticipated airway problems, an awake intubation in the sitting position was achieved. Cardiopulmonary bypass was not considered because of the possibility of excessive bleeding after heparinization. Despite this, the patient bled about 4,500 mL. Tracheomalacia was suspected intraoperatively. A larger endotracheal tube was inserted at the end of the procedure and the patient ventilated electively overnight. He was successfully extubated 24 hours later.

Keywords: Airway obstruction, cardiopulmonary bypass, mediastinal tumour.

Introduction

Anaesthesia for excision of mediastinal tumours can pose a great challenge, particularly related to airway control. They may also compress on the great vessels, oesophagus and other structures in the mediastinum. Anterior mediastinal tumours usually pose a greater threat to the airway compared to posterior mediastinal tumours.

These patients should be carefully evaluated before subjecting them to anaesthesia as even large mediastinal masses can present without any clinical symptoms of airway compression.[1,2] These large mediastinal tumours with apparently normal airways preoperatively may develop an obstructed airway after induction of general anaesthesia (GA).[2] Sometimes, a life threatening airway compression can occur even after an uneventful endotracheal intubation. Performing an emergency tracheostomy to relieve obstruction may prove futile, as the obstruction may be distal to the tube (lower airway obstruction).[3] In the presence of severe symptoms of cardiorespiratory compression such as positional dyspnoea, orthopnoea, stridor, syncope, and superior vena cava syndrome (SVCS), administration of GA may be fatal.[4-6] The profound hypoxia may also be due to compression of great vessels in the presence of a patent airway.[1,7,8] In high risk patients with a mediastinal mass, irreversible cardiorespiratory collapse can occur with the use of sedative premedication, induction of anaesthesia, with the use of muscle relaxants,[2] initiation of intermittent positive pressure ventilation (IPPV),[9] by making the patient supine,[1] change of posture,[7] and tumour resection or manipulation.[1,10] It is also possible that tracheobronchomalacia, *i.e.*, softening of tracheal wall, due to prolonged compression by the mediastinal mass, may potentiate the airway collapse with the onset of relaxation produced by anaesthesia or commonly after tracheal extubation or emergence.[2,11] Therefore, airway management in patients with large mediastinal masses with or

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How to cite this article: Shenoy A, Kamath S. Anaesthetic management of a huge mediastinal tumour with tracheobronchial compression. Ind J Resp Care 2013; 2:328-31.

without the evidence of airway obstruction poses a difficult challenge to the anaesthesiologist. An excellent review article by Tempe and Datt details the problems in such patients.[12]

We present here a case of posterior mediastinal tumour with symptoms of airway obstruction.

Case report

A 20 year old man weighing 60 kg presented with history of orthopnoea for 2 months and cough with moderate expectoration for 1 month. Cough was more in the supine position. Sputum was dark brownish in colour. He had no history of haemoptysis, chest pain or palpitations. He had multiple swellings all over the body from the age of 5 years and was diagnosed to have multiple neurofibromatosis.

He was moderately built and nourished but lethargic. His heart rate was 108/min, blood pressure 120/84 mmHg and respiratory rate was 2 L/min. Airway examination was normal except for a slight deviation of the trachea to the left. His oxygen saturation (SpO_2) on room air was 97%. Neck veins were distended and bilateral pedal oedema was present.

He seemed to be breathing with an inspiratory to expiratory ratio of 1:1. There was significant indrawing of the chest on the right hemithorax. Air entry was found diminished on most of the right side of the chest. Chest Xray showed a huge rounded mass in the right hemithorax occupying the upper half of the chest *(Figure 1)*. The entire lower half of the trachea was compressed and deviated to left and the right bronchus was compressed and shifted down. A computed tomogram (CT) of the thorax showed a mass 13.7 x 11.4 x 13 cm in the right hemithorax compressing and deviating the trachea to the left *(Figure 2)*. Contrast CT showed that it was a highly vascular tumour. Compression of superior vena cava (SVC) was evident. Pleural effusion was present bilaterally. Multiple abdominal neurofibromas were present. A CT-guided fine needle aspiration cytology (FNAC) of the chest swelling showed neurofibroma but sarcomatous changes could not be ruled out.

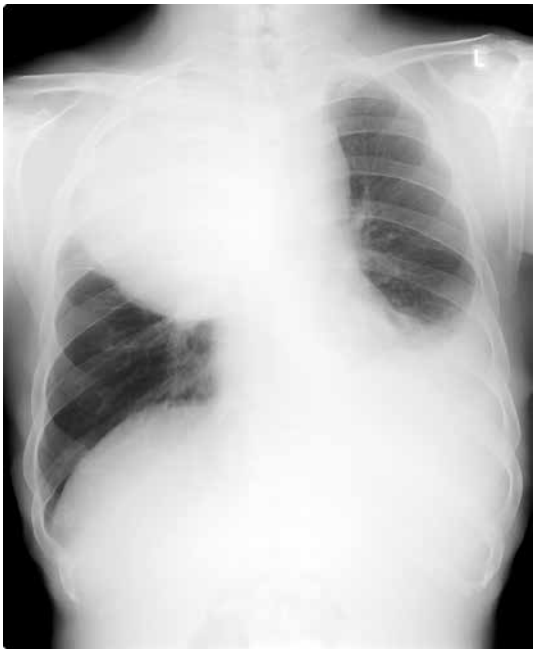

Figure 1: Chest X-ray showing huge mediastinal mass in the right upper zone of the lung

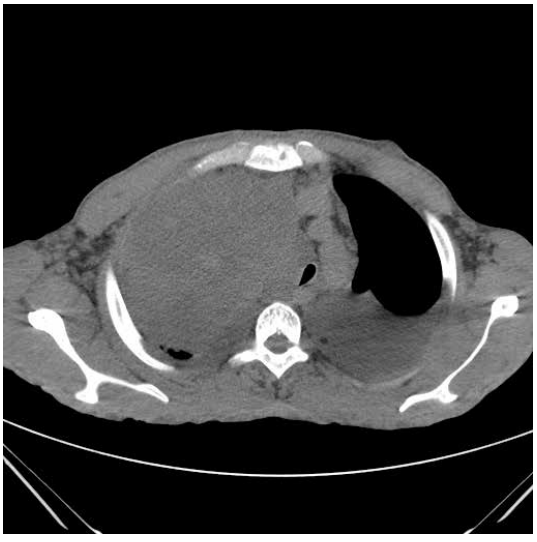

Figure 2: CT scan of the thorax shows the mediastinal mass occupying the entire upper part of the right hemithorax. The trachea is deviated to the left and compressed (the narrowest diameter being 7.9 mm)

CT scan showed distortion of the mid-trachea and a shift to the left side. The narrowest part of the trachea was 7.9 mm in diameter. No difficulty in laryngoscopy was expected. An awake intubation with 5.5 mm internal diameter (ID) flexo-metallic tube (7.5 mm external diameter - ED) was planned. A 5 mmID microlaryngeal tube (6.9 mm ED) and fibreoptic bronchoscope were also kept ready.

As the tracheal diameter was almost 50% obstructed and the patient was orthopnoeic, there was a possibility of losing control of the airway during the course of the anaesthetic. In the event of an emergency, the use of femoro-femoral cardiopulmonary bypass was discussed as an option but later ruled out by the surgeon as the tumour was very vascular and it would have been difficult to control bleeding in a heparinized patient.

No premedication was given. The patient was brought to theatre in sitting position. After securing a 14 G cannula, 4 L/min of oxygen by face mask was commenced. His left radial artery was cannulated. An intercostal drain was inserted under local anaesthesia on the left side to drain pleural effusion. One litre of blood-stained pleural fluid was drained but this gave him only minimal relief of dyspnoea. The airway was anaesthetised with nebulisation with 5 mL of 2% lignocaine and the throat was sprayed with 10% lignocaine. Fentanyl 20 μg and midazolam 0.5 mg were given intravenously to calm him enough to permit a direct laryngoscopy. He was intubated with ease in 80° sitting position with 5.5 size well-lubricated reinforced (flexometallic) endotracheal tube. It was fixed at 26 cm at the lip to ensure that it was beyond the narrowed part of the trachea. Air entry was found to be equal on both sides and end tidal CO_2 confirmed its position. Anaesthesia was induced with sevoflurane in oxygen. After establishing ease of ventilation, he was paralysed with vecuronium. Anaesthetic was maintained with oxygen, air and sevoflurane. Analgesia was provided initially with fentanyl (100 μg) and then with morphine (8 mg).

A triple lumen central venous catheter was inserted in the left internal jugular vein as there were many tortuous superficial veins on the right side. Central venous pressure (CVP) was 26 mm Hg. The right femoral vein was cannulated so as to have a venous access in the inferior vena caval drainage area, in case the SVC had to be clamped. Intermittent positive pressure ventilation (IPPV) was maintained with I:E ratio of 1:1 with a tidal volume of 500 mL. The surgery was allowed to commence after positioning him supine with a mild left tilt. The thorax was opened and the tumour had to be excised piece-meal. Superior vena caval compression got released after tumour excision and the CVP dropped to 11 mm Hg. Blood loss was calculated to be around 4,500 mL. A total of 7 units of packed cells, 4 units of fresh frozen plasma and 5 units of platelets were transfused. The patient was stable haemodynamically and oxygenation was adequate throughout the surgery.

At the end of surgery, the surgeon suspected tracheomalacia. The endotracheal tube was changed to 7.0 mm ID endotracheal tube (Portex) with ease. The patient was electively ventilated overnight and slowly weaned off. In preparation for extubation, the endotracheal tube was withdrawn to the proximal half of the trachea and fixed at 22 cm at the lip and he was kept on continuous positive airway pressure (CPAP) mode of the ventilator. He was observed for two hours for signs of any airway obstruction keeping in mind the possibility of tracheomalacia. As there were no signs of airway obstruction, the patient was extubated successfully.

Discussion

Airway obstruction during induction or maintenance of general anaesthesia is the most feared complication of anaesthesia for excision of mediastinal tumours. Airway obstruction may be symptomatic or asymptomatic. It is advisable to exercise caution even in the asymptomatic patients as it can manifest after induction of general anaesthesia. These airway obstructions can be difficult to manage as they tend to be intrathoracic. An array of equipment such as smaller endotracheal tubes, endobronchial tubes, microlaryngeal tubes (smaller diameter but long with a low pressure-high volume cuff), and rigid and flexible fibreoptic bronchoscopes may be kept readily available. Helium-oxygen mixture may be useful.

When the airway narrowing is more than 50%, it is advised that femoro-femoral cardiopulmonary bypass be kept as a standby. There are several instances in the literature regarding the use of cardiopulmonary bypass to overcome airway obstruction.[4,13-16] Cannulating femoral vessels in an emergency, faced with a hypoxic patient or worse, a patient in cardiac arrest is not an easy option and precious time may pass by and all efforts may turn futile. Thus, it is advisable that the femoral vessels be

cannulated prophylactically under local anaesthesia prior to induction if > 50% airway obstruction is present.

Cannulation of femoral vessels prophylactically would require at least a small dose of heparin and in our patient, the surgeons were concerned about increased bleeding as this tumour was known to be vascular. As expected, the excision was associated with a large amount of blood loss (4,500 mL). It is possible that the bleeding would have been much more, had he been heparinized. We were fortunate that securing the airway at the beginning of surgery and extubation the day after did not pose any major problems and required only basic precautions. The postoperative course was uneventful and the patient was discharged home on the tenth postoperative day. Histopathology showed neurofibroma with sarcomatous changes.

Conclusion

Mediastinal tumours pose special problems to the anaesthetist, the most prominent of which is airway obstruction. The presence of superior vena caval syndrome poses an additional hazard. Appropriate caution includes keeping femoral vessels cannulated for institution of cardiopulmonary bypass (at least as a standby option) in the event of an emergency. If this is agreed upon, it is also advisable to keep blood conservation techniques such as a cell-saver available. There is definitely a higher possibility of a re-exploration in the postoperative period for increased bleeding. Thus, the pros and cons of this technique have to be carefully weighed and a collective decision taken.

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