

# Acute Hypoxaemia and Right Ventricular Compression By A Mediastinal Mass

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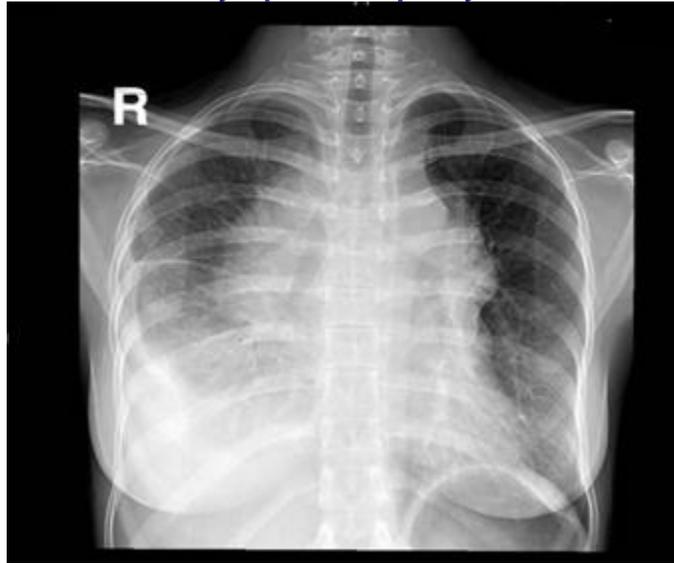
## Abstract

Patients with mediastinal masses rarely face unexpected life threatening airway obstruction perioperatively ( <sup>1</sup> ). With recent awareness of intra-operative management of these patients, major airway obstruction is now occurring more frequently post-operatively ( <sup>2</sup> ). The degree of obstruction is related to the size of mediastinal mass and its extension into the thoracic cavity. Critical narrowing of the airways can occur and severe hypoxemia may supervene which may not respond to conventional therapy and outcome can be fatal ( <sup>3</sup> ). Several modes of management have been suggested in several case reports ( <sup>4</sup> , <sup>5</sup> ). In this case presentation, we describe a life threatening upper airway obstruction in a patient with a large mediastinal mass. Her acute respiratory failure was managed by Pressure Control Ventilation (PCV).

## Case Report

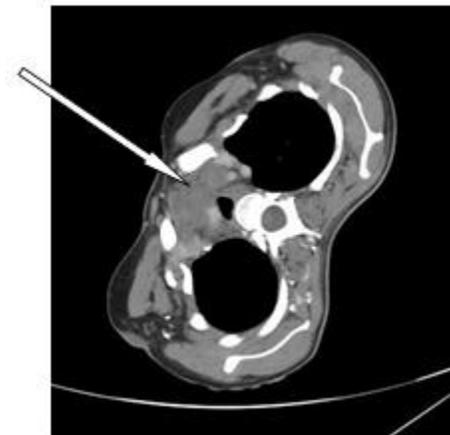
A 19 year old lady presented with a 45 days history of dyspnea and palpitations. The dyspnea was worse in the supine position making her unable to lie flat. She also gave a history of productive cough and hemoptysis for 15 days associated with weight loss of 10 Kg and night sweats. On examination, she was sitting in bed, afebrile, tachypneic at rest (RR-24/mt), neck veins were engorged, no palpable lymphadenopathy or organomegaly were noted. Her heart rate was 110/minute with normal blood pressure (100/60mmHg). Chest exam showed dullness to percussion and diminished breath sounds in the base of the right lung and the heart examination was unremarkable. Apart from high serum lactate dehydrogenase of 756 U/L (normal range 100-190 U/L) all other hematological and biochemical blood results were normal. Alpha –fetoprotein and carcino-embryogenic antigen were unremarkable. Chest radiograph showed a moderately large right sided pleural effusion and anterior mediastinal mass (Fig.1).

**Figure 1: Chest radiograph. Note the bi-hilar and mediastinal lymphadenopathy**



Pleural fluid analysis was consistent with transudate with no malignant cells. CT scan of chest revealed a mass occupying the anterior and superior mediastinum, encompassing the superior vena cava and compressing the lower trachea, right and left main bronchi (Fig.2).

**Figure 2: Arrow indicates compressed trachea by mediastinal mass while the patient on right lateral position.**



Echocardiogram showed dilated right atrium and right ventricle. Both right ventricle and right ventricular outflow tract were compressed.

CT-guided trough-cut biopsy of the mediastinal mass was inconclusive.

Subsequently, the patient underwent left anterior mediastinoscopy under general anaesthesia for mediastinal mass biopsy. The patient had an awake intubation with a reinforced endotracheal tube. Because the patient could not keep the

saturation and oxygenation acceptable, bag ventilation was maintained throughout the procedure. The intra-operative course was uneventful. Post operatively an attempt of extubation failed because of CO<sub>2</sub> retention; hence she was re-intubated and transferred to MICU for mechanical ventilation.

On arrival to the MICU, she was hypotensive and ABG on ambu-bag ventilation with FiO<sub>2</sub>-1 was pH- 7.14; PO<sub>2</sub>-245 mmHg; PCO<sub>2</sub>-66; HCO<sub>3</sub><sup>-</sup>-22; O<sub>2</sub> saturation-99%

IV fluids and dopamine infusion were started immediately and she was started on volume controlled mechanical ventilation (VCV) mode, with tidal volume (Vt) of 400 ml at an inspiratory flow rate of 60L/min, respiratory rate of 14 breaths/min, FiO<sub>2</sub> of 1 and Positive End Expiratory Pressure (PEEP) of 5 cmH<sub>2</sub>O. Unfortunately her oxygen saturation kept on falling, and she remained hypoxemic with exhaled Vt. 160ml. Inverse ratio ventilation with I:E ratio-1:1 was tried with no improvement. Eventually pressure controlled ventilation (PCV) mode with PC of 25cm H<sub>2</sub>O, PEEP of 5 cm H<sub>2</sub>O with inspiratory time (Ti) of 1.0 sec was initiated. Following that her saturation improved and she had no further desaturation. Further attempts at reducing the inspiratory pressure below 25cmH<sub>2</sub>O failed due to desaturation. ABG 30 minutes after PCV mode of ventilation was pH- 7.31; PO<sub>2</sub>-111 mmHg; PCO<sub>2</sub>-48;HCO<sub>3</sub><sup>-</sup>-23; O<sub>2</sub> saturation-98% FiO<sub>2</sub>-1.

Bronchoscopy revealed dynamic tracheal compression during expiration. Histopathology of mediastinal mass confirmed the diagnosis of large B-cell Non-Hodgkin's lymphoma. Chemotherapy with dexamethasone was started. Patient was successfully extubated on day seven and transferred to the general ward. Subsequent echocardiography showed resolution of RV outflow tract obstruction.

## Discussion

In this case report, the trachea, the RV outflow tract and the heart were compressed by an anterior mediastinal mass. The actual incidence of mediastinal compression in adults is difficult to know as most of these cases occurred in pediatric population ( <sup>6</sup> ). During anesthesia, the supine posture accentuates compression of the trachea and adjacent structures (superior vena cava, heart, and pulmonary arteries) due to loss of muscle tone caused by anesthetic agents and elimination of negative intra-pleural pressure ( <sup>7</sup> ). This leads to haemodynamic compromise and blood gas disturbance, mostly due to ventilation perfusion mismatch and changes in lung mechanics ( <sup>8</sup> ). If the patient is unable to lie supine, the anesthetist should anticipate possible airway complications peri-operatively and special techniques for maintaining oxygenation may be warranted ( <sup>4</sup> , <sup>5</sup> )

Our patient had a dilatation of the right heart chambers secondary to RV outflow tract obstruction. Along with the mass effect on the low pressure right ventricle

and RV outflow tract, an increase in intra-thoracic pressure and dynamic hyperinflation induced by positive pressure ventilation created by gas flow obstruction led to a decrease in venous return, which further enhances vascular compression. Animal models of anterior mediastinal mass suggested decrease in cardiac output and RV enlargement due to increase in RV afterload with impingement of the left ventricular distensibility ( <sup>9</sup> ). No clinical study yet has examined the symptoms and signs of right heart obstruction in this population; however, it is imperative not to limit the clinical assessment to tracheo-bronchial tree.

Further, with regard to the ventilatory management of the mediastinal mass it would appear that several modes of therapy have been suggested in the literature including cardio-pulmonary bypass and extra-corporeal membrane oxygenation which were not applied in our case ( <sup>4</sup> , <sup>5</sup> ). However, there are no published comparative series on the merits of various modes of ventilation in such cases. Previous case reports had shown failure of implications of conventional modes of ventilation ( <sup>10</sup> , <sup>11</sup> , <sup>12</sup> ). In this case, PCV was successfully applied after failure of volume controlled ventilation (VCV). In PCV, the preset inspiratory pressure is immediately achieved by initial high inspiratory gas flow which is then maintained through out the inspiratory phase. We postulated that, the high peak inspiratory flow in PCV rendered the already collapsed trachea opened. In contrary, volume is attained by constant inspiratory flow in VCV which may not be high enough to open the already collapsed airways. Therefore, from our limited experience we recommend to try PCV in patients who are unable to maintain their oxygenation due to mediastinal mass before proceeding to other invasive techniques.

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