



Case Report

Dynamic Airway Collapse – A Case Report

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ABSTRACT

The prevalence of obstructive airway diseases in our country is very high. Tracheobronchomalacia and dynamic airway collapse without an antecedent cause in adult population are rare clinical entities. We report one such case which presented as classical obstructive airway disease demonstrating no response to standard therapy thus warranting further investigations.

KEYWORDS: Dynamic airway collapse, Tracheobronchomalacia.

INTRODUCTION

Tracheomalacia (TM) is excessive expiratory airway collapse due to weakness of the airway walls as a result of softening of the tracheal cartilaginous supports and/or redundancy of the posterior tracheal membranous wall[1]. The term tracheobronchomalacia (TBM) refers to excessive collapse of both the trachea and main bronchi and has often been used interchangeably in the literature with TM [1]. Tracheobronchomalacia (TBM) and excessive dynamic airway collapse (EDAC) are both dynamic forms of central airway obstruction characterized by a decrease of $\geq 50\%$ in the cross-sectional area of the tracheobronchial lumen.

CASE REPORT

A 80 year old male patient presented to the emergency medicine department with breathlessness, productive cough and low grade fever since two days. In view of respiratory distress the patient was immediately shifted to the critical care unit. This sudden onset breathlessness, associated with chest tightness and productive cough had progressed from MMRC (Modified Medical Research Council) grade 1 to grade 4 in two days. There was obvious use of accessory muscles of respiration. On auscultation bilateral rhonchi

with right sided coarse crepitations were appreciated., saturation on room air was 84 % with marginal rise to 90% after applying a non rebreathing mask. Patient denied any history of similar episode in the past.

Presence of leucocytosis and features of community acquired pneumonia prompted immediate appropriate antibiotics, blood cultures and sputum cultures were sent. An arterial blood gas revealed hypoxia with hypercarbia with p_{CO_2} of 50 with a p_{aO_2}/f_{iO_2} ratio of 200. Patient was started on bronchodilators in view of the above picture. However in view of lack of response the patient was given a non invasive ventilator trial. Patient did express a subjective improvement after the commencement of the non invasive ventilator support. Blood gases after 1 hour of non invasive support revealed improvement in hypoxia and hypercarbia.

Leucocyte count normalized on day three with procalcitonin demonstrating insignificant values indicating appropriate control of infection. Subsequently the cultures revealed microorganisms sensitive to existing antibiotics. Despite being on maximal support via nebulized as well as injectable bronchodilators, removal of non invasive support even for a few minutes entailed exacerbation of the respiratory distress.

On day 5 a decision was made to do a computed tomography (CT) [Fig: 1] to rule out endobronchial cause of the persistent bronchospasm. The scan revealed bilateral bronchopneumonia along with narrowing of the trachea.

However as no cause was identified for narrowing hence a CT scan [Fig: 2] was repeated in lateral decubitus. Surprisingly the tracheal lumen seemed widened on the lateral decubitus.

Figure: 1 CT scan HRCT showing narrowing of tracheal lumen.

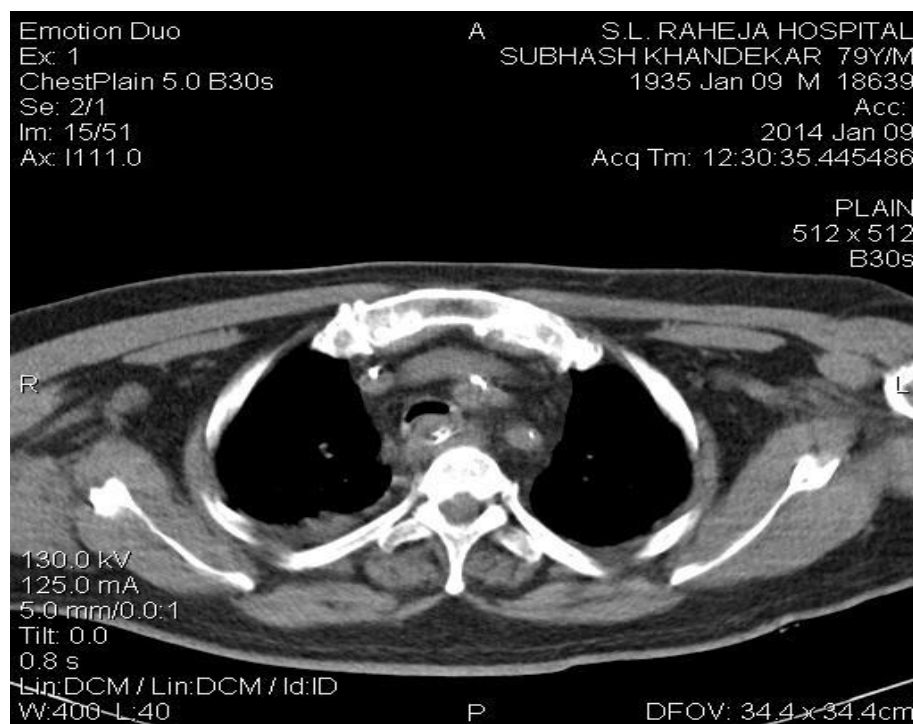
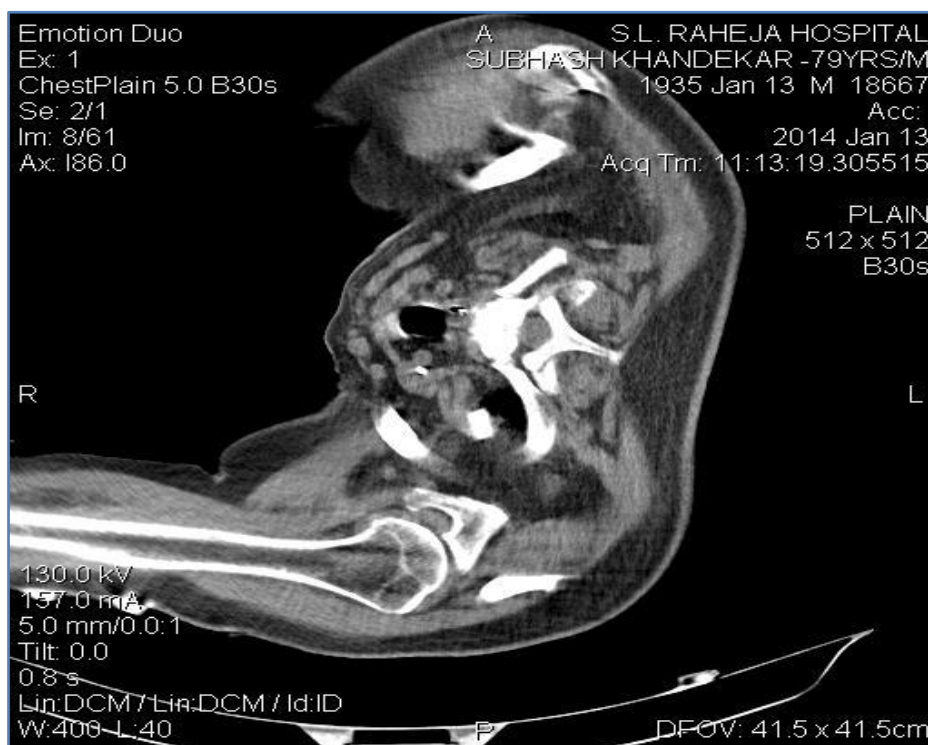


Figure: 2 CT scan HRCT showing no narrowing of tracheal lumen in lateral decubitus.



This dynamic airway collapse was considered as the cause of the respiratory distress and non invasive ventilation was continued. However two days later patient had a respiratory arrest and patient was intubated and put on mechanical ventilation. An emergency bronchoscopy was done which demonstrated collapse of the tracheal wall as well as bronchi during expiration with presence of mucus plugging in bilateral bronchi. As there was active inflammation and involvement of large segment of trachea and bronchi airway stenting was deferred. A tracheostomy was done and patient

DISCUSSION

EDAC is marked by excessive bulging of the posterior tracheal membrane into the airway lumen during exhalation [2]. This condition is unfortunately under diagnosed as such patients present with symptoms very similar to patients with obstructive ventilatory disorders such as asthma and COPD.

The patient described above was an elderly male smoker with previous history of progressive exertional breathlessness resulted in him having being treated along the lines of chronic obstructive pulmonary disease. Compounding the fact further was the absence of a formal pulmonology evaluation like a pulmonary function testing and absence of better imaging modality. The fact that he was not showing a clinical improvement on the standard therapy prompted us to rule out other causes. A CT scan was done primarily to rule out other causes like endobronchial obstruction, however we detected narrowing of the trachea which was absent when the scan was repeated on lateral decubitus. This dynamicity established the diagnosis.

Tracheobronchomalacia in adults goes unrecognized because of associated respiratory illness with overlapping symptoms such as chronic cough and dyspnea, as well as physical and clinical findings such as sputum production, recurrent pulmonary infections, and, rarely, hemoptysis [3]. In both tracheobronchomalacia and COPD, large swings in the intrathoracic pressure may occur as patients work to overcome increased airway resistance due to bronchospasm, mucus plugging, and airway inflammation. Tracheobronchomalacia must be differentiated from difficult-to-control asthma, intraluminal obstruction, foreign body aspiration, and other diseases. One must have a high index of suspicion for the disease. As per a very old article by Gupta et al [4] the uncommon diagnosis of Tracheobronchomalacia may be due to our failure to suspect it.

When first classified radiologically in 1965, Rayl defined airway collapse as abnormal when the cross-sectional area was reduced to at least half its diameter during coughing, as observed on bronchography [5]. Now a days diagnosis is confirmed by dynamic radiologic imaging studies or bronchoscopy which is considered gold standard [6]. Several classification systems exist for tracheomalacia. Tracheomalacia can be classified according to the appearance of the trachea. Patients who have anteroposterior tracheal narrowing are said to have crescent (ie, scabbard shape) tracheomalacia as in this case, while patients who have lateral tracheal narrowing are said to have saber-sheath (ie, fissure shape) tracheomalacia. Tracheomalacia can also be classified according to its distribution, as either segmental or diffuse. This is particularly useful for guiding

therapy. Tracheomalacia can be classified as congenital (ie, primary) or acquired (ie, secondary). Acquired TM is more common than congenital TM in adults.

Dynamic CT with inspiratory and expiratory phases is particularly useful in the assessment of tracheomalacia. Inspiratory CT is unable to make the diagnosis, however a dilated trachea (> 3 cm), especially with posterior bowing of the membranous portion (thus becoming circular) may indicate over compliance of the trachea and thus suggest the diagnosis as seen in our patient. During expiration collapse of the trachea is seen, with bowing of the posterior membranous portion anteriorly, creating a crescent shape in the axial plane. Typically a decrease of the anterior-posterior diameter by 50% or greater is used as a cut-off [7,8].

Our patient was given continuous positive airway pressure (CPAP) as treatment which was not beneficial and owing to the dynamic airway collapse which was generalized permanent tracheostomy was done and patient was on invasive ventilator intermittently even at home. When TBM is incidentally detected on either bronchoscopy or cross-sectional imaging in patients without clinical symptoms, treatment is not indicated. In mildly symptomatic cases of TBM in both children and adults, supportive treatment is usually recommended, including treatment of respiratory infections and concomitant disease such as asthma or COPD, humidified oxygen therapy, and pulmonary physiotherapy.

In more severe cases, where conservative management has been unsuccessful, treatment options include continuous positive airway pressure (CPAP), stenting, and tracheoplasty. CPAP functions as a pneumatic stent by altering expiratory flow and increasing lung volume, thus maintaining airway patency. CPAP is used in both children and adults with TBM, but there are no controlled trials to prove a lasting clinical improvement using the therapy. It is not thought of as a curative therapy but rather as a "bridge" to more definitive management. Surgical therapy is indicated when conservative measures fail. The indications for tracheostomy are severe symptoms, failure of conservative therapy, and proximal or diffuse tracheomalacia. Surgical techniques include aortopexy, tracheopexy, tracheobronchoplasty, and tracheostomy. In distal tracheomalacia that is idiopathic, pulsatile, associated with tracheoesophageal fistula, or from vascular anomalies, aortopexy with concomitant intraoperative bronchoscopy appears to be the procedure of choice. In proximal or diffuse tracheomalacia, tracheostomy or the use of stents is beneficial. [9,10].

CONCLUSION

Diagnostic studies to pin point the exact cause of airway obstruction should be encouraged in case of failure to reasonably respond to maximal pharmacological broncho dilatation. Physicians should keep the diagnosis of tracheomalacia or EDAC in mind.

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