

# Acute Progressive Dyspnea in a Patient With Chronic Obstructive Pulmonary Disease

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## ABSTRACT

The cause of acute dyspnea in chronic obstructive pulmonary disease is challenging. A 69-year old man complained of dyspnea, and cough. He had a diagnosis of chronic obstructive pulmonary disease for 6 years. Oxygen therapy started for respiratory failure. At the 6th day of hospitalization, dyspnea progressively increased. Chest X-ray showed a right-sided radiolucency. The preliminary diagnosis was spontaneous pneumothorax but chest computerized tomography-scan demonstrated a space-occupying lesion in left main bronchus. The latter diagnosis was mucoid impaction but bronchoscopy revealed a malignant lesion. In conclusions, lung cancer can be a rare cause of acute dyspnea.

**Keywords:** Dyspnea, respiratory insufficiency, pulmonary disease, chronic obstructive

## 1. INTRODUCTION

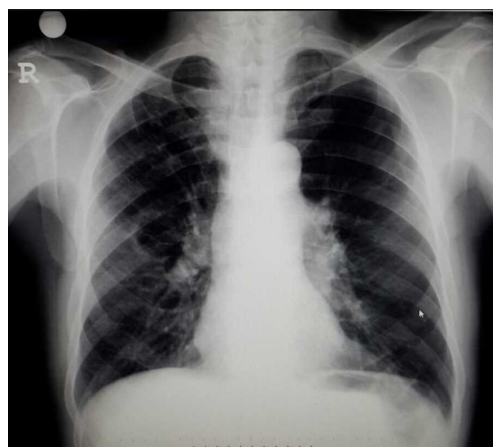
The many causes of dyspnea make it a diagnostic challenge. Rapid evaluation and diagnosis are crucial for reducing mortality especially in the patients with respiratory failure. The most common underlying causes for the exacerbation of chronic obstructive pulmonary disease are pneumonia, pneumothorax, pulmonary embolism, cardiac diseases such as cardiac arrhythmia, ischemia, and heart failure (1). In this educative case, we present another cause of acute progressive dyspnea in a patient with chronic obstructive pulmonary disease. The clinical picture was associated with right-sided radiolucency and left hilar enlargement.

## 2. CASE PRESENTATION

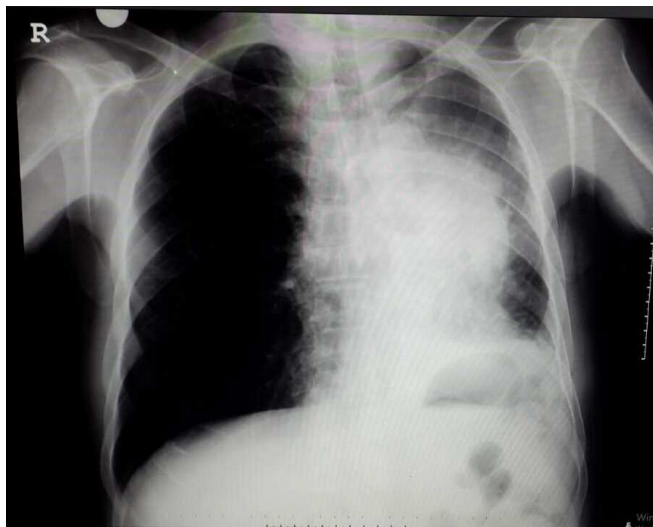
A 69-year old man was hospitalized for chronic obstructive pulmonary disease. Pulmonary function tests showed a forced vital capacity (FVC) of 1680 mL (48%), forced expiratory volume in 1 second (FEV1) of 1060 mL (39%), and FEV1/FVC of 63%. Arterial blood gas analyses were as follows: PaCO<sub>2</sub>: 44 mmHg, PaO<sub>2</sub>: 53 mmHg, pH: 7.48, bicarbonate: 31.8 mmol/L. Chest x-ray showed left hilar enlargement, a small cardiothoracic ratio, and prominent bronchovascular markings in the right lung (Figure 1). Oxygen therapy (2-3 L/min) was given by a nasal cannula. The patient was treated with ampicillin+sulbactam, nebulized salbutamol and ipratropium, methylprednisolone (40 mg/day), and ranitidine.

On the 6<sup>th</sup> day of hospitalization, the patient suffered from acute dyspnea under oxygen therapy. Pulse was 124/min, blood pressure was 100/70 mmHg, and SpO<sub>2</sub> decreased to 85%. A chest x-ray demonstrated total radiolucency in the right

hemithorax (Figure 2). This picture was strongly suggestive of pneumothorax. However, the *tracheal air column* was seen to have shifted to the left and elevated the hemidiaphragm in the left-side. These findings indicated a volume loss due to atelectasis. On the other hand, a progression in the left hilar region was also observed. A chest computerized tomography (CT) was performed for the underlying mechanisms. Chest CT-scan revealed an obstructive lesion in the left main bronchus, left upper lobe collapse, minimal pneumothorax on the left-side, and compensatory herniation of the right lung (Figure 3). This picture strongly suggested mucoid impaction but fiberoptic bronchoscopy showed a malignant lesion (Figure 4). Bronchoscopic biopsy demonstrated malignant epithelial tumor. The patient underwent external beam radiotherapy for total atelectasis of the left lung.



**Figure 1.** Chest x-ray showing a non-homogenous opacity in the right middle zone, and the left hilar enlargement



**Figure 2.** A total radiolucency in the right hemithorax, progression of the left hilar opacity, and volume loss in the left hemithorax



**Figure 3.** A space occupying lesion in the left main bronchus, and a minimal partial pneumothorax in the left side



**Figure 4.** A malignant endobronchial tumor in the left main bronchus

### 3. DISCUSSION AND CONCLUSION

Acute dyspnea in patients with known chronic obstructive pulmonary disease (COPD) can be a clinical challenge due to the nonspecific nature of atypical presentations. Acute progressive dyspnea, right-sided radiolucency, mediastinal shift, and left-sided volume loss displayed by our patient might have been due to tension pneumothorax but *the patient was not hypotensive* and it was necessary to explain the left hilar mass. The frequency of pneumothorax in COPD was reported as 8.5% (2). There were only two case reports about tension pneumothorax in COPD (3,4). In our patient, volume loss in the left lung was caused by compensatory over-inflation and hyperlucency in the right hemithorax. Minimal pneumothorax in the left side may be due to acute check valve bronchial obstruction.

Radiolucency and acute progressive dyspnea in a patient with COPD may be due to ruptured aortic or pulmonary aneurysm (5), late-onset atelectasis after abdominal surgery (6), diaphragmatic hernia with sepsis (7), congenital lobar emphysema (8). Also, aspergillus tracheobronchitis (9), pulmonary capillary hemangiomatosis (10), hemophagocytic syndrome (11), and hepatocellular carcinoma (12) were reported as other causes of acute dyspnea in patients with COPD but radiolucency did not accompany with these diseases.

In conclusion, dyspnea has a broad differential diagnosis even in patients with known pre-existing COPD. Our patient was unique because there was not another report of acute progressive dyspnea presenting with left hilar tumor and right-sided radiolucency. Because hyperlucency can be primary or secondary, it is necessary to look counter side before correct diagnosis. It should be kept in mind that lung cancer can be a rare cause of acute progressive dyspnea in the intensive care setting.

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