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**Hidden biases in clinical decision-making: potential solutions, challenges, and perspectives**

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## Clinical bias case report

A 70-year-old male was referred to the hospital by his primary care physician for worsening known chronic obstructive pulmonary disease (COPD, 15 years since diagnosis) and Chronic Respiratory Failure (CRF) with long oxygen therapy (LTOT) and nocturnal non-invasive ventilation (NIV). He reported that shortness of breath had been increasing for three days. The patient reported panic, dyspnea, and generalized weakness. Furthermore, he added that “this is similar to what was observed in the past during worsening episodes of COPD”. The patient had also a history of hypertension and atrial fibrillation. SABA was added to triple LABA+ ICS + LAMA and oral steroids as oral antibiotics were added by general practitioners and confirmed by a specialist.

The leading symptoms in this patient were dyspnea and generalized weakness. The first consideration in this patient is a COPD relapse. This diagnosis is supported by a progressive worsening of symptoms over three days and the general loss of strength. An important consideration is to establish if an infection is present. Pneumonia could be a complication of COPD. Clinical examination should focus on evaluating for typical signs of COPD. Workup should also consider other causes of acute dyspnea. Oxygen saturation was low instead of 3 liters of O<sub>2</sub>, heart rate was increased. The electrocardiogram (ECG) was normal as were basic laboratory studies on admission (blood count, electrolytes, C-reactive protein, creatine kinase, liver enzymes, creatinine). Initial blood gas analysis showed hypoxia (pO<sub>2</sub>: 50.0 mmHg) and normocapnia (pCO<sub>2</sub>: 42.6 mmHg) with otherwise normal results (pH: 7.47, Bases excess: 0.9 mmol/L, HCO<sub>3</sub><sup>-</sup>: 25 mmol/L). This was interpreted as hyperventilation compensating for hypoxemia.

There is no clear evidence of infection. However, other cardiac and pulmonary causes of dyspnea have not yet been ruled out, and further examination should focus on common cardiopulmonary etiologies such as congestive heart failure, pneumonia, acute coronary syndromes, and pulmonary embolism. The patient did not present clinical improvement. He presented another acute worsening of dyspnea accompanied by pleuritic chest pain. Revision of clinical history and failure to respond to treatment should broaden the range of differential diagnoses. Finally, D-dimer was requested and shows elevated values at 2413 ng/mL. Arterial blood gas analysis revealed normal but reduced pCO<sub>2</sub> of 40.2 mmHg with constant hypoxemia under 40% of FiO<sub>2</sub>. A chest radiograph

was normal. Under the suspicion of vascular cause to explain the clinical situation, left proximal and distal deep vein thromboses were detected by ultrasound.

Pulmonary embolism was finally suspected and computed tomography of the chest with contrast was requested: extensive segmental and sub-segmental pulmonary emboli were revealed. Enoxaparin was started with progressive and constant clinical improvement.