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Pneumonia with Hypercapnia in a Patient with COPD and Infected Bronchiectasis: A Case Report

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Abstract: Chronic obstructive pulmonary disease is a major global health problem with high morbidity and mortality, often complicated by acute exacerbations triggered by lower respiratory tract infections such as pneumonia. The coexistence of pneumonia, chronic obstructive pulmonary disease, and bronchiectasis creates a complex pathophysiological interaction that may lead to impaired gas exchange and hypercapnic respiratory failure. We report a case of a patient with severe chronic obstructive pulmonary disease and infected bronchiectasis presenting with pneumonia and hypercapnia. The diagnosis was established based on clinical findings, radiological imaging, and laboratory examination including arterial blood gas analysis. The patient developed type II respiratory failure characterized by carbon dioxide retention and respiratory acidosis. Management included controlled oxygen therapy, appropriate antibiotic administration, bronchodilator therapy, and supportive care. Careful oxygen titration was essential to avoid worsening hypercapnia. The patient showed clinical improvement with integrated management. This case highlights the importance of understanding the interaction between pneumonia, chronic obstructive pulmonary disease, and bronchiectasis, as well as the need for appropriate oxygen strategy in managing hypercapnic respiratory failure.

Keywords: Pneumonia; Hypercapnia; Chronic obstructive pulmonary disease; Bronchiectasis; Respiratory failure

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Introduction

Chronic Obstructive Pulmonary Disease (COPD) is one of the leading causes of global morbidity and mortality, with an increasing death rate and currently ranked as the third leading cause of death worldwide (Budianti, 2025; Miravitlles *et al*., 2023). This disease is characterized by persistent and progressive airflow limitation, accompanied by structural lung changes such as emphysema, which can be evaluated through imaging modalities (Rodrigues *et al*., 2021; Martini & Frauenfelder, 2020). COPD is frequently associated with acute exacerbations, most commonly triggered by lower respiratory tract infections, particularly community-acquired pneumonia (CAP) (Oh, 2023; Torres *et al*., 2021; Khan *et al*., 2025). The occurrence of pneumonia in COPD patients is associated with worse clinical outcomes, including increased risk of hospitalization, respiratory failure, and mortality. These outcomes can be assessed using severity scoring systems such as CURB-65 and the Pneumonia Severity Index (PSI) (Carlos *et al*., 2023; Kim *et al*., 2019).

The interaction between COPD and pneumonia leads to complex gas exchange impairment due to ventilation-perfusion (V/Q) mismatch, increased airway resistance, and mucus accumulation. (Long *et al*.,

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2022; Rodrigues *et al*., 2021). This condition may be further aggravated by the presence of bronchiectasis, a structural abnormality characterized by permanent bronchial dilation that serves as a reservoir for chronic infection (Hariyanto & Hasan, 2016; Chotirmall & Chalmers, 2018). Bronchiectasis contributes to persistent bacterial colonization and prolongs inflammatory processes, thereby worsening airway obstruction (O'Donnell, 2018). The coexistence of these three conditions creates a significant pathological burden on the respiratory system.

One of the most serious complications is type II (hypercapnic) respiratory failure, defined by an elevated arterial partial pressure of carbon dioxide ($PaCO_2$ >45 mmHg) accompanied by respiratory acidosis (Davidson *et al*., 2016). In COPD patients, hypercapnia is typically caused by alveolar hypoventilation, increased physiological dead space, and respiratory muscle fatigue (Csoma *et al*., 2022; Shiraishi *et al*., 2023). This condition is commonly observed during acute exacerbations and is associated with increased risk of intensive care admission and mortality (Akbaş & Güneş, 2023; Ullah *et al*., 2024; Chung *et al*., 2024). Additionally, hypercapnia at hospital admission is recognized as a predictor of poor clinical outcomes, including death (Vonderbank *et al*., 2020; Nin *et al*., 2017).

The management of this condition poses a significant clinical challenge, particularly in the administration of oxygen therapy, as uncontrolled oxygen supplementation may exacerbate carbon dioxide retention through mechanisms such as the Haldane effect and the loss of hypoxic pulmonary vasoconstriction (Sarkar *et al*., 2022). Therefore, ventilatory management in hypercapnic respiratory failure must be conducted cautiously in accordance with established clinical guidelines (Davidson *et al*., 2016). In addition, newer exacerbation classification approaches, such as the Rome proposal, provide valuable support in severity stratification and therapeutic decision-making (Oh *et al*., 2023; Reumkens *et al*., 2023; Zeng *et al*., 2024).

In the elderly population, pneumonia in patients with COPD becomes increasingly complex due to the presence of comorbidities and age-related decline in physiological function, thereby requiring a comprehensive management approach (Yatera & Yamasaki, 2025). Therefore, a thorough understanding of the interaction between COPD, pneumonia, and bronchiectasis, as well as appropriate management strategies, is essential to prevent clinical deterioration. This case report aims to describe the clinical manifestations, pathophysiological mechanisms, and management approach in a patient with pneumonia accompanied by hypercapnia in COPD with infected bronchiectasis.

Case Illustration

An 82-year-old female presented to the emergency department with a chief complaint of worsening shortness of breath one day prior to admission, with a history of progressively worsening dyspnea over the past three months that increasingly interfered with daily activities. The symptoms were accompanied by a productive cough with yellowish-green sputum for one week prior to admission, along with a history of recurrent chronic cough. The patient also reported a history of fever one week prior and decreased appetite. The patient had a history of smoking for approximately 30 years and had quit smoking 10 years prior to admission, with a moderate Brinkman index. The patient had a history of smoking for approximately 30 years before quitting in 2006, with an estimated Brinkman Index of 300.

On initial physical examination, the patient appeared weak with a decreased level of consciousness (GCS E2V2M4), tachypnea (28 breaths/min), tachycardia (104 beats/min), and severe hypoxemia with an oxygen saturation of 56% on room air, which improved to 99% with a non-rebreathing mask at 10 L/min. Lung examination revealed asymmetric chest wall movement with reduced expansion on the right side and bilateral crackles without wheezing.

Laboratory findings showed leukocytosis (17,920/µL) with neutrophil predominance (95%) and thrombocytosis. Arterial blood gas analysis on admission revealed severe acidemia (pH 7.19) with marked hypercapnia (PCO_2 78.0 mmHg) and elevated HCO_3^- (29.4 mmol/L), consistent with respiratory acidosis and type II respiratory failure. Subsequent arterial blood gas analysis on February 25, 2026 showed persistent acidemia (pH 7.19), PCO_2 59.0 mmHg, PO_2 105.0 mmHg, and HCO_3^- 21.9 mmol/L. On February 27, 2026, the pH and PCO_2 improved to 7.43 and 45.0 mmHg, respectively, although hypoxemia persisted with a PO_2 of 60.0 mmHg, consistent with type II respiratory failure with carbon dioxide retention. Chest radiography showed hyperaeration with bilateral patchy infiltrates (Figure 1), while high-resolution computed tomography (HRCT) of the thorax revealed bilateral bronchiectasis with surrounding infiltrates and multifocal consolidation, consistent with pneumonia in infected bronchiectasis. Thoracic CT was not performed during hospitalization because the diagnosis and management were initially based on the patient's clinical presentation, chest radiographic findings, and other supporting examinations. Given the patient's acute clinical condition, CT was not considered essential for immediate management. Therefore, the diagnosis of bronchiectasis in this case was based on the available clinical and radiological findings, with the limitation that bronchiectasis could not be definitively confirmed

by CT. Spirometry before bronchodilator administration showed an FVC of 30% predicted, FEV1 of 26% predicted, and an FEV1/FVC ratio of 66.67%, indicating severe restrictive and obstructive ventilatory impairment. Post-bronchodilator spirometry showed an FVC of 40% predicted, FEV1 of 32% predicted, and an FEV1/FVC ratio of 61.45%. No significant bronchodilator response was observed. Overall, the findings were consistent with mixed ventilatory impairment and supported the diagnosis of chronic obstructive pulmonary disease (COPD). The patient was diagnosed with severe community-acquired pneumonia (PSI score 132, risk class V), infected bronchiectasis, acute exacerbation of COPD, and type II respiratory failure with hypercapnia.

Management included controlled oxygen therapy targeting oxygen saturation of 90–94%, intravenous antibiotics (ceftazidime and levofloxacin), nebulized bronchodilators, corticosteroids, and supportive therapy including nutritional support and close monitoring. Serial arterial blood gas evaluation showed gradual improvement, with a decrease in pCO_2 to 45 mmHg and normalization of pH to 7.43 by the third day of hospitalization. Clinically, the patient showed improvement in dyspnea, level of consciousness, and overall condition.

The patient was managed using a multidisciplinary approach involving specialists in neurology, pulmonology, and clinical nutrition. The neurology team contributed to the evaluation and management of impaired consciousness suggestive of hypoxic encephalopathy secondary to hypoxemia and hypercapnia. The pulmonology team played a central role in establishing the diagnosis and managing the underlying respiratory conditions, including pneumonia, infected bronchiectasis, and acute exacerbation of chronic obstructive pulmonary disease, while also performing close monitoring of type II respiratory failure and implementing controlled oxygen therapy strategies. Meanwhile, the clinical nutrition team was responsible for assessing the patient's nutritional status, which indicated severe protein–energy malnutrition, and for planning appropriate nutritional interventions to support recovery and improve overall clinical outcomes.

Figure 1. Chest radiographic findings

Discussion

The diagnosis of community-acquired pneumonia (CAP) in this patient was established based on a combination of clinical, laboratory, and radiological findings consistent with the guidelines of the Indonesian Society of Respirology and the American Thoracic Society/Infectious Diseases Society of America, including acute respiratory symptoms such as dyspnea and productive cough, the presence of pulmonary infiltrates on imaging, and leukocytosis as a marker of systemic inflammation (Torres *et al.*, 2021; Khan *et al.*, 2025). Interestingly, fever was not observed at presentation, representing an atypical manifestation commonly seen in the geriatric population. This can be explained by immunosenescence, an age-related decline in immune system function, including reduced T-lymphocyte activity and decreased production of proinflammatory cytokines such as interleukin-1, interleukin-6, and tumor necrosis factor-alpha, resulting in a blunted febrile response (Yatera & Yamasaki, 2025; Long *et al.*, 2022). Therefore, in elderly patients, a high index of suspicion for infection should be maintained even in the absence of fever, particularly when there is deterioration in respiratory function or mental status.

The assessment of pneumonia severity in this patient demonstrated differing results between the CURB-65 score and the Pneumonia Severity Index (PSI). A CURB-65 score in the moderate to high category provides an initial estimate of the need for hospitalization; however, it has limitations in capturing the complexity of patient comorbidities (Carlos *et al.*, 2023). In contrast, a PSI score of 132 (risk class V) more comprehensively reflects a high risk of mortality, as it incorporates factors such as age, altered mental status, and laboratory parameters including acidosis (Kim *et al.*, 2019). This finding emphasizes that, in geriatric patients with multiple comorbidities, the PSI is more strongly recommended than CURB-65 for determining disease severity and the need for intensive care.

In addition to pneumonia, the patient had severe chronic obstructive pulmonary disease (COPD), which served as a major predisposing factor for infection and clinical deterioration. A long history of smoking contributed to chronic airway inflammation, bronchial epithelial damage, and impairment of mucociliary defense mechanisms (Rodrigues *et al.*, 2021; Budianti, 2025). Spirometry findings showing a post-bronchodilator FEV_1/FVC ratio of <0.70 with an FEV_1 of approximately 32% of predicted confirmed severe irreversible airflow obstruction, consistent with the Global Initiative for Chronic Obstructive Lung Disease classification (Miravitlles *et al.*, 2023). In this condition, pulmonary functional reserve is markedly limited, making acute infections such as pneumonia capable of easily triggering severe exacerbations and respiratory decompensation (Oh, 2023).

The presence of infected bronchiectasis in this patient further aggravated the clinical condition. Bronchiectasis is characterized by permanent bronchial dilation leading to mucus accumulation, impaired mucociliary clearance, and chronic bacterial colonization (Hariyanto & Hasan, 2016; Chotirmall & Chalmers, 2018). This creates a vicious cycle of recurrent infection and inflammation, ultimately worsening airway obstruction and increasing the risk of secondary infections (O'Donnell, 2018). From a physiological perspective, this condition also increases ventilatory dead space and exacerbates ventilation–perfusion (V/Q) mismatch, thereby contributing to hypoxemia and hypercapnia (Martini & Frauenfelder, 2020).

The main complication in this case was type II (hypercapnic) respiratory failure, characterized by elevated $PaCO_2$ and respiratory acidosis. The pathophysiology of this condition is multifactorial and can be explained by the concept of a "double-hit" phenomenon. In COPD, increased dead space results from alveolar destruction, while pneumonia causes alveolar consolidation and reduced effective ventilation. Concomitant bronchiectasis further worsens the condition through mucus retention and airway obstruction (Csoma *et al.*, 2022). This combination significantly increases the work of breathing, eventually leading to respiratory muscle fatigue and alveolar hypoventilation (Shiraishi *et al.*, 2023). In addition, severe ventilation–perfusion mismatch results in intrapulmonary shunting, leading to inadequate oxygenation despite oxygen supplementation (Chung *et al.*, 2024).

In this context, oxygen therapy becomes a crucial aspect of management. Uncontrolled oxygen administration in patients with chronic CO_2 retention may lead to oxygen-induced hypercapnia. The primary mechanisms include the Haldane effect, in which reduced hemoglobin affinity for carbon dioxide increases CO_2 release into the plasma, as well as the loss of hypoxic pulmonary vasoconstriction, resulting in the redistribution of blood flow to poorly ventilated lung regions (Sarkar *et al.*, 2022; Nin *et al.*, 2017). Therefore, international guidelines recommend more conservative oxygen saturation targets to prevent worsening hypercapnia (Davidson *et al.*, 2016).

Antibiotic management in this patient consisted of a combination of ceftazidime and levofloxacin, providing broad-spectrum coverage against Gram-negative bacteria and atypical pathogens. In patients with severe pneumonia and comorbid chronic lung disease, there is an increased risk of infection with Gram-negative organisms such as *Pseudomonas aeruginosa*, making the use of broad-spectrum antibiotics a rational choice (Khan *et al.*, 2025). In addition, bronchodilator therapy administered via nebulization with a combination of short-acting beta-agonists and short-acting muscarinic antagonists remains the mainstay of treatment in severe exacerbations (Oh, 2023).

Another important aspect is the patient's nutritional status. In this case, severe protein–energy malnutrition was identified, which may impair immune function and delay the healing process. Malnutrition in COPD patients has been associated with increased mortality, reduced respiratory muscle strength, and a higher risk of infection (Akbaş & Güneş, 2023; Ullah *et al.*, 2024). Furthermore, hypercapnia itself is associated with poorer long-term outcomes, including increased mortality (Vonderbank *et al.*, 2020).

Moreover, newer exacerbation classification approaches, such as the Rome proposal, aid in severity stratification and therapeutic decision-making (Oh, 2023; Reumkens *et al.*, 2023; Zeng *et al.*, 2024). The clinical improvement observed in this patient, as evidenced by the gradual normalization of arterial blood gas parameters and improvement in respiratory symptoms and level of consciousness, demonstrates that a multidisciplinary approach and appropriate management can lead to favorable outcomes even in severe clinical conditions. This case highlights the importance of early recognition of atypical pneumonia manifestations in the elderly, understanding the complex interaction between COPD, bronchiectasis, and infection, and implementing appropriate therapeutic strategies to prevent life-threatening complications.

Conclusion

This case highlights the complex interaction between severe COPD, pneumonia, and infected bronchiectasis, which contributed to type II hypercapnic respiratory failure in an elderly patient. Careful assessment of disease severity and controlled oxygen therapy, combined with appropriate antibiotic and bronchodilator treatment, resulted in improvement of

arterial blood gas parameters and clinical condition. Early recognition of hypercapnic respiratory failure and individualized management are essential to prevent further respiratory deterioration in elderly patients with chronic pulmonary disease.

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