International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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ARTICLE TITLE	MANAGEMENT OF A 71-YEAR-OLD PATIENT WITH COPD EXACERBATION, BRONCHIECTASIS, AND PSEUDOMONAS AERUGINOSA COLONIZATION – CASE REPORT
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4873
RECEIVED	08 October 2025
ACCEPTED	16 December 2025
PUBLISHED	23 December 2025
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MANAGEMENT OF A 71-YEAR-OLD PATIENT WITH COPD EXACERBATION, BRONCHIECTASIS, AND PSEUDOMONAS AERUGINOSA COLONIZATION – CASE REPORT

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ABSTRACT

Chronic Obstructive Pulmonary Disease (COPD) frequently coexists with non-cystic fibrosis bronchiectasis (NCFB), creating a clinical overlap associated with persistent airway infection, frequent exacerbations, and accelerated functional decline. Among pathogens, Pseudomonas aeruginosa colonization confers a particularly unfavorable prognosis. We report a 71-year-old male admitted with severe dyspnea and productive cough with purulent sputum. Baseline arterial blood gases confirmed type II (hypercapnic) respiratory failure. Chest radiography excluded new focal consolidations. Bronchoscopy revealed abundant purulent secretions; bronchoaspirate culture grew P. aeruginosa susceptible to meropenem and colistin. In line with current practice and GOLD 2025 principles for acute exacerbation management, the patient received targeted antimicrobial therapy, short-acting bronchodilators, a short course of systemic corticosteroids, long-term oxygen therapy (LTOT), and daily airway clearance. The hospital course was favorable with $PaCO_2$ decreasing and marked symptomatic improvement; at three months he re-presented with recurrent purulent cough and again had P. aeruginosa in culture, prompting extended inhaled colistin and consideration of prophylactic macrolide therapy. This case underscores practical aspects of COPD–bronchiectasis overlap, highlights decision points around eradication strategies for P. aeruginosa, and illustrates the role of individualized, multidisciplinary follow-up aligned with GOLD 2025 and ERS guidance.

KEYWORDS

COPD, Bronchiectasis, Pseudomonas Aeruginosa, Exacerbation

CITATION

Wiktor Golus, Katarzyna Andrzejewska, Paweł Edyko, Bartosz Golis, Zuzanna Przybyła, Hubert Woźniak, Anna Gęsińska, Krystian Czyżykowski, Alicja Babula, Helena Szelka (2025) Management of a 71-Year-Old Patient With COPD Exacerbation, Bronchiectasis, and *Pseudomonas Aeruginosa* Colonization – Case Report. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4873

Background

COPD is a leading cause of morbidity and mortality worldwide. Exacerbations drive the disease trajectory by accelerating lung function decline, impairing quality of life, and increasing mortality risk [1, 7]. The GOLD 2025 report places renewed emphasis on preventing and promptly treating exacerbations, refining the role of dual bronchodilation, selective use of inhaled corticosteroids (ICS) based on blood eosinophils and exacerbation history, and appropriately timed non-invasive ventilation (NIV) in hypercapnic respiratory failure [1, 7]. Non-cystic fibrosis bronchiectasis (NCFB) commonly coexists with COPD, a phenotype often termed COPD–bronchiectasis overlap. Patients in this overlap have chronic mucus hypersecretion, frequent infections, and persistent airway inflammation [6, 8, 9]. Colonization by *Pseudomonas aeruginosa* is a key adverse prognostic factor, associated with more frequent exacerbations, hospitalizations, accelerated FEV_1 decline, and higher mortality [2, 3, 6]. Current European Respiratory Society (ERS) guidance recommends considering eradication strategies and long-term inhaled antibiotics for chronic *Pseudomonas* infection in bronchiectasis, alongside airway clearance and pulmonary rehabilitation [2, 6]. This report presents a real-world scenario integrating these principles: the diagnostic reasoning used to exclude pneumonia, the initiation of targeted anti-pseudomonal therapy, the supportive measures (oxygen, airway clearance), and the longitudinal plan to reduce future exacerbation risk.

Case Presentation

Patient history: A 71-year-old man (BMI ~26 kg/m²) was admitted to a Pulmonology and Allergology Department with dyspnea at minimal exertion (mMRC grade 4) and persistent productive cough with large amounts of purulent sputum. He denied fever, hemoptysis, or pleuritic chest pain.

Past medical history: Long-standing COPD and bronchiectasis (NCFB) under outpatient follow-up, chronic *Pseudomonas aeruginosa* airway colonization, and chronic respiratory failure. He had multiple prior

hospitalizations for exacerbations. Long-term therapy included inhaled bronchodilators (LABA+LAMA+ICS, specifically indacaterol/mometasone in recent months), intermittent nebulized anti-pseudomonal antibiotics (colistin and gentamicin per prior protocols), and daily airway clearance. Long term oxygen therapy (LTOT) had been initiated two weeks before admission. Other comorbidities included arterial hypertension and heart failure. He was a former smoker (30 pack-years), abstinent for 20 years.

Objective findings: The patient appeared dyspneic but afebrile. BMI was about 26 kg/m². Lung auscultation revealed bilateral wheezes and coarse crackles. Heart rate was 103 bpm, blood pressure 150/84 mmHg, and baseline oxygen saturation was 82%. No peripheral edema was noted.

Laboratory findings: Selected results are summarized below (values at admission):

- WBC: 12.3 × 10^9/L (neutrophils 76.9%, lymphocytes 12.3%)
- CRP: 19.7 mg/L
- pH 7.42; $PaCO_2$ 50.8 mmHg; PaO_2 46.1 mmHg; HCO_3^- 32.8 mmol/L; O_2 saturation 82.2%

These results indicate type II (hypercapnic) respiratory failure in the context of an acute deterioration [1].

Chest radiography: showed no new focal consolidations, arguing against community-acquired pneumonia. This was clinically important given the purulent sputum and auscultatory crackles.

Other findings: Flexible bronchoscopy revealed hyperemic, edematous mucosa and abundant purulent secretions; bronchial toilet was performed. Bronchoaspirate culture grew *Pseudomonas aeruginosa* susceptible to meropenem and colistin, consistent with chronic colonization/reactivation [2, 6].

Diagnosis

- Acute exacerbation of COPD (severe dyspnea, increased sputum purulence and volume, and hypercapnic respiratory failure), Group E [1]
- Bronchiectasis (NCFB) with chronic *P. aeruginosa* colonization [2, 6]
- Exclusion of new-onset pneumonia by chest X-ray and absence of systemic features (e.g., fever)
- Type II respiratory failure

This constellation is characteristic of COPD–bronchiectasis overlap with an infection-driven exacerbation in a previously colonized airway [6, 8, 9].

Management and Immediate Course

Antimicrobial therapy (targeted)

Empirical community-acquired pneumonia therapy was not initiated because imaging and clinical features did not support pneumonia. Instead, targeted anti-pseudomonal therapy was started based on culture and prior history:

- Meropenem 1 g IV every 8 hours,
- Colistin 3 million IU IV three times daily, plus nebulized colistin 3 million IU twice daily (eradication intent and high airway concentrations).

This approach aligns with ERS guidance for bronchiectasis with *Pseudomonas*, where combined systemic and inhaled therapy is often used in eradication attempts or during severe, culture-guided exacerbations [2, 6].

Anti-inflammatory and bronchodilator therapy

- Systemic corticosteroid: oral methylprednisolone 30 mg/day (administered in two divided doses: 24 mg + 8 mg) for a short course, then tapered off;
- Short-acting bronchodilators: ipratropium 0.5 mg + salbutamol 2.5 mg via nebulization four times daily;
- Maintenance inhaled therapy: indacaterol/mometasone as previously prescribed (the patient had been using this at home); the maintenance regimen was continued and to be re-evaluated at follow-up (considering dual bronchodilation and ICS indications per GOLD 2025) [1].

Supportive treatment:

- LTOT via nasal cannula 2–3 L/min with a target saturation of 88–92% [1];
- Daily airway clearance with the Simeox device and supervised breathing exercises [2, 3, 6];
- Usual cardiovascular medications were continued (nebivolol, losartan, low-dose aspirin; LMWH for thromboprophylaxis; trimetazidine per cardiology).

Clinical response

Over the hospitalization, dyspnea, sputum volume, and auscultatory abnormalities improved. Acid-base balance normalized toward the patient's baseline with $PaCO_2$ decreasing to 45 mmHg. Given stabilization on LTOT and pharmacotherapy, NIV was not required during this admission. After approximately two weeks, the patient was discharged in good general condition with instructions to continue nebulized colistin to complete a 3-month eradication attempt, maintain his inhaled bronchodilator regimen, and continue daily airway clearance and vaccination updates [1–3, 6].

Follow-Up and Recurrence

At approximately three months, despite good adherence, the patient reported recurrent purulent cough and increased dyspnea. Repeat airway clearance and bronchoscopy were undertaken; sputum/bronchoaspirate again yielded *P. aeruginosa*. The decision was to extend inhaled colistin for an additional period and to consider adjunctive long-term macrolide prophylaxis (e.g., azithromycin 250 mg once daily or 500 mg three times weekly), particularly suitable for former smokers with frequent exacerbations despite optimized inhaled therapy [1, 4, 6]. In line with COPD strategies for frequent exacerbators, roflumilast could be considered if FEV_1 <50% predicted and chronic bronchitis phenotype is present [1]. The plan also emphasized pulmonary rehabilitation, rigorous airway clearance, and vaccination (influenza, pneumococcal, COVID-19, and pertussis booster as indicated) [1–3].

Discussion

The coexistence of COPD and bronchiectasis is clinically important. Impaired mucociliary clearance, structural airway changes, and a biofilm-prone microbiome favor persistent infection and recurrent exacerbations [6, 8, 9]. Patients with this overlap have higher bacterial loads and a predisposition to *P. aeruginosa* colonization, which is linked to greater symptom burden and worse outcomes [2, 3, 6]. Recognizing this phenotype is crucial because management extends beyond standard COPD algorithms, incorporating bronchiectasis-specific measures (airway clearance, culture-guided antibiotics, and—in selected cases—long-term inhaled antibiotics) [2, 3, 6]. *P. aeruginosa* is adept at forming biofilms and developing antimicrobial resistance. In bronchiectasis, ERS guidelines support eradication attempts at first isolation and long-term inhaled antibiotic therapy (e.g., colistin) in patients with chronic *Pseudomonas* infection and frequent exacerbations [2, 6]. Combining a short systemic course (guided by susceptibility) with inhaled therapy enhances airway drug concentrations and may improve symptom control and reduce exacerbation frequency [2, 6]. In our patient, meropenem + colistin (IV) plus inhaled colistin aligned with the culture and was clinically effective—though recurrence at three months highlights the difficulty of complete eradication and the need for adjunct strategies (macrolide prophylaxis, rigorous airway clearance, and rehabilitation) [2–4, 6].

While GOLD primarily focuses on COPD, many elements are directly applicable to overlap cases:

- Prompt identification of exacerbation type (infection-related with hypercapnia here), early short-acting bronchodilators, and a short steroid course to hasten recovery [1, 7].
- Maintenance optimization: Dual bronchodilation (LAMA/LABA) as the backbone; ICS consideration guided by blood eosinophils and exacerbation history (balancing pneumonia risk). Our patient had been on LABA/ICS; in frequent exacerbators, escalation to LAMA/LABA (±ICS) should be individualized [1, 7].
- Antibiotics: culture-guided for bacterial exacerbations, with attention to prior colonization history [1–3, 6].
- Oxygen: titrated to 88–92% [1].
- NIV: GOLD emphasizes early NIV in acute hypercapnic respiratory failure not improving with medical therapy; it reduces mortality and length of stay [1, 5, 7]. In this admission, ABG normalized with LTOT and bronchodilators/steroids, so NIV was not required—but it remains a key rescue strategy if $PaCO_2$ remains elevated with acidosis despite initial measures or if there is persistent hypercapnia following the acute episode [5, 7].
- Prevention: vaccination, smoking cessation, pulmonary rehabilitation, and consideration of long-term macrolides in selected frequent exacerbators [1–4].

Macrolide prophylaxis and roflumilast

Azithromycin prophylaxis reduces exacerbation frequency in COPD frequent exacerbators (notably in former smokers) and also shows benefits in bronchiectasis by attenuating airway inflammation and possibly disrupting biofilms [4, 6]. Roflumilast is an option in severe COPD with chronic bronchitis and FEV_1 <50% predicted, particularly when exacerbations persist despite optimal bronchodilation; it is not an antibiotic but an anti-inflammatory PDE-4 inhibitor that lowers exacerbation risk in the appropriate phenotype [1].

Airway clearance and pulmonary rehabilitation

Daily airway clearance techniques (e.g., devices like Simeox, active cycle of breathing techniques, autogenic drainage) are foundational in bronchiectasis. Pulmonary rehabilitation improves exercise capacity, dyspnea, and quality of life, and should be integrated early, especially after an exacerbation [2, 3].

Antimicrobial stewardship and safety

In older, multimorbid patients, antibiotic exposure must balance benefit vs. toxicity (e.g., colistin nephrotoxicity/neurotoxicity). Culture-guided regimens, defined durations, and close biochemical monitoring are essential. The recurrence at three months in our case—despite appropriate therapy—underlines the chronicity of colonization rather than failure of care, and supports a stepwise, multi-modal plan (inhaled antibiotics ± macrolide, plus non-pharmacologic measures) [2, 3, 6].

Conclusions

COPD–bronchiectasis overlap with chronic *Pseudomonas aeruginosa* colonization demands individualized, multidisciplinary care. In the acute setting, culture-guided antibiotics, short-acting bronchodilators, short-course systemic corticosteroids, and appropriately titrated oxygen remain central; NIV should be promptly employed when hypercapnia persists with acidosis despite initial management [1, 5, 7]. For long-term stability, dual bronchodilation, selective ICS, airway clearance, pulmonary rehabilitation, vaccination, and (in frequent exacerbators) macrolide prophylaxis and inhaled anti-pseudomonal therapy can reduce exacerbation frequency. Even when full eradication is elusive, a structured, guideline-aligned plan can improve symptoms, physiology, and quality of life [1–3, 6–8].

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