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Case Report

A Rare Case of Concurrent Nocardiosis and COVID-19 in a Patient with Severe Bronchial Asthma

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Abstract

Nocardiosis, a rare opportunistic infection caused by Nocardia spp., primarily affects immunocompromised individuals. We present a 65-year-old male with severe bronchial asthma and type 2 diabetes mellitus, who presented with recurrent hospitalizations due to persistent fever, progressive dyspnea, and pleuritic chest pain. Despite empiric broad-spectrum antibiotic therapy, diagnostic delays occurred because clinical and radiological features overlapped with those of other pulmonary pathologies. During hospitalization for concurrent COVID-19 infection, Nocardia farcinica was identified in a subsequent bronchial aspirate via modified acid-fast staining and Matrix-Assisted Laser Desorption/Ionization Time-of-Flight Mass Spectrometry (MALDI-TOF MS) analysis. The patient achieved clinical and radiological improvement following targeted antimicrobial therapy (Linezolid + Trimethoprim-Sulfamethoxazole + Amikacin) before discharge. This case highlights the critical importance of considering nocardiosis in patients with chronic respiratory comorbidities, particularly when overlapping infections such as COVID-19 are present, to mitigate diagnostic delays and optimize outcomes.

Key Words: nocardiosis; nocardia farcinica; covid-19; bronchial asthma

Introduction

Nocardiosis is a rare disease caused by aerobic, Gram-positive, weakly acid-fast bacteria from the genus Nocardia [1]. In the US, the estimated annual incidence is between 500 and 1,000 cases, although this depends on the geographical region [2, 3]. The overall average annual age-adjusted hospitalization rate in Western Europe is 0.04 per 100,000 inhabitants: 0.05 per 100,000 in males and 0.03 per 100,000 in females [4]. Nocardia comprises more than 80 species that are present in soil, long-standing dust, stagnant water, and decomposing plants. Human infection can occur through inhalation of the microorganisms or through skin injuries, causing local infections and/or dissemination to other organs via the bloodstream [1, 2, 5]. Among the over 50 potentially pathogenic Nocardia species (which vary by geographic region), N. asteroides, N. brasiliensis, N. farcinica, N. cyriacigeorgica, and N. nova are the most commonly identified causative agents [3-7]. Nocardiosis is usually an opportunistic infection occurring mainly in immunocompromised persons, solid organ transplant recipients, patients with hematological malignancies, and individuals on long-term immunosuppressive therapy [4, 6, 8]. Some reports indicate that COPD, bronchiectasis, alcoholism, and bronchial asthma can also predispose individuals to nocardiosis [9-13]. Nocardia farcinica, due to its higher pathogenicity, is more likely than other Nocardia species to invade even immunocompetent hosts, causing pulmonary and/or systemic infections [14-16].

The identification of Nocardia spp. and appropriate diagnosis can be delayed due to clinicians' unfamiliarity with nocardiosis, non-specific symptoms (e.g., cough, fever, chest pain), radiological mimics (pneumonia, tuberculosis, lung cancer) [4,5,7, 17,18], and difficulties in bacterium culture [1,2]. Mortality rates vary widely (10–60%), influenced by species, infection site (skin, lungs, central nervous system [CNS], disseminated), host immunity, treatment timeliness and appropriateness of antibiotic therapy [2,5–7].

We present a case of a patient with severe bronchial asthma presenting with recurrent fever and worsening dyspnea, requiring multiple hospitalizations prior to definitive diagnosis of N.farcinica infection.

This case report aims to:

1. Describe the diagnostic and therapeutic challenges in a patient with severe bronchial asthma and COVID-19 coinfection later diagnosed with Nocardia farcinica pneumonia.

2. Emphasize the importance of considering nocardiosis in refractory respiratory infections, particularly in patients with chronic lung diseases receiving immunomodulatory therapy.

3. Highlight the role of advanced microbiological techniques (e.g., MALDI-TOF MS) in accelerating diagnosis.

We hypothesize that:

4. Undiagnosed Nocardia farcinica colonization in patients with chronic asthma may be unmasked by COVID-19-induced immune dysregulation and corticosteroid therapy.

5. Delayed recognition of nocardiosis in such cases contributes to prolonged morbidity and necessitates tailored antimicrobial strategies.

Case presentation:

We report a 65-year-old male with severe asthma, nasal polyps and type 2 diabetes mellitus who presented with recurrent hospitalizations over a 9-month period (March 7–December 30, 2024; total hospitalization: 156 days) due to persistent fever (38–39°C), fluctuating productive/dry cough, progressive dyspnea, pleuritic chest pain, and generalized weakness.

Period of hospitalization and location [ ], symptoms	**Abnormal laboratory parameters at the time of hospital admission.**		**at dis-charge**	**Microbiological results**	**Additional key laboratory and diagnostic findings**	**Antibiotic treatment**	**Additional treatment**
07/03-21/03/2024 [1] fever 39.20C productive cough worsening dyspnea, chest pain	CRP (mg/l) Hb (g/dl) WBC (x10^9/l) Neu% Glucose mg/dl	446.1 14.7 14.1 83 261	11.6 17.1 14.4 76 144	Blood culture: negative Sputum culture: negative Urine culture: negative	HS Troponin <10 ng/l pH=7.37, pCO_2=41 mmHg , pO_2=78 mmHg echocardiography: small amount of pericardial fluid, EF=55% abdominal ultrasonography: normal	Ceftriaxone iv. plus Ciprofloxacin i.v.	ICS, LABA, inh.Ach Insulin s.c. Clexane sc Dexamethasone iv 2x4 mg theophiline
06/05-17/05/2024 [2] Fever 39.1^{0}C Productive cough exaggerated dyspnea at rest	CRP (mg/l) Hb (g/dl) WBC (x10^9/l) Neu % Eo % Glucose mg/dl	200.1 14.4 12.3 75 1.1 206	2.05 15.3 8.7 57 1.0 116	Sputum culture: Haemophilus parainfluenzae	FEV1=1.29 l (41% pred.) FEV1/FVC=57% FEV1=1.82 l (59% pred.) FEV1/FVC=64%	Amoxicillin-clavulanic acid i.v.	ICS, LABA, inhAch Insulin s.c. Clexane s.c Dexamethasone iv 2x4 mg montelukast
18/06-02/07/2024 [2] fever 38.5^{0}C productive cough exaggerated dyspnea at rest weakness	CRP (mg/l) Hb (g/dl) WBC (x10^9/l) Neu % Eo % Glucose mg/dl	322.3 13.0 12.1 86 0.1 253	5.2 11.9 10.9 74 0.1 80	anty-Borelia IgG and IgM: negative anty-Mycoplasma IgG, IgM and IgA: negative sputum culture: coagulase negative Stapylococcus, Streptococcus gamma-haemoliticus blood and urine culture: negative	Nasal polyps and sinusitis in CT FEV1=1.19 l (38% pred.) FEV1/FVC=58% pH=7.46, pCO2=32 mmHg, pO2=61 mmHg IgE=68 IU/ml, NTproBNP=93pg/ml antibodies anty-SS-A, anty-Jo-1, anty-SCl-70, anty-dsDNA-screen negative echocardiography: small amount of pericardial fluid and fibrin, EF=58%, mild mitral valvulae insufficiency	Ceftriaxone iv. plus Clarithromycin i.v.	ICS, LABA, inhAch Insulin-Actrapid Clexane sc Dexamethasone iv montelukast
06/08-23/08/2024 [2] fever 38.8^{0}C productive cough worsening dyspnea at rest, weakness, chest pain	CRP (mg/l) Hb (g/dl) WBC (x10^9/l) Neut % Eo% D-dimer (ng/ml) Glucose mg/dl	234.0 11.8 13.5 86 0 7150 122	4.94 13.9 8.3 58 2.6 ND 140	SARS-CoV-2 antigen: negative Virus influaenze A and B: negative	Fiberoptic bronchoscopy: negative FEV1=1.49 l (49% pred.) FEV1/FVC=59% FEV1=1.95 l (64% pred.) FEV1/FVC=69% pH=7.40, pCO2=40 mmHg, pO2=75 mmHg	Ceftazidime i.v.plus Ciprofloxacin Amoxicillin-clavulanic ccidi.v.	ICS, LABA, inhAch Insulin-Actrapid Clexane sc Dexamethasone iv 2x4 mg i.v. montelukast

				Legionella pneumophila antygen: negative Bronchial aspirate: Myc. TB negative, Str. viridans, coagulase negative Staphylococcus Anty-CMV IgG: positive Anty-CMV IgM: negative	pANCA and cANCA: negative ECG: atrium fibrillation 115/min Procalcitonin:0.08 ng/ml		
23/09-10/10/2024 [3] fever 38.6^{0}C dry cough worsening dyspnea	CRP (mg/l) Hb (g/dl) WBC (x10^9/l) Neu % Eo % D-dimer (ng/ml) Glucose mg/dl	458 12.2 18.3 81 0.2 470 94	329 12.7 7.7 91 0.1 1022 62	**SARS-Cov-2 antigen: positive (PCR)** Blood culture: negative Urine culture: negative Sputum: Candida spp, Gram-negative bacteria - single Enterobacteriaceae	angioCT: exclusion of pulmonary embolism, progression but bilateral consolidations, nodules, cavitations antyphospholipid antibody negative Procacitonin:0.45 ng/ml	Ceftriaxone i.v + Levofloxacin; next Ceftazidime i.v. + Fluconazole i.v. + Gentamycin	Oxygen: nasal prongs 4l/min ICS, LABA, inhAch Insulin-Clexane sc Dexamethasone iv 8mg Montelucast
10/10-29/10/2024 [2] pulmonary changes fever, dyspnea at rest	CRP (mg/l) Hb (g/dl) WBC (x10^9/l) Neu % Eo % D-dimer (ng/ml) Glucose mg/dl	342 12.7 7.39 93 0.02 933 168	107 11.4 21.5 86 0.1 ND 112	Antigen Legionella spp- negative Antybodies: IgA and IgG anty-Chlamydia and IgG and IgM anty-Mycoplasma negative Blood culture-negative Culture from bronchial aspirate: **Klebsiella pneumoniae** Myc. Tbc negative	pH=7.47, pCO2=43 mmHg , pO2=55 mmHg NTproBNP=760 pg/ml HIV duo-negative	Meropenem i.v. Gentamycin i.m. Fluconazole i.v. Colistin inh. TMP-SMX i.v.	Oxygen : nasal prongs 4l/min Prednisone 30 mg ICS, LABA, inhAch Insulin-Actrapid Clexane sc Montelucast
29/10-30/10/2024 [4] diagnostic stay for lung biopsy	**Multiplex-PCR** from bronchial aspirate: Acinetobacter baumani compex, Enterobacter cloace complex, Haemophilus inf., Escherich coli, Klebsiella aerogenes, Klebsiella oxytoca, Klebsiella pneumoniae, Moraxella catarrhalis, Proteus spp., Pseudomonas				BF; Transbronchial lung biopsy: specimens taken from left lower lobe- NSIP?/ amorphic masses in alveolar spaces	As above	As above

	aeruginosa, Seratia marcescens, Staphylococcus aureus, Streptococcus pneumoniae, Legionella pneumoniae, Chlamydia pneumoniae, Mycoplasma pneumoniae Human Metapneumo-virus, MERS-CoV-2, virus RSV, virus Influenza A and B: **all negative**						
30/10-03/12/2024 [2] fever 37.8 dyspnea, cough, fatigue	CRP (mg/l) Hb (g/dl) WBC (x109/l) Neu % Eo % Glucose mg/dl	89.9 10.2 16.0 83 0.02 105	92.3 10.3 18.4 70 0,2 77	Bronchial aspirate: **weak fast acid Gram-positive bacteria Nocardia farcinica positive** Bronchial aspirate: Candida crusei, coagulase negative Staphylococcus SARS-Cov-2 antygen: negative	pH=7.42, pCO2=45 mmHg, pO2=61 mmHg procalcytonin=0.17 ng/ml chest CT- partial regression consolidations and pleural fluid IgA,IgM, IgG- normal levels HIV Duo Quick: negative	Combination of TMP-SMX i.v.plus Linezolidi.v. plus Amikacin i.v.	Oxygen: nasal prongs 2l/min Prednisone 30 mg ICS, LABA, inhAch Insulin-Actrapid Clexane, prednisone,
03/12-31/12/2024 [5] continuation of treatment and rehabilitation	CRP (mg/l) Hb (g/dl) WBC (x109/l) Neu % Eo % Glucose mg/dl	66.8 10.4 18.5 79 0.02 79	22.6 9.5 9.7 69 0.03 80	SARS-Cov-2 antygen: negative	Procalcitonin=0.04 ng/ml Antibodies anty- HCV negative	Linezolid and Amikacin gradually discontinued TMP-SMX continuation	ICS, LABA, inhAch Insulin-Actrapid Clexane

Table 1: summarizes the dates and locations of the patient's hospitalizations, symptoms, laboratory and microbiological test results, antibiotic therapy details, and other adjunctive medications administered.

Hospitalization Location: [1]- Department of Internal Medicine at Częstochowa Municipal Integrated Hospital.

[2] Department of Pulmonology at the Virgin Mary Provincial Specialist Hospital in Częstochowa.

[3] Department of Infectious Dieseases at Virgin Mary Provincial Specialist Hospital in Częstochowa.

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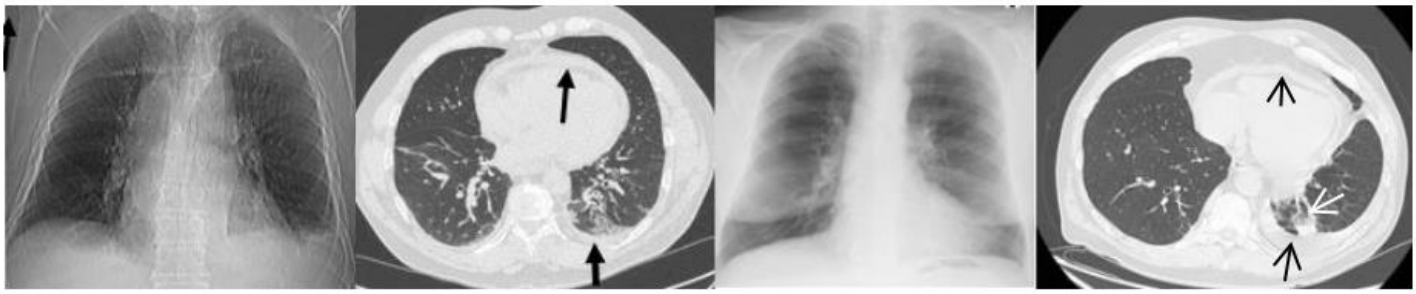
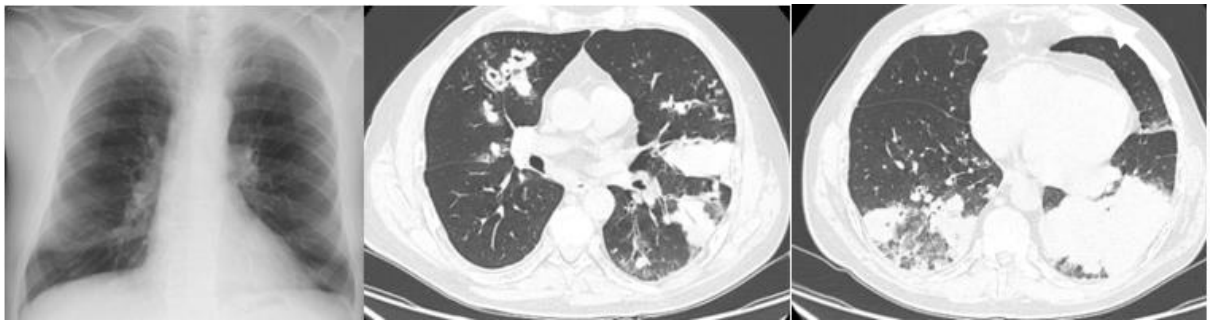


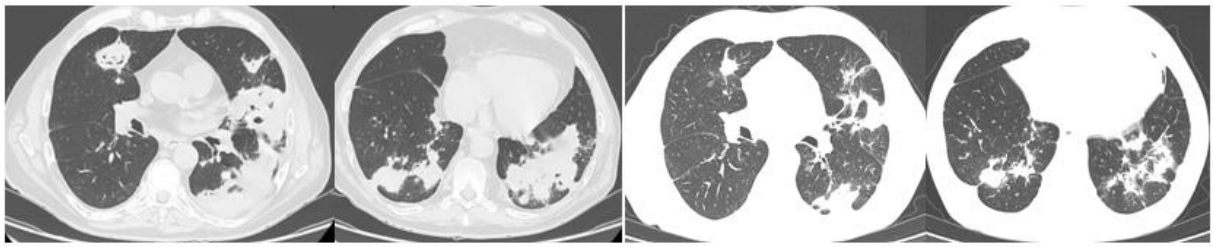
Figure 1: shows chest X-ray images and serial chest computed tomography (CT) scans of the lungs obtained during successive hospitalizations from 7 March to 30 December 2024.

*05.03.2024 The chest CT scan shows a small volume of fluid in the left pleura (separation < 2 cm) and a minimal amount of fluid in the pericardial sac." (arrows). The chest X-ray (P-A view) shows blunting of the costophrenic angle on the left side. 10/05/2024 The chest X-ray shows no significant pathology.19/06/2024 The computed tomography shows persistent fluid in the left pleura and in the pericardial sac (black arrows), as

well as minor infiltrative changes in the left lung (indicated by open white arrows).



22.08.2024 The chest X-ray shows no significant pathology 04.10.2024 CT shows bilateral asymmetric massive consolidative and infiltrative changes, as well as nodules with necrosis, and small areas of ground-glass opacities



*20/11/2024 CT shows persistent consolidative and infiltrative changes with slightly altered localization, but there is visible regression of fluid in the left pleural cavity. 23/12/2024 – CT shows partial regression of the consolidative and infiltrative changes, along with regression of fluid in the left pleural cavity."

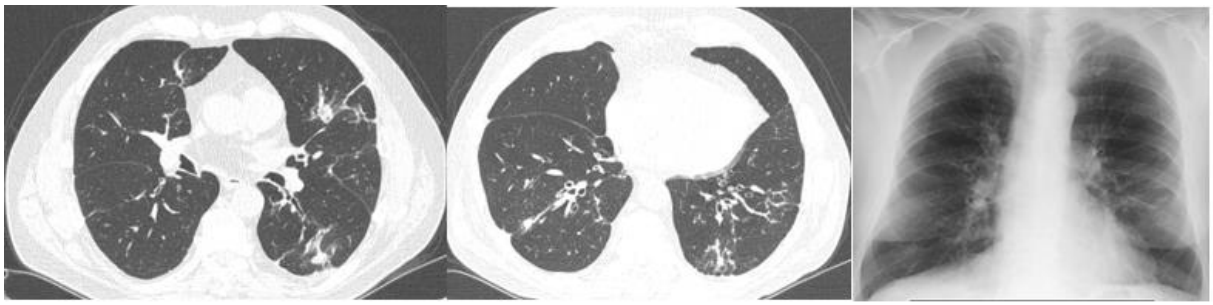


Figure 2: Depicts a chest X-ray image and CT scans performed after discharge in December 2024.

18/02/2025 – CT shows almost complete remission of the lesions: only residual fibrotic changes are visible at the sites of previous consolidations and infiltrates; no cavitation was found. 11/07/2025 The chest X-ray shows no significant pathology

Initial empiric therapy, including broad-spectrum antibiotics, systemic and inhaled corticosteroids (ICS), long-acting beta-agonists (LABA), inhaled anticholinergics (inhACh), short-acting beta-agonists (SABA), and insulin, yielded transient clinical improvement (reduced dyspnea, normalized body temperature, and decreased CRP/WBC levels). Chest computed tomography (CT) on admission (March 2024) demonstrated minimal bilateral pleural and pericardial effusions, which persisted on subsequent imaging. Extensive diagnostic workup excluded connective tissue diseases (lupus, scleroderma, polymyositis), granulomatosis with polyangiitis, malignancies, mycobacterial infections, fungal pathogens, and HIV. On September 29, 2024, the patient returned with acute respiratory failure and tested positive for SARS-CoV-2 antigen (nasopharyngeal swab). Despite treatment with dexamethasone, oxygen therapy, and empiric antibiotics, chest CT revealed bilateral infiltrates, cavitating consolidations, solitary nodules, and ground-glass opacities. CT angiography excluded pulmonary embolism, and antiphospholipid antibodies were negative. Following SARS-CoV-2 clearance, he was transferred to the Pulmonary Department (October 10, 2024), where Klebsiella pneumoniae was isolated from bronchial aspirate but later deemed a colonizer. A transbronchial lung biopsy and repeated

microbiological analyses (October 29, 2024) at the Medical University of Łódź were inconclusive. Escalation to meropenem, colistin, and fluconazole in the Pulmonary Department in Częstochowa provided minimal benefit. Definitive diagnosis was achieved on November 11, 2024, when Gram-positive, branching, partially acid-fast filamentous bacilli were identified in bronchial aspirate via Giemsa and modified Ziehl-Neelsen staining. Matrix-Assisted Laser Desorption/Ionization Time-of-Flight Mass Spectrometry (MALDI-TOF MS; bioMérieux VITEK® MS) confirmed Nocardia farcinica. Metastatic screening (brain CT, abdominal/skin ultrasonography) showed no abscess formation. Antimicrobial therapy was adjusted to intravenous linezolid (600 mg twice daily), trimethoprim-sulfamethoxazole (TMP-SMX; 10 mg/kg trimethoprim component every 8 hours), and amikacin (500 mg twice daily) for synergistic coverage. Steady clinical and radiological improvement permitted sequential de-escalation: amikacin was discontinued first, followed by linezolid. The patient was discharged on December 30, 2024, with instructions to continue oral TMP-SMX (960 mg four times daily) for six months.

Follow-Up and Outcomes:

Post-discharge chest CT (February 18, 2025) revealed near-complete resolution of consolidations, replaced by minor fibrotic changes. At 6-month follow-up (July 10, 2025), the patient remained afebrile with normal inflammatory markers (CRP: 8 mg/L, WBC: 8,8 × 10^3/μL) and organ function (creatinine: 0.9 mg/dL, ALT: 28 U/L, AST: 19 U/L). Arterial blood gas analysis demonstrated adequate oxygenation (pH: 7.45, PaO_2: 77 mmHg, $PaCO_2$: 35 mmHg), and chest X-ray showed no active disease. Spirometry, however, indicated persistent severe obstructive dysfunction (FEV_1: 1.1 L, 30% predicted; FEV_1/FVC: 56%), likely reflecting underlying severe asthma with airway remodeling.

Discussion

To our knowledge, this represents the first documented case of concurrent COVID-19 and N.farcinica coinfection in a patient with severe bronchial asthma, as reported in the English-language literature. The patient's nonspecific presentation—fever, productive cough, and dyspnea—aligns with prior studies describing pulmonary nocardiosis, where overlapping symptoms delay diagnosis in 67–89% of cases [4,6,19]. Chronic respiratory conditions, including asthma, COPD, and bronchiectasis, are well-established risk factors for nocardiosis, with a Western European cohort identifying preexisting lung disease in 58% of cases [4]. In asthma, chronic inflammation drives goblet cell hyperplasia and mucin hypersecretion, impairing broncho-ciliary clearance and fostering bacterial colonization [11]. Concurrent inhaled corticosteroid (ICS) use may further compromise mucosal immunity, as ICS suppresses local neutrophil activity and epithelial defense mechanisms [11,12]. Initial imaging revealed rare manifestations of nocardiosis: minimal pleural and pericardial effusions. Pleural involvement occurs in 10–30% of pulmonary cases [4,6,19], while pericardial effusion—though exceptionally rare—has been associated with life-threatening tamponade, even in immunocompetent hosts [20,21]. Given the scant fluid volume, thoracentesis was deferred; however, fine-needle aspiration with cytochemical staining (e.g., Giemsa, modified Ziehl-Neelsen) could expedite diagnosis in such scenarios achieving sensitivity of 78–92% [22,23]. Notably, conventional sputum or blood cultures are suboptimal for Nocardia detection due to its slow growth (4–6 weeks on aerobic media) and susceptibility to overgrowth by commensals [1,2]. In our case, repeated cultures were discarded prematurely (standard protocol: 2–3 weeks), a practice linked to 31% false-negative rates in patients pretreated with antibiotics [1,7]. Microscopic identification of branching, beaded Gram-positive bacilli warrants advanced diagnostics, such as MALDI-TOF MS, 16S rRNA sequencing, or metagenomic next-generation sequencing (mNGS) [2,24] particularly given that 23% of pulmonary nocardiosis cases involve mixed infections (e.g., Aspergillus fumigatus, cytomegalovirus, Streptococcus pneumoniae) [6]. Post-COVID-19 readmission revealed in our patient radiographic progression with consolidations, cavitations, and nodules—features present in 10–23% of nocardiosis cases [4,17,18]. While similar CT patterns may arise in bacterial (e.g., Klebsiella pneumoniae, Staphylococcus aureus) or fungal (mucormycosis, Pneumocystis jiroveci) superinfections [25], bacterial co-infections remain often underrecognized in COVID-19 affecting 11% of hospitalized and 22.5% of critically ill patients [26]. Although Nocardia coinfection with COVID-19 is exceedingly rare [27–32], SARS-CoV-2-induced immune dysregulation and corticosteroid therapy may unmask latent infection. We hypothesize that undiagnosed N. farcinica colonization can precede COVID-19 infection [27] with transient suppression by broad-spectrum antibiotics (e.g., meropenem) insufficient to eradicate the pathogen. Subsequent dexamethasone administration for COVID-19—a known risk factor for nocardiosis (OR: 4.7) [19]—likely facilitated disease progression. Diagnostic delays spanned 250 days from initial admission, assuming early effusions signaled nocardiosis onset. If COVID-19 triggered symptomatic progression, the delay aligns with reported intervals (17–42 days) [4,8]. Concurrent ICS use during hospitalization aligns with recent reports of rapid Nocardia emergence post-COVID-19 (5–50 days) [33]. Treatment challenges in our patient mirror those in prior cases of asthma-associated nocardiosis [12,13,14], which reported pulmonary consolidations and nodular changes managed with broad-spectrum antibiotics, high-dose inhaled and systemic corticosteroids, long-acting beta-agonists (LABA), short-acting beta-agonists (SABA), inhaled anticholinergics (e.g., tiotropium). Our case confirms that clinicians may initially attribute imaging findings to asthma exacerbations or routine infections, delaying targeted testing (e.g., cultures, biopsies) for Nocardia. Prolonged use of empirical antibiotics (e.g., for bacterial pneumonia) can mask symptoms while allowing nocardial infection to progress. The coinfection of COVID-19 and nocardiosis poses unique diagnostic and therapeutic challenges due to overlapping risk factors, immune dysregulation, and treatment-related complexities, which collectively may contribute to mortality in some cases [30]. Notably, N. farcinica exhibits intrinsic resistance to third-generation cephalosporins, carbapenems, and fluoroquinolones [3], rendering empiric regimens ineffective. Combination therapy with trimethoprim-sulfamethoxazole (TMP-SMX), linezolid, and amikacin—selected based on susceptibility data [3]—achieved clinical resolution, underscoring the importance of tailored prolonged (6-12 months) regimens. Despite N. farcinica's high mortality in immunocompromised hosts (30–40%) [34,35], early appropriate therapy reduces mortality to 5–10% [35]. Six-month oral TMP-SMX maintenance, guided by comorbidities, resulted in near-complete radiological resolution, though persistent spirometric obstruction (FEV_1: 31% predicted) likely reflects irreversible asthma-related remodeling.

The patient's prolonged course may reflect age-related immuno-senescence and metabolic dysregulation (e.g., diabetes), both linked to reduced Sirtuin 1 (SIRT1) activity—a critical regulator of inflammation and cellular stress responses [36]. While we did not assess SIRT1 levels in this case, future studies could explore its role in patients with overlapping infections.

Conclusion

This case underscores the imperative to consider nocardiosis in asthmatic patients on immunomodulators presenting with refractory pneumonia, particularly during COVID-19. Diagnostic delays—exacerbated by overlapping symptoms and empiric antibiotic use—can be mitigated through repeat bronchoscopic sampling and advanced diagnostics (e.g., MALDI-TOF MS). Our findings support the hypothesis that COVID-19-related immune perturbations and corticosteroid therapy may unmask latent Nocardia colonization. Heightened clinical suspicion and prolonged tailored regimens are essential to optimize outcomes in this vulnerable population.

Ethical Statement

The patient provided written informed consent for the publication of this case report. The study protocol and publication were approved by the Ethics Committee of the Virgin Mary Provincial Specialist Hospital in Częstochowa, Poland

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Conflict of Interest

The authors declare no conflict of interest

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