

Pulmonary nocardiosis in West Texas: A case series and literature review

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ABSTRACT

Background: Pulmonary nocardiosis is an uncommon but potentially life-threatening opportunistic infection that often presents with nonspecific clinical and radiographic findings, leading to delayed diagnosis. Although classically associated with immunosuppression, it may also occur in patients with chronic lung disease or other predisposing conditions.

Case Presentations: We describe three cases of pulmonary nocardiosis diagnosed at a tertiary referral center in West Texas. Two patients had systemic lupus erythematosus receiving chronic immunosuppressive therapy, while one had interstitial lung disease treated with high-dose corticosteroids. Clinical presentations included dyspnea, cough, pleuritic chest pain, cavitary pulmonary lesions, empyema, and multifocal consolidations. Initial diagnostic evaluations frequently suggested alternative etiologies, including bacterial pneumonia, mycobacterial infection, or invasive fungal disease. Definitive diagnosis was established by microbiological isolation of *Nocardia* species from empyema fluid or respiratory specimens. Management required prolonged, susceptibility-guided antimicrobial therapy, with some patients requiring surgical intervention or indefinite suppressive treatment because of persistent immunosuppression. All patients demonstrated clinical improvement following targeted therapy.

Discussion: These cases highlight the broad clinical spectrum of pulmonary nocardiosis and reinforce the importance of maintaining a high index of suspicion in immunocompromised patients and individuals with chronic lung disease receiving corticosteroids. The disease frequently mimics tuberculosis, fungal infections, and pulmonary malignancy, making early microbiologic diagnosis essential. Species identification and antimicrobial susceptibility testing are critical to guide individualized therapy and optimize outcomes.

Conclusion: Pulmonary nocardiosis remains a challenging diagnosis because of its heterogeneous presentation and overlap with other pulmonary diseases. Early recognition, prompt microbiological confirmation, evaluation for disseminated infection, and individualized prolonged antimicrobial therapy are essential to improve patient outcomes, particularly in high-risk populations.

Keywords: Nocardia infection, pneumonia, case series

INTRODUCTION

Pulmonary nocardiosis is a rare but potentially life-threatening infection caused by aerobic actinomycetes of the genus *Nocardia*. These Gram-positive,

weakly acid-fast bacteria are ubiquitous in soil and decaying organic matter, and they typically infect immunocompromised individuals, although cases in immunocompetent hosts have also been reported. The infection primarily manifests as a pulmonary disease, often mimicking tuberculosis, fungal infections, or malignancies, which can lead to delays in diagnosis and treatment. Clinical presentation varies widely, ranging from subacute or chronic pneumonia to lung abscesses and cavitary lesions, with the potential for

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dissemination to the central nervous system, skin, and other organs.^{1,2,3}

The diagnosis of pulmonary nocardiosis remains challenging due to its nonspecific clinical and radiological features, as well as the difficulty in isolating and identifying *Nocardia* species in clinical specimens. Advances in molecular techniques, such as 16S rRNA gene sequencing and matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS), have improved diagnostic accuracy. However, a high index of clinical suspicion is essential, particularly in patients with underlying conditions such as HIV/AIDS, chronic lung disease, or long-term corticosteroid use.^{4,5}

Treatment typically involves prolonged antibiotic therapy, often with sulfonamides as the cornerstone, although resistance patterns and the need for tailored regimens based on species identification are increasingly recognized. Despite appropriate therapy, the prognosis of pulmonary nocardiosis remains guarded, especially in cases of disseminated disease or delayed diagnosis.⁶ This paper aims to provide an updated overview of epidemiology, pathogenesis, clinical features, diagnostic approaches, and management strategies for pulmonary nocardiosis, highlighting the importance of early recognition and intervention in improving patient outcomes.

CASE PRESENTATIONS

CASE 1

A 44-year-old woman with past medical history of systemic lupus erythematosus complicated by lupus nephritis and on chronic immunosuppression presented for evaluation of multiple weeks of dyspnea. She had previously been seen in the emergency department and had chest x-rays performed which showed right sided pneumonia. She had further workup performed with computed tomography (CT) of the chest which revealed a right sided loculated pleural effusion and multiple nodular opacities in the right lung of varying size. She was started on broad-spectrum antibiotics with metronidazole and levofloxacin and had a catheter thoracostomy placed. Pleural fluid studies were consistent with empyema with a positive

fluid culture of *Staphylococcus capitis* and a pleural fluid lactate dehydrogenase (LDH) of 653 IU/L. She was initiated on intrapleural fibrinolytics with tissue plasminogen activator (tPA) and dornase. After completing her series of intrapleural fibrinolytics, she had residual empyema and underwent thoracotomy for drainage and subsequent placement of surgical chest tube. Her intraoperative empyema fluid would grow *Nocardia pneumoniae*. Metronidazole and levofloxacin were discontinued and ceftriaxone and amikacin were started. She had a prolonged hospitalization due to delayed recovery from her thoracotomy requiring a second thoracotomy but ultimately stabilized and was able to discharge on minocycline. She has subsequently been followed in the infectious disease clinic and is on indefinite therapy with minocycline due to her immunosuppressed state.

CASE 2

A 63-year-old man with past medical history of interstitial lung disease and pulmonary embolus on apixaban presented for evaluation of right sided pleuritic chest pain, dyspnea, and cough. He had been started on high-dose steroids with 60 mg daily of prednisone for the month prior to presentation and had not been receiving prophylactic antibiotics. A CT of the chest showed right lung infiltrates, a right pleural effusion, and a cavitory lesion in the right upper lobe. The pulmonology service was initially consulted for concern of mycobacterial infection. He subsequently underwent bronchoscopy with a bronchoalveolar lavage (BAL) of the right upper lobe. The BAL fluid studies showed adequate sampling with 50% macrophages, 22% neutrophils, and 23% lymphocytes. He underwent thoracentesis which was exudative by light criteria with a pleural fluid LDH of 577 IU/L and showed a neutrophilic predominance. Culture of the BAL and pleural fluid showed no growth. A sputum culture was obtained the day after bronchoscopy which revealed *Nocardia pneumoniae*. His antibiotics were subsequently switched from cefepime to trimethoprim-sulfamethoxazole. Infectious disease was consulted and he has subsequently been followed in the infectious disease clinic. He required a 6-week course of ceftriaxone after hospital discharge before completing

a 6-month course of trimethoprim–sulfamethoxazole for his nocardia pneumonia.

CASE 3

A 38-year-old woman with past medical history of systemic lupus erythematosus on chronic immunosuppression with hydroxychloroquine, bendamustine, and rituximab; chronic hypoxic respiratory failure as a sequela of COVID; recurrent pneumonia; and pulmonary embolism on apixaban who presented for evaluation of dyspnea and cough. She was hospitalized a week prior to presentation for pneumonia which she was treated with levofloxacin and a day after discharge from that hospitalization she presented for reevaluation due to worsening of her symptoms. A CT scan of the chest revealed multiple bilateral areas of consolidation and cavitations. Workup was initiated with sputum culture, viral respiratory infection testing, and fungal testing. She was started on broad-spectrum antibiotics with linezolid and piperacillin–tazobactam. Fungal testing for *Aspergillus*, *Candida*, *Coccidioides*, histoplasmosis, and *Blastomyces* were negative and viral respiratory infection panels were negative. Sputum culture grew *Nocardia cyriacigeorgica*. Infectious disease was consulted, and her antibiotics were de-escalated to monotherapy with linezolid. An MRI of the brain showed no evidence of central nervous system involvement of her pulmonary nocardiosis. She had a short hospitalization and was discharged on linezolid and followed up in the infectious disease clinic, and in the pulmonology clinic. Susceptibility testing showed sensitivity to trimethoprim–sulfamethoxazole, linezolid, ceftriaxone, and tobramycin. Due to a drug reaction from linezolid, her antibiotic was changed to trimethoprim–sulfamethoxazole. She has had follow-up CT of the chest which has shown near resolution of her bilateral consolidations with some residual cavitations. She was continued on antibiotic therapy indefinitely due to her immunosuppressed state.

DISCUSSION

Nocardia species are usually thought to be opportunists, causing infection mostly in immunocompromised patients. Several large series from tertiary

care centers have shown that approximately 80% of patients were immunocompromised.⁷ A few reports have also noted that immunocompetent patients can develop nocardiosis, most of them with underlying conditions including chronic lung disease, diabetes, and alcoholism.⁸ In an observational study by Steinbrink et al, immunocompromised patients with nocardiosis had a more severe disease with higher mortality.⁹ In case 1, we note that despite the patient being treated with broad spectrum antibiotics, catheter thoracostomy with intrapleural fibrinolytics, the patient still had residual empyema, which required a second procedure, after which *Nocardia* grew on cultures. After tailored antibiotics, the patient was stabilized and ultimately discharged.

Case 2 also reinforces the fact that patients who are immunocompetent but have underlying conditions, including chronic lung disease (interstitial lung disease in this case) have a higher risk for developing nocardia-associated pneumonia with cavitation. The lung remains the primary source of infection in non-immunocompromised patients, described in an observational study from Steinbrink et al.⁹ Historically, sulfonamides have remained the cornerstone of treatment for *Nocardia*, with a duration of treatment typically recommended for six months, to avoid relapses. This recommendation relies on small, non-randomized studies published decades ago.¹⁰

The importance of considering other differential diagnosis is always important. Frequent radiological findings include one or more lung nodules, consolidations and necrotizing granulomas. These findings are usually bilateral.¹¹ Cavitation is reported more frequently in immunocompromised.¹² In case 3 the patient with known immunocompromise due to systemic lupus erythematosus presented with worsening cough despite being previously treated for suspected pneumonia. Imaging studies reported bilateral areas of consolidation and cavitations, which are strongly suggestive of fungal infection. Despite extensive workups for bacterial, viral and fungal infections, they were all negative. Sputum cultures grew *Nocardia* species. The patient was initially treated with linezolid but later transitioned to sulfonamide (trimethoprim–sulfamethoxazole) indefinitely given her immunocompromised state.

CONCLUSION

Our cases highlight the diverse clinical presentations and diagnostic challenges of pulmonary nocardiosis, particularly in patients with immunosuppression or underlying chronic lung disease. Because clinical and radiographic findings frequently mimic other pulmonary infections, maintaining a high index of suspicion is essential to avoid delays in diagnosis and treatment. Early microbiologic identification, evaluation for disseminated disease, and individualized antimicrobial therapy guided by species identification and susceptibility testing are critical for optimizing outcomes, consistent with current expert recommendations.¹³

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