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Case Report

Pulmonary manifestation of a cutaneous pathogen: a fatal report of *Nocardia brasiliensis* pneumonia in elderly with no apparent immunocompromised status

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ABSTRACT

Nocardiosis, a rare but potentially life-threatening opportunistic infection, typically affects immunocompromised individuals. However, this report describes an unusual case of primary pulmonary nocardiosis caused by a cutaneous pathogen-*Nocardia brasiliensis* in a previously healthy, immunocompetent woman. The patient, with a history of chronic obstructive pulmonary disease (COPD), presented with non-specific symptoms including fever, chest pain, productive cough, and progressive dyspnoea, initially leading to a diagnosis of acute exacerbation of COPD with bacterial pneumonia. However, further diagnostic investigation revealed the presence of *Nocardia brasiliensis* in sputum samples, confirmed by Gram staining and modified Ziehl-Neelsen (ZN) staining. Despite appropriate initial clinical suspicion, the patient was not started on targeted therapy with cotrimoxazole in a timely manner. As a result, her condition rapidly worsened, progressing to multi-organ dysfunction syndrome (MODS), and she succumbed to the infection before effective treatment could be initiated. This case highlights two rare findings: the atypical presentation of pulmonary nocardiosis in an immunocompetent host and its rapid progression, emphasizing the critical importance of timely diagnosis and intervention. This case underscores the diagnostic challenges posed by *Nocardia* species, which can mimic other pulmonary infections, such as tuberculosis or fungal pneumonia. Given the increasing incidence of nocardiosis, especially in individuals with chronic lung diseases like COPD, heightened clinical awareness, early microbiological diagnosis, and prompt initiation of appropriate antimicrobial therapy is essential to improving patient outcomes and preventing fatality.

Keywords: *Nocardia brasiliensis*, Pulmonary nocardiosis, Immunocompetent hosts, Diagnostic challenges, Antimicrobial susceptibility, Chronic obstructive pulmonary disease

INTRODUCTION

Nocardiosis, a rare infection, usually affects immunocompromised patients but surprisingly occurs in one-third of cases with normal immune systems, making it difficult to diagnose.[1-3] The clinical manifestations of nocardiosis most frequently localize to pulmonary infection from inhalation or inoculation of *Nocardia* species.[1] These infections prolong hospital stays, increase costs, and can be fatal by mimicking smear-negative tuberculosis, highlighting the need for rapid, reliable testing in vulnerable patients.[4]

CASE REPORT

An apparently healthy, non-diabetic, non-hypertensive woman in her seventies presented with a 10-day history of mild-to-moderate fever and 6 days of non-radiating chest pain worsened by deep inspiration but unaffected by activity. She also had productive cough with copious greyish expectoration for last 6 days and breathing

difficulty for 2 days. There was no hemoptysis, hematemesis, or other comorbidities like steroid use, tuberculosis, smoking, or alcohol consumption. The patient had a past history of mild chronic obstructive airway disease for the last 15 years, for which she was receiving intermittent inhalers of bronchodilators (salbutamol). On clinical examination in OPD, the patient was afebrile, with a pulse of 110 bpm, blood pressure of 105/73 mm Hg, respiratory rate of 20 breaths/minute, and oxygen saturation of 96% on room air. Lung auscultation revealed bilateral basal crepitations. A preliminary diagnosis of acute COPD exacerbation with community-acquired pneumonia was made.

Investigations

Chest X-ray showed bilateral lung opacities, while high-resolution CT revealed peripheral nodules with a feeding vessel sign and cavitary consolidations in left lower lobe.

The complete hemogram showed neutrophilic leukocytosis with a WBC count of 26.88 thousand/µL (94.2% neutrophils). Hepatic and renal function showed modest deviations, while serological tests were negative for HIV, hepatitis B, and hepatitis C antibodies.

On the next day of admission, blood cultures, sputum for direct microscopy and aerobic bacterial culture, and CBNAAT for tuberculosis were sent, all of which were negative. Blood cultures showed no growth after 7 days.

Gram stain of expectorated sputum had an acceptable Bartlett score and showed the presence of filamentous, slender, branching gram-positive bacilli with beaded appearance (Figure 1 A). Subsequently, the bacteria were non-acid-fast on ZN stain with 25% H_2SO_4 as a decolourizer, but acid-fast on modified ZN stain with 1% H_2SO_4 as a decolourizer (Figure 1 B), which was consistent with *Nocardia* species.

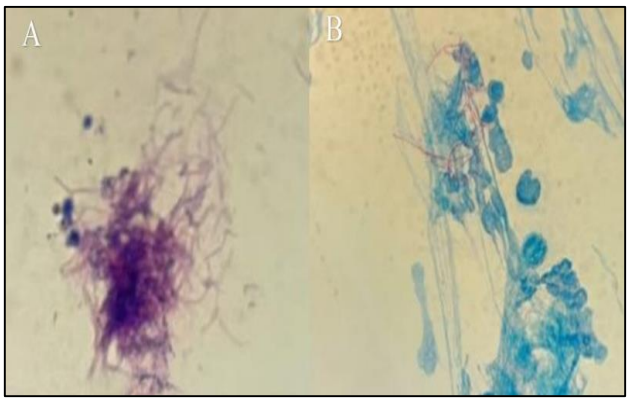

Figure 1: (A) Thin filamentous branching Gram positive bacteria. (B) acid fast bacteria on modified (1%) ZN stain.

The sputum specimen was inoculated onto 5% sheep blood, chocolate, and MacConkey agars and incubated at 37°C. After 48 hours, no growth was seen, but after two more days, white, dry, wrinkled colonies with a rough surface and characteristic potting soil smell appeared on blood and chocolate agars (Figure 2 A and B). Gram staining of isolated colonies showed gram-positive, branching filamentous bacilli that were acid-fast on modified ZN staining, confirming *Nocardia* species. Subcultures on Sabouraud dextrose Agar and Lowenstein Jensen media were performed to observe colony morphology (Figure 2 C and D).

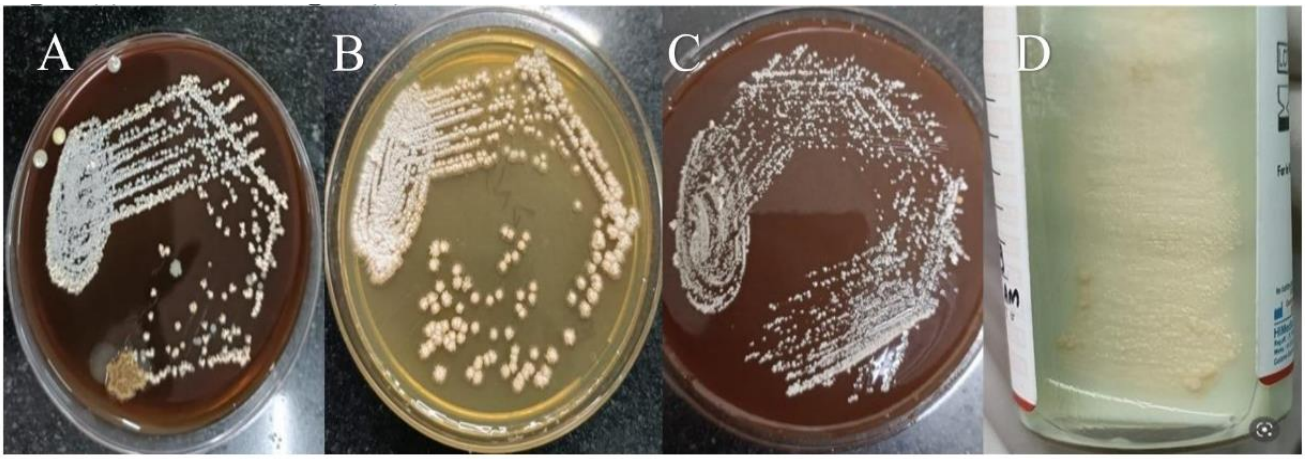

Figure 2: Rough white dry wrinkled colonies on (A) Blood agar, (B) Sabouraud dextrose agar, (C) chocolate agar and (D) Lowenstein Jensen media.

Preliminary colony examination, along with Gram and ZN staining, suggested genus-level identification, followed by biochemical tests to confirm species-level classification of the *Nocardia* isolates. Table 1 outlines the biochemical and antimicrobial susceptibility patterns of Nocardia species, aiding differential identification and highlighting species-specific pathogenic traits.

The isolate's biochemical profile, ascertained through a comprehensive panel of tests, identified it as belonging to

"*Nocardia brasiliensis*" (Figure 3 A). Antimicrobial susceptibility testing by Kirby-Bauer disk diffusion method was performed for various antimicrobials (as mentioned in CLSI M-24 document) on Mueller-Hinton agar (Figure 3 B), though difficult to perform and standardize broth microdilution method is the recommended method for *Nocardia* species.[5] Although standard reference bodies don't specify clinical breakpoints for disk diffusion, all drugs were susceptible based on the following zone sizes-cotrimoxazole (26 mm); amikacin (30 mm); gentamicin (29 mm); ciprofloxacin (40 mm); ceftriaxone (32 mm); cefotaxime (33 mm); cefepime (40 mm); imipenem (36 mm); meropenem (27 mm), except erythromycin (17 mm) being resistant, based on breakpoints mentioned in few studies.[6]

Nonetheless, the susceptibility pattern corroborated with the identification of the isolate as *Nocardia brasiliensis* (Table 1).[6]

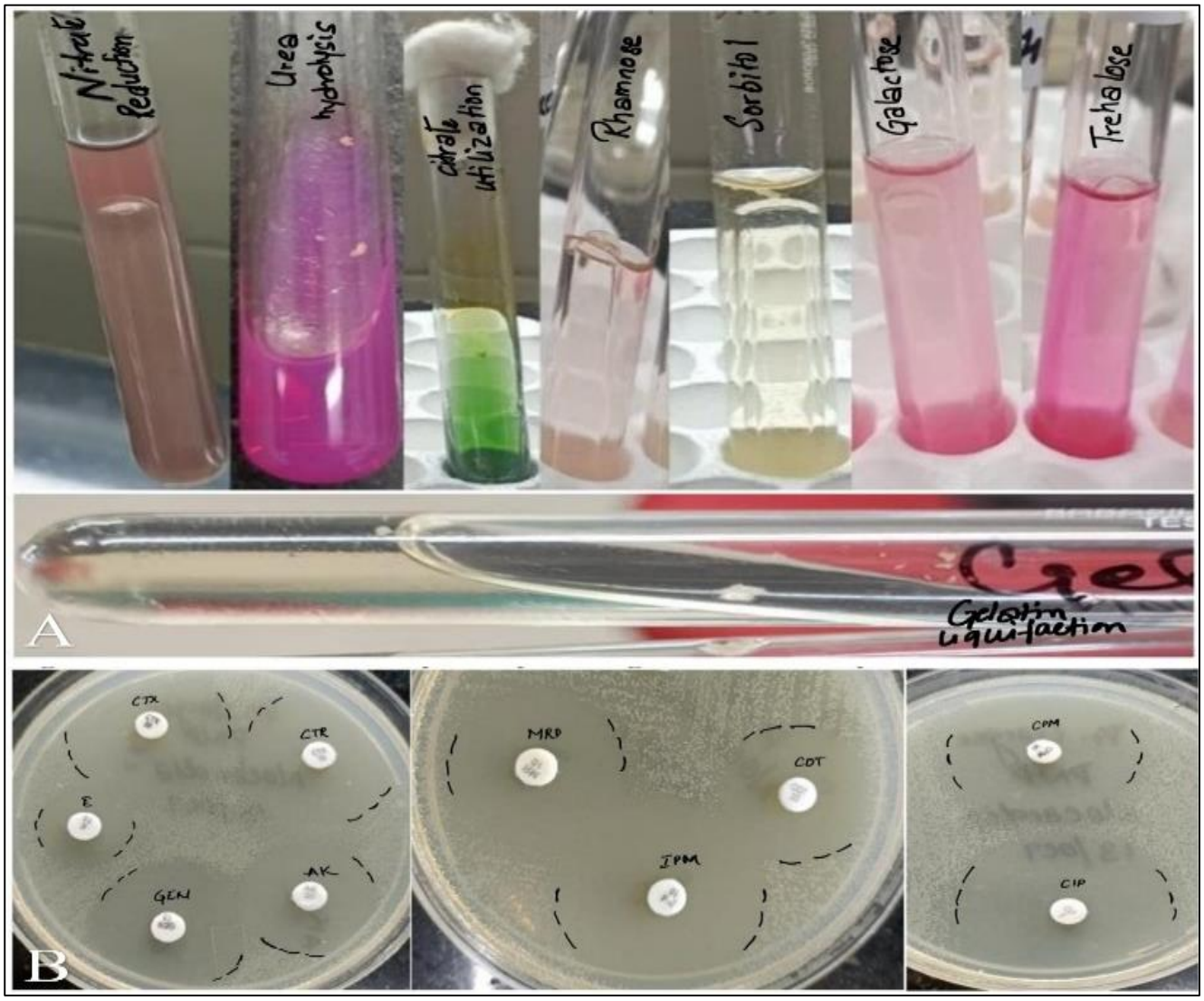

Figure 3: (A) Phenotypic characterisation of *Nocardia brasiliensis, [Nitrate reduction test, Urea hydrolysis test, Citrate utilization test, carbohydrate fermentation test for rhamnose, sorbitol, galactose & trehalose (from left to right); gelatin liquefaction test (below)] and* (B) antimicrobial susceptibility testing for *Nocardia* species.

*Antibiotic disk used- AK-Amikacin (30 µg); CPM-cefepime (30 µg); CTX-cefotaxime (30 µg); CTR-ceftriaxone (30 µg); CIP-ciprofloxacin (5 µg); COT-cotrimoxazole (25 µg); GEN-gentamicin (10 µg); E-erythromycin (15 µg); IPM-Imipenem (10 µg); MRP-meropenem (10 µg).

Differential diagnosis

The initial differential diagnosis included pulmonary tuberculosis, chronic pulmonary infections like mycosis, and primary lung malignancy or secondary metastasis.

Treatment

Empirical therapy was started with intravenous ceftriaxone (1-gram q12h), inhalational bronchodilators, inhalational

steroids (budesonide) and supplemental oxygen via high-flow nasal cannula.

Outcome and follow-up

The suspected *Nocardia* species on direct microscopic examination of expectorated sputum was reported to the clinical team on day 2 of admission. Due to some reason, the patient was not started on cotrimoxazole and remained on ceftriaxone.

Despite resuscitation efforts, the patient's condition rapidly worsened, progressing to MODS, and death occurred on day 3 before cotrimoxazole could be initiated.

Table 1: Various biochemical tests to identify *Nocardia* species.

Variables	Isolates							
	N. asteroides	***N. farcinica***	***N. nova***	***N. transvalensis***	***N. brasiliensis***	***N. otitidi scaviarum***	***N. pseudo brasiliensis***	***N. africana***
Growth at 45˚C[7]	–	+	–	V	–	V	–	+
Nitrate reduction[7]	NA	–	+	+	+	+	–	–
Urea hydrolysis[8]	+	+	+	+	+	+	+	–
Citrate utilization[9]	–	–	–	+	–	–	–	–
Equivalent growth at 35˚/45˚[6]	–	+	–	–	–	–	–	+
Gelatin liquefaction[7]	–	–	–	–	+	–	–	NA
Acid production from								
Rhamnose[8]	–	+	–	–	–	–	–	–
Sorbitol[8]	–	–	–	V	–	–	–	–
Galactose[10]	–	–	–	+	+	–	+	NA
Trehalose[10]	–	–	–	+	+	+	+	NA
Sensitivity by Kirby-Bauer disk diffusion[6]								
Gentamicin (10 µg)	S	R	S/R	R/S	S	S	S	NA
Amikacin (30 µg)	S	S	S	R	S	S	S	NA
Erythromycin (15 µg)	R	R	S	R	R	R	R	NA

DISCUSSION

N. brasiliensis infection is strongly linked to compromised cell-mediated immunity, particularly in HIV patients, bone marrow transplant recipients, and long-term steroid users.[11] In contrast, the patient in our study had no major risk factors, except COPD, deviating from typical pattern of nocardiosis in immunocompromised hosts.[12,13] Factors associated with COPD, such as altered bronchial architecture, steroid use, frequent hospitalizations, antibiotic exposure, and comorbidities, may predispose patients to *Nocardia* infection, as seen in our case with a COPD patient on inhaler therapy.[14,15] Chronic corticosteroid use is a known risk factor for *Nocardia* infections, but our patient with COPD, typically more susceptible, treated with salbutamol instead, presenting a unique case that deviates from the typical risk profile.[16]

Previous literature supports our findings of nocardiosis in immunocompetent hosts, an atypical presentation diverging from the usual association with immunocompromised states.[12] Unlike the typical lymphocutaneous and disseminated presentations of *N. brasiliensis* in immunocompromised individuals, our study describes an unusual case of isolated pulmonary involvement in an immunocompetent host, broadening the pathogen's clinical spectrum.[12,13,17] Pulmonary nocardiosis, a rare but deadly respiratory pathogen, showed high mortality in our case, consistent with documented fatality rates.[16]

Diagnosing pulmonary nocardiosis requires high clinical suspicion and isolation of *Nocardia* species from clinical specimens. While cutaneous infection is common after direct inoculation, inhalational exposure to *N. brasiliensis* has been reported in some cases.[16]

CONCLUSION

Physicians must recognize pulmonary nocardiosis for early diagnosis, as resistance to first-line antibiotics like cotrimoxazole can lead to treatment failure and poor outcomes. Performing antibiotic susceptibility tests on *Nocardia* isolates and early screening is crucial to preventing severe disease.

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