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CASE REPORT

First Documented Case of Pneumonia with *Nocardia africana* and SARS-CoV-2 Co-Detection in Mainland China

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Abstract: *Nocardia* spp. are zoonotic pathogens that can cause infections ranging from localized lesions to systemic dissemination, primarily via pulmonary inhalation or percutaneous inoculation. We report the first confirmed case of *Nocardia africana* pneumonia with SARS-CoV-2 co-detection in mainland China, diagnosed through bronchoalveolar lavage fluid (BALF) analysis using metagenomic next-generation sequencing (mNGS) and matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). A 76-year-old male presented with persistent cough and fever, accompanied by radiographic evidence of progressive pneumonia. Targeted antimicrobial therapy with trimethoprim-sulfamethoxazole and amoxicillin-clavulanate resulted in clinical resolution within 12 days. This case underscores three critical implications for post-COVID-19 pandemic medicine: the requirement for heightened vigilance for opportunistic pathogens (eg, *Nocardia* species) in pneumonia patients with recurrent fever, the essential role of advanced diagnostics (eg, mNGS and MALDI-TOF MS) in identifying fastidious organisms like actinomycetes, and the potential for antimicrobial resistance in *N. africana*, which necessitates susceptibility-guided therapy.

Keywords: pneumonia, *Nocardia africana*, SARS-CoV-2, opportunistic pathogen, MALDI-TOF MS, mNGS

Introduction

Nocardia species are opportunistic pathogens that predominantly affect immunocompromised individuals. Infection primarily manifests as pulmonary disease, cutaneous abscesses, or disseminated disease.[1–11] To date, approximately 54 *Nocardia* species have been identified as human pathogens. Among them, the most clinically significant include *Nocardia asteroides, Nocardia brasiliensis, Nocardia farcinica, Nocardia nova, Nocardia otitidiscaviarum, Nocardia pseudobrasiliensis, Nocardia transvalensis*, and *Nocardia cyriacigeorgica*.[12–18] Diagnostic challenges persist due to nonspecific clinical manifestations, radiographic findings that overlap with other pulmonary conditions, and the low sensitivity of conventional microbiological methods.[19] *Nocardia africana* is a rare species, first isolated in Africa from the sputum of a chronic pneumonia patient who showed progressive pulmonary consolidation on imaging and failed to respond to empirical anti-tuberculosis treatment.[20] Significant knowledge gaps remain regarding its antimicrobial susceptibility profile, virulence factors, and prognostic indicators. Here, we report the first confirmed case of *N. africana* pneumonia in mainland China, accompanied by a comprehensive review of globally reported infections caused by this zoonotic pathogen. Moreover, cases of *Nocardia* infection following or concurrent with SARS-CoV-2 infection are rare, and we will further elaborate on this aspect in the subsequent section.

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Case Presentation

Clinical Feature

A 76-year-old male with a persistent fever (peak temperature of 39 °C) lasting more than seven days was transferred to our infectious disease department for further evaluation and treatment. At the referring hospital, he had received intravenous omadacycline and meropenem as empirical therapy; however, there was no clinical improvement, as evidenced by persistent fever, productive cough, and progressive pulmonary consolidation on chest CT. Physical examination revealed the following: no icterus, petechiae, or lymphadenopathy; neurologically intact; pupils equal and reactive to light; clear bilateral breath sounds without dry or wet rales; regular cardiac rhythm without murmurs; a soft, non-tender abdomen without hepatosplenomegaly; and no cyanosis or edema of the extremities. Notably, the patient had no history of long-term antibiotic use, chronic respiratory symptoms, or underlying comorbidities. The patient's history of COVID-19 vaccination and the date of the initial infection are unknown. But he had a significant 50-year smoking history (approximately 30 cigarettes per day), quitting one year prior. Biomarkers of systemic inflammation were significantly elevated, with CRP measuring 110.3 mg/L and ESR at 87 mm/h, both exceeding standard reference intervals (Table 1). Empirical therapy with Sulperazon (cefoperazone-sulbactam) 2 g intravenously every eight hours was initiated but failed to resolve the fever or improve clinical symptoms. On hospital day 4, bronchoscopy was performed, revealing findings accompanied by copious purulent exudate adhering to the luminal surface (Figure 1).

Etiological Examination

The metagenomic next-generation sequencing (mNGS) results of the patient's bronchoalveolar lavage fluid (BALF) specimen revealed: *Nocardia* spp. (2185.19 RPM) and COVID-19 (34.11 RPM). Microscopic findings were as follows: Neutrophils were predominant among nucleated cells, with purple, branched, elongated hyphae observed under Wright-Giemsa staining (Figure 2A); Acid-fast staining showed red bacterial filaments (Figure 2B), while Gram staining revealed Gram-positive bacteria displaying beaded, branching filaments (Figure 2C), morphologically consistent with *Nocardia* spp. After 3 days of incubation, yellow-white, dry, wrinkled colonies were observed on the blood agar plate, with a transparent hemolytic ring at the base of the agar (Figure 2D–F). Matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS) identified the pathogen as *Nocardia africana* with 99.9% confidence (Figure 2G). Under the combined application of multiple technologies, we identified the pathogenic bacteria. The antibiotic susceptibility results are summarized in Table 2. All tested isolates were susceptible to all antibiotics included in the panel.

Table 1 Hematological Examination Results of the Patient

Laboratory Indicators	Before Treatment	After Treatment (TMP-SMX + AMC)	Reference Range
WBC	6.91×10^9/L	5.40×10^9/L	$3.50–9.50\times10^9$/L
CRP	110.3 mg/L	18.2 mg/L	≤10.0 mg/L
Percentage of neutrophils	89.3%	86.2%	40-75%
ESR	87 mm/h	/	0-15 mm/h
Serum PCT	0.95 ng/mL	0.08 ng/mL	≤0.25ng/mL
Serum total protein	59.2 g/L	61.1 g/L	65.0–85.0 g/L
Serum albumin	26.8 g/L	34.1 g/L	40.0–55.0 g/L
Serum GPT	86 U/L	29 U/L	9-50 U/L
Serum ferritin	>2000 μg/L	/	21.8–274.7 μg/L
Serum ANA	Positive	/	Negative
Plasma fibrinogen	6.19 g/L	/	2.00–4.00 g/L
Plasma D-Dimer	2820 μg/L	/	≤550.0 μg/L
Plasma FDP	9900 μg/L	/	≤5000.0 μg/L
Plasma antithrombin III	64.9%	/	72.0–120.0%

Abbreviations: WBC, white blood cell count; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; PCT, procalcitonin; GPT, glutamic-pyruvic transaminase; ANA, antinuclear antibody; FDP, fibrinogen degradation products.

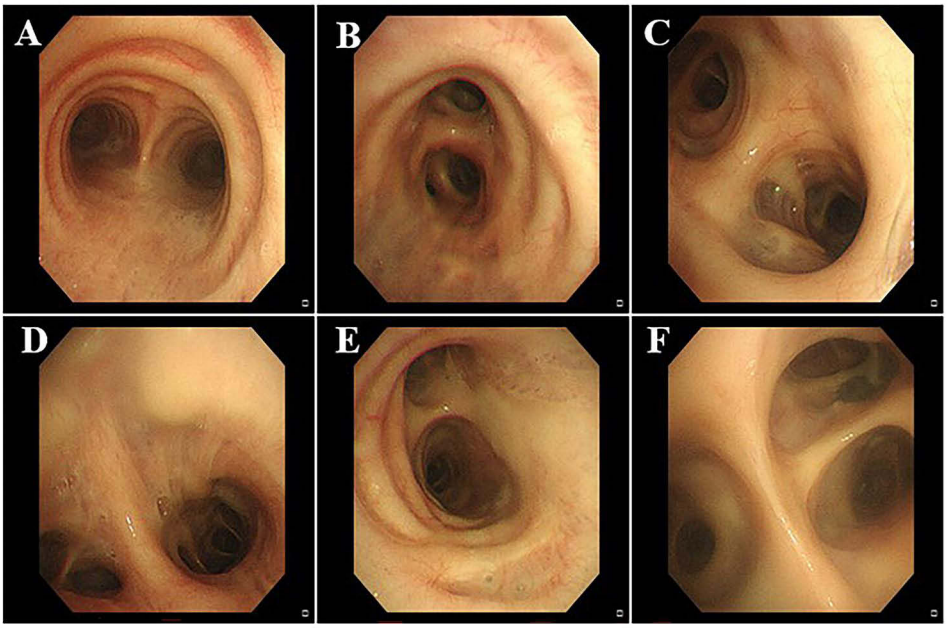

Figure 1 Images of the patient's Bronchoscope. Carina (**A**). Left main bronchus (**B**). Left upper lobe (**C**). Left lower lobe (**D**). Right main bronchus (**E**). Right upper lobe (**F**). The bronchial lumen is patent, with smooth and pale mucosa, accompanied by copious purulent exudate adherent to the luminal surface.

Imaging Examination

During the mid-stage of illness, the patient's pulmonary imaging findings progressed, with chest CT revealing multiple nodular and patchy consolidations accompanied by diffusely distributed ground-glass opacities in both lungs, characterized by blurred margins. Additionally, a small amount of bilateral pleural effusion was noted (Figure 3A–C). After initiation of pathogen-directed therapy, follow-up chest CT demonstrated partial absorption of the pulmonary lesions compared to earlier imaging, along with near-complete resolution of the bilateral pleural effusion (Figure 3D–F).

Treatment and Outcome

Following microbiological confirmation of *Nocardia africana* infection, antimicrobial therapy was adjusted from empirical cefoperazone-sulbactam to targeted treatment with trimethoprim-sulfamethoxazole (TMP-SMX, dosed based on mg/kg orally every 8 hours) in combination with linezolid (administered based on mg/kg intravenously every 12 hours). Concurrent anti-SARS-CoV-2 therapy with simnotrelvir/ritonavir was also initiated. However, due to gastrointestinal intolerance to linezolid, the regimen was modified based on a comprehensive consideration of the characteristics of *Nocardia* species and the antimicrobial susceptibility profile to intravenous amoxicillin-clavulanate (1.2 g every 8 hours) while continuing TMP-SMX, supplemented with metoclopramide for symptomatic relief. The patient showed clinical improvement with this optimized regimen, as evidenced by follow-up chest CT on August 16, which demonstrated significant absorption of pulmonary consolidations (Figure 3D–F). Hematological parameters also showed a trend toward improvement (Table 1). Contrast-enhanced MRI of the brain is unremarkable with no evidence of abscess, meningitis, or ring-enhancing lesions. Blood culture results showed no bacterial growth after 5 days of incubation. Bronchoscopic brush specimens were sent for pathological examination, which revealed no evidence of malignant cells. After discharge, the patient was advised to continue oral TMP-SMX (0.96 g every 8 hours) and amoxicillin-clavulanate (1.2 g every 8 hours). Supportive treatment included simnotrelvir/ritonavir for COVID-19 and metoclopramide to control nausea. A structured follow-up plan was implemented, including clinical assessments and laboratory tests (routine blood tests, CRP, PCT, renal and hepatic function panels) at 2 weeks, 1, 2, 3, and 6 months. A repeat chest CT was planned at 3 months to evaluate treatment response. The patient was also educated on recognizing and reporting potential signs of drug toxicity (eg, myelosuppression, hepatotoxicity). A detailed timeline of therapeutic interventions and clinical milestones is provided in Figure 4.

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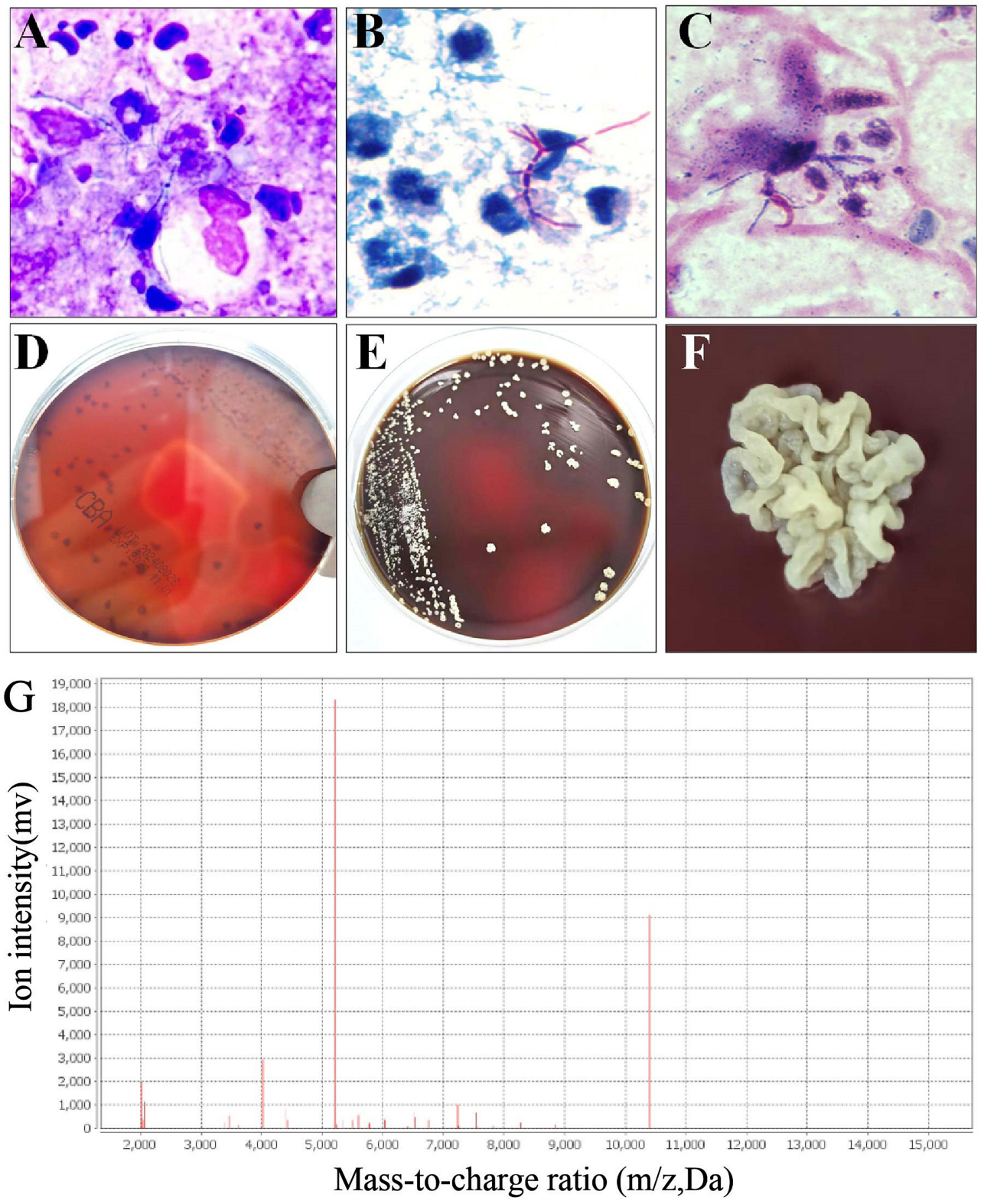

Figure 2 Patient's etiological results. Wright-Giemsa staining (**A**). Acid-fast staining (**B**). Gram stain (**C**). Reverse side of the blood agar plate showing (**D**). Obverse side of the blood agar plate showing (**E**). Magnified view of colonies on the blood agar plate (**F**). MALDI-TOF mass spectrum of *N. africana* (**G**).

Table 2 Drug Sensitivity Results of the *Nocardia Africana*

Antibiotic	Method	Result	Sensitivity Level	Breakpoint
Amoxicillin-clavulanate	Etest	4μg/mL	S	8-32
Imipenem	Etest	0.064μg/mL	S	1-4
Ciprofloxacin	Etest	0.25μg/mL	S	0.25–1
Linezolid	Etest	0.064μg/mL	S	4-8
Ceftriaxone	KB	30 mm	S	19-23
Amikacin	KB	35 mm	S	14-17
TMP-SMX	KB	36 mm	S	10-16
Minocycline	KB	28 mm	S	12-16

Abbreviations: TMP-SMX, Trimethoprim-Sulfamethoxazole; E-test, Epsilometer test; KB, Kirby-Bauer method; S, Sensitive.

Discussion

This report details the first documented case of *Nocardia africana* pneumonia co-detection with SARS-CoV-2 in mainland China, expanding the epidemiological understanding of *Nocardia* co-infections during the COVID-19 pandemic. Stamos et al reported a case of *Nocardia pseudobrasiliensis* co-infection with SARS-CoV-2 and summarized 10 cases of nocardiosis that occurred during or shortly after COVID-19 infection. Most patients developed nocardiosis within 5 to 50 days after SARS-CoV-2 diagnosis, with a mean interval to co-infection of 17 days. Their review further indicated that the lung is the most common site of *Nocardia* infection, followed by the central nervous system.[21–30] In reviewing our case, it is regrettable that the patient did not undergo SARS-CoV-2 nucleic acid testing either prior to or during hospitalization. However, based solely on the mNGS results of bronchoalveolar lavage fluid, which detected SARS-CoV-2 at a level of 34.11 RPM, we speculate that the patient likely experienced a period of co-infection with *Nocardia* and SARS-CoV-2. Despite the absence of classic immunodeficiency, the patient had an established risk factor of long-term smoking history. Chronic tobacco exposure is well-documented to impair mucociliary clearance and compromise respiratory epithelial integrity, which may explain the increased susceptibility to *Nocardia* invasion via the respiratory tract, the primary route of infection for pulmonary nocardiosis. The majority of patients diagnosed with pulmonary nocardiosis present with dyspnea,

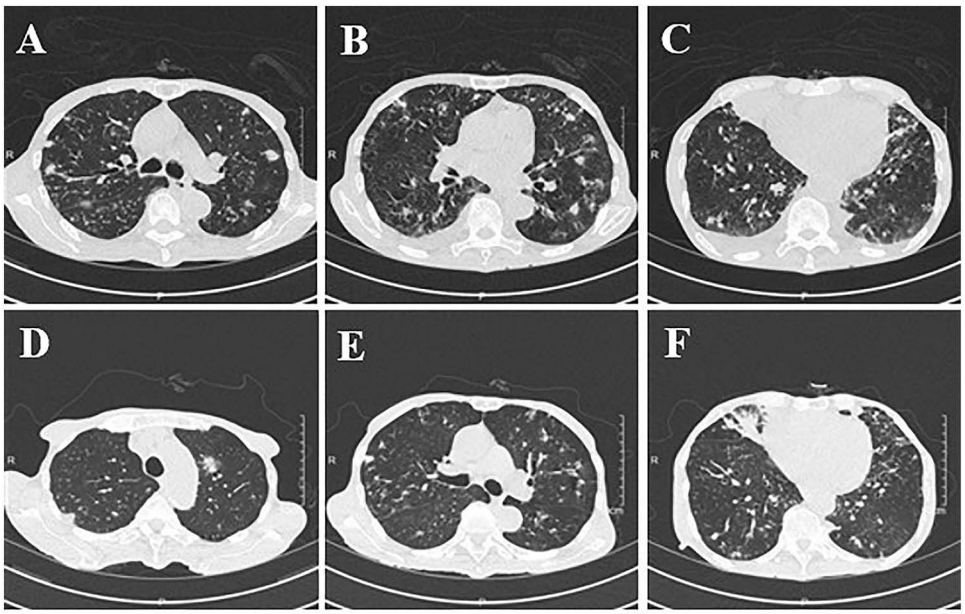

Figure 3 Pre- and post-treatment chest CT images of the patient. (**A–C**) Images prior to treatment demonstrate multiple nodules and patchy shadows in both lungs, with widespread bronchial wall thickening (**A**). The largest of these nodules is situated in the right middle lobe (**B**). A small amount of fluid density is evident in both pleural cavities (**C**). (**D–F**) Follow-up images after treatment show interval resolution of focal pulmonary opacities compared to prior imaging (**D**). The larger nodule in the right middle lobe remains stable in size and configuration (**E**). The previously noted bilateral pleural effusion has nearly completely resolved (**F**).

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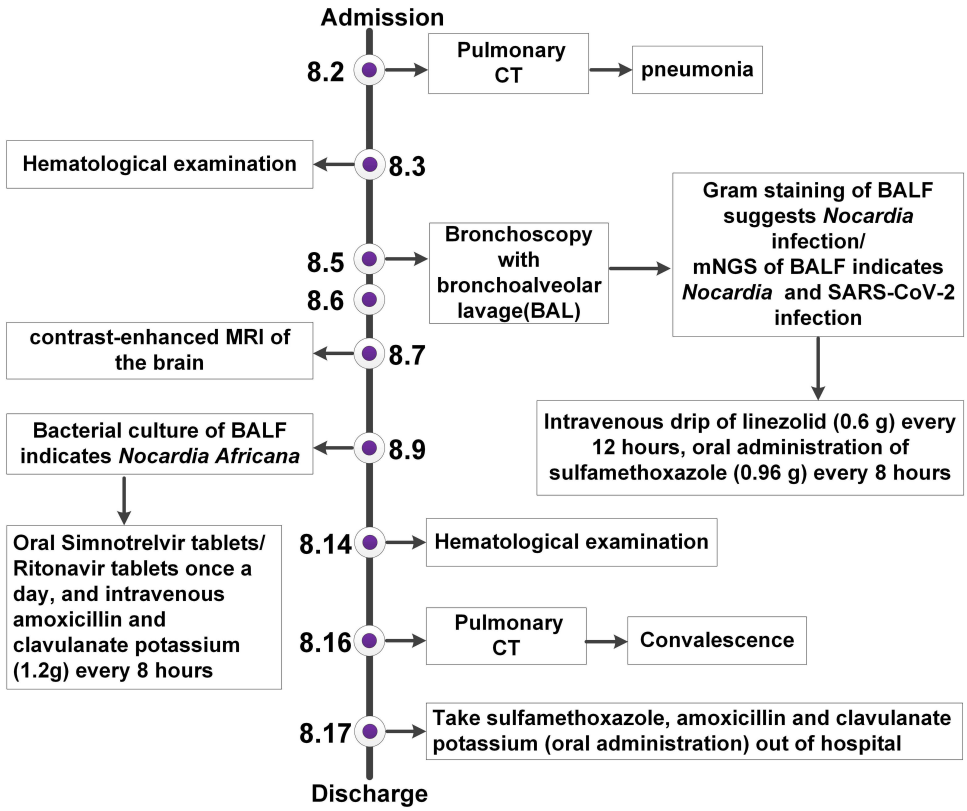

Figure 4 Treatment timeline.

cough, and sputum production; hemoptysis and pleuritic chest pain are relatively uncommon.[31] A comprehensive literature search was conducted using PubMed (keywords: "*Nocardia africana*") identified seven documented cases since 2003, comprehensively summarized in Table 3. These findings confirm the zoonotic potential of *N. africana*, with confirmed infections spanning both mammalian hosts (humans: 4 cases; domestic animals: 3 cases) across diverse immunological

Table 3 Reported Cases of *Nocardia Africana* in Immunocompetent and Immunocompromised Hosts

Number	Year	Country	Organism	Age	Organ Involved	Immunodeficiency Factors	Treatment Plan	Outcome	Reference
1	2003	Japan	Male cat	5	Base of the tail and on the abdomen.	–	Enrofloxacin 5 mg/kg/day and trimethoprim-sulfadimethoxine 30 mg/kg/day for 30 days.	Not respond	Hattori Y et al[32]
2	2009	Japan	Man	71	Right lower thigh	Rheumatoid arthritis and take oral prednisolone 5 mg/day.	Oral minocycline and trimethoprim-sulfamethoxazole for 10 months.	Rehabilitation	Ichikawa Y et al[33]
3	2012	Brazil	Female cat	1	Left mandible	–	Trimethoprim-sulfonamide (30 mg/kg orally every 12 hours for 3 weeks); Amikacin (10 mg/kg intravenously every 12 hours) and Ceftiofur (4.4 mg/kg subcutaneously every 12 hours)	Death	de Farias MR et al[34]

(*Continued*)

Table 3 (Continued).

Number	Year	Country	Organism	Age	Organ Involved	Immunodeficiency Factors	Treatment Plan	Outcome	Reference
4	2021	India	Woman	40	Left arm	–	Cotrimoxazole-DS (TMP/SMX 160/800 mg) PO BID; Moxifloxacin 400 mg PO QD	Rehabilitation	Bhandari M et al[35]
5	2022	Korea	Male Great Dane	1	Right ear	Treated with prednisolone and cyclosporine	Meropenem (8 mg/kg three times daily, intravenous) and doxycycline (5 mg/kg twice daily, oral).	Rehabilitation	Yoon JS et al[36]
6	2023	Thailand	Man	70	Left eye	–	2% amikacin	Rehabilitation	Chaidaroon W et al[37]
7	2023	Colombia	Man	47	Left cerebral hemisphere	–	Imipenem-cilastatin and Trimethoprim-sulfamethoxazole	Rehabilitation	Márquez AI et al[38]

states: five of seven cases occurred in immunocompetent hosts, while two involved immunodeficient individuals (with long-term corticosteroid use). This epidemiological pattern underscores its ability to exploit both intact and compromised host defenses. A limitation of our study is that the literature search did not include Chinese databases. Future studies incorporating a broader range of databases would be valuable to obtain a more comprehensive understanding.

Emerging evidence highlights diagnostic cross-reactivity between *Nocardia* infections and fungal biomarker assays. β-D-glucan (BDG) is a well-known biomarker for fungal infections. Sawai et al documented disseminated nocardiosis with elevated serum biomarkers (galactomannan index 0.6, BDG 94.7 pg/mL),[39] while Koncan et al reported cerebrospinal fluid BDG elevation in a brain abscess caused by *Nocardia abscessus*.[40] These observations align with Yagyu's hypothesis proposing BDG as a potential indicator of pulmonary nocardiosis.[41] In our case, the bronchoalveolar lavage fluid (BALF) galactomannan level was elevated (1.7 ng/mL; reference ≤0.8 ng/mL), in which repeated BALF cultures and mNGS analysis failed to detect Aspergillus, and serial chest CT scans showed no characteristic radiological features of invasive aspergillosis, also supports that the elevated test result was attributable to cross-reactivity with certain *Nocardia* species. Suggesting two clinical imperatives: diagnostic vigilance to consider *Nocardia* infection in patients with elevated fungal biomarkers lacking mycological evidence, and pathogen-specific testing—such as modified acid-fast staining and MALDI-TOF MS—when BDG/GM elevations accompany pneumonia refractory to antifungal therapy.

Therapeutic management of nocardiosis requires species-specific antimicrobial selection guided by susceptibility profiles. According to Clinical and Laboratory Standards Institute (CLSI) susceptibility standards, the following antimicrobial agents are recommended for initial susceptibility testing: aminoglycosides (amikacin, tobramycin); β-lactam/β-lactamase inhibitor combinations (amoxicillin-clavulanate); extended-spectrum cephalosporins (ceftriaxone); carbapenems (imipenem); DNA synthesis inhibitors (ciprofloxacin, moxifloxacin); protein synthesis inhibitors (clarithromycin, linezolid, minocycline); and folic acid synthesis inhibitors (trimethoprim-sulfamethoxazole).[42] For central nervous system involvement or disseminated disease, combination therapy is necessary.[1,43] Treatment should integrate infection site, species-specific antimicrobial susceptibility, host tolerance, and consideration of combination therapy or adjunct surgical interventions, with duration stratified by disease severity: 1–3 months for localized cutaneous infections, 6–12 months for pulmonary or disseminated disease, and ≥12 months for central nervous system involvement.[1,44,45] Therapy should be guided by serial clinical-radiological reassessment (CT/MRI volumetry) and inflammatory marker trends (eg, CRP/PCT).

Emerging evidence challenges the universal efficacy of TMP-SMX against *Nocardia africana*, as demonstrated by Hattori et al and de Farias et al[32,34] in feline nocardiosis cases where TMP-SMX treatment failed to achieve clinical resolution. This resistance pattern extends to canine isolates, with Yoon et al[36] reporting TMP-SMX-resistant *N. africana* strains causing refractory cutaneous lesions in dogs. Antimicrobial susceptibility testing (AST) is essential for optimal

treatment of nocardial infections, as emerging resistance necessitates testing before initiating TMP-SMX therapy despite its status as first-line treatment.[7,46]

Conclusion

Overall, the concurrent detection of *Nocardia africana* and SARS-CoV-2 in a patient with pneumonia is rare. The treatment regimen combining TMP-SMX and amoxicillin-clavulanate that we ultimately adopted resulted in preliminary clinical success. Importantly, microscopic examination of Gram-stained specimens served as the diagnostic cornerstone, while mNGS and MALDI-TOF MS played crucial roles in accelerating pathogen identification. These approaches were essential for enabling timely antimicrobial stewardship, reducing diagnostic uncertainty, and optimizing treatment strategies for complex polymicrobial infections.

Data Sharing Statement

Data available on request from the corresponding author.

Ethics Approval and Consent to Participate

Ethical approval for this study, including the publication of the case report, was granted by the Ethics Committee of Zhejiang People's Hospital (Approval Number: 2022JS008). All procedures complied with the ethical standards of the Declaration of Helsinki.

Consent for Publication

Written informed consent was obtained from the patient.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interests in this work.

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