Cureus
Part of SPRINGER NATURE

Open Access Case Report

Disseminated Nocardia farcinica Presenting as an Intramuscular Shoulder Abscess With Asymptomatic Cerebral Involvement in a Kidney Transplant Recipient

Review began 10/30/2026
Review ended 11/09/2026
Published 11/13/2026

© **Copyright** 2026
Animalu. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119624

Chinelo Animalu [1]

1. Infectious Diseases, The University of Tennessee Health Science Center, Memphis, USA

Corresponding author: Chinelo Animalu, chinelo.animalu@mlh.org

Abstract

Nocardia species are weakly acid-fast bacteria that most commonly cause pulmonary and central nervous system infections. Musculoskeletal involvement is considered unusual and suspicious for disseminated disease. Infection with this organism is most often encountered among immunocompromised people, although there have been cases seen in people with normal immune status. Since it is considered an opportunistic infection, its occurrence is uncommon and challenging to diagnose because of its indolent presentation and resemblance to more typical bacterial infections. We present a case of disseminated *Nocardia farcinica* (*N. farcinica*) infection in a kidney transplant recipient on immunosuppressive medications presenting with a left intramuscular shoulder abscess and clinically silent intracranial abscesses. He underwent surgical debridement of the affected shoulder, and surgical cultures were positive. He was initiated on a two-drug regimen of ceftriaxone and trimethoprim-sulfamethoxazole (TMP-SMX), but after identification of brain lesions, linezolid was added. The triple therapy regimen was complicated by severe antimicrobial toxicity, requiring a switch to a salvage antibiotic regimen to complete the treatment course. He subsequently clinically responded and completed his 12-month treatment course without further complications.

Categories: Transplantation, Internal Medicine, Infectious Disease
Keywords: cerebral abscess, disseminated nocardiosis, intramuscular abscess, nocardia farcinica, salvage antimicrobial therapy

Introduction

Nocardia species are aerobic, filamentous, Gram-positive bacteria that primarily act as opportunistic pathogens in immunocompromised hosts [1]. Solid organ transplant (SOT) recipients are at substantial risk of nocardiosis due to chronic T-cell suppression. Reported incidence rates vary according to the transplanted organ, with higher rates generally observed among thoracic organ transplant recipients [2,3]. In a 10-year retrospective cohort study and literature review by Viotti et al., they found that nocardiosis affected approximately 0.2% of SOT recipients, predominantly in kidney transplant recipients [4].

Among the genus, *Nocardia farcinica* is notable for its increased virulence, higher propensity for hematogenous spread, and propensity for the central nervous system (CNS) [1,4]. Disseminated nocardiosis is defined as an infection involving two or more noncontiguous organs. It carries significant mortality, particularly when diagnosis is delayed by slow culture growth or non-specific clinical presentations [2,3]. We report on a case of disseminated *N. farcinica* in a renal transplant recipient whose course was complicated by severe antimicrobial toxicities, requiring a transition to a salvage oral regimen with a successful outcome.

Case Presentation

A 58-year-old male with a history of end-stage renal disease, status post deceased-donor kidney transplant eight months prior to presentation and maintained on immunosuppressive medications consisting of a calcineurin inhibitor (Envarsus XR 2 mg daily), mycophenolate mofetil 540 mg twice daily, and a corticosteroid (prednisone 5 mg daily), presented to the emergency room with initial complaints of a one-week history of right anterior chest wall pain that later migrated to the left chest wall associated with a palpable, tender mass on the left shoulder. On arrival, he was afebrile and hemodynamically stable. Vital signs were temperature: 36.9 degrees centigrade (98.4 degrees Fahrenheit); heart rate (HR) 81 beats per minute; blood pressure (BP) 135/85 millimeters of mercury (mmHg); respiratory rate (RR) 16. Admitting laboratory indices showed hemoglobin (Hb) 9.7 g/dL, elevated white blood cell count (WBC) 11.04 thou/mcL, platelets 259 thou/mcL, creatine kinase (CK) 34, creatinine 1.78 mg/dL, glomerular filtration rate (GFR) 44 ml/min, blood urea nitrogen (BUN) 32.4 mg/dL, potassium 5.0 mmol/L, and elevated C-reactive protein (CRP) of 71.1 mg/L (Table *1*).

How to cite this article
Animalu C (November 13, 2026) Disseminated Nocardia farcinica Presenting as an Intramuscular Shoulder Abscess With Asymptomatic Cerebral Involvement in a Kidney Transplant Recipient. Cureus 18(11): e119624. DOI 10.7759/cureus.119624

Parameter	Patient Value	Reference Range
Temperature	36.9 °C	36.5-37.5 °C
Heart rate	81 bpm	60–100 bpm
Blood pressure	135/85 mmHg	<120/80 mmHg
Oxygen saturation (SpO_2)	97%	>95%
Hemoglobin	9.7 g/dL	12–16 g/dL
White blood cell count	11.04 thou/mcL	4.0–10.2 x10^3/µL
Platelets	259 thou/mcL	150–400 x10^3/µL
Creatine kinase	34	22-98 U/L
GFR	44 mL/min	>90 mL/min
BUN	32.4 mg/dL	7–20 mg/dL
Potassium	5.0 mmol/L	3.5–5.0 mmol/L
CRP	71.1 mg/L	<5 mg/L

TABLE 1: Patient vital signs and laboratory indices at time of admission

ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; GFR: glomerular filtration rate; g/dl: grams per deciliter; bpm: beats per minute; °C: degree centigrade; mmHg: millimeters of mercury; thou/mcL: thousands per microliter; mmol/L: millimoles per liter; mg/dl: milligrams per deciliter; ml/min: milliliters per minute; mm/hr: millimeters per hour; mg/L: milligrams per liter; U/L: units per liter; BUN: blood urea nitrogen

Computed tomography (CT) of the chest was performed and revealed multiple bilateral pulmonary nodules with spiculated margins and mass-like consolidation, a radiographic pattern highly suggestive of pulmonary nocardiosis (Figure *1*).



FIGURE 1: CT of the chest with contrast showing multiple bilateral pulmonary nodules (small blue arrow) with spiculated margins and mass-like consolidation (blue arrow)

Magnetic resonance imaging (MRI) of the left shoulder confirmed a 3.4 cm intramuscular collection within the pectoralis musculature suspicious for an abscess (Figure *2*).

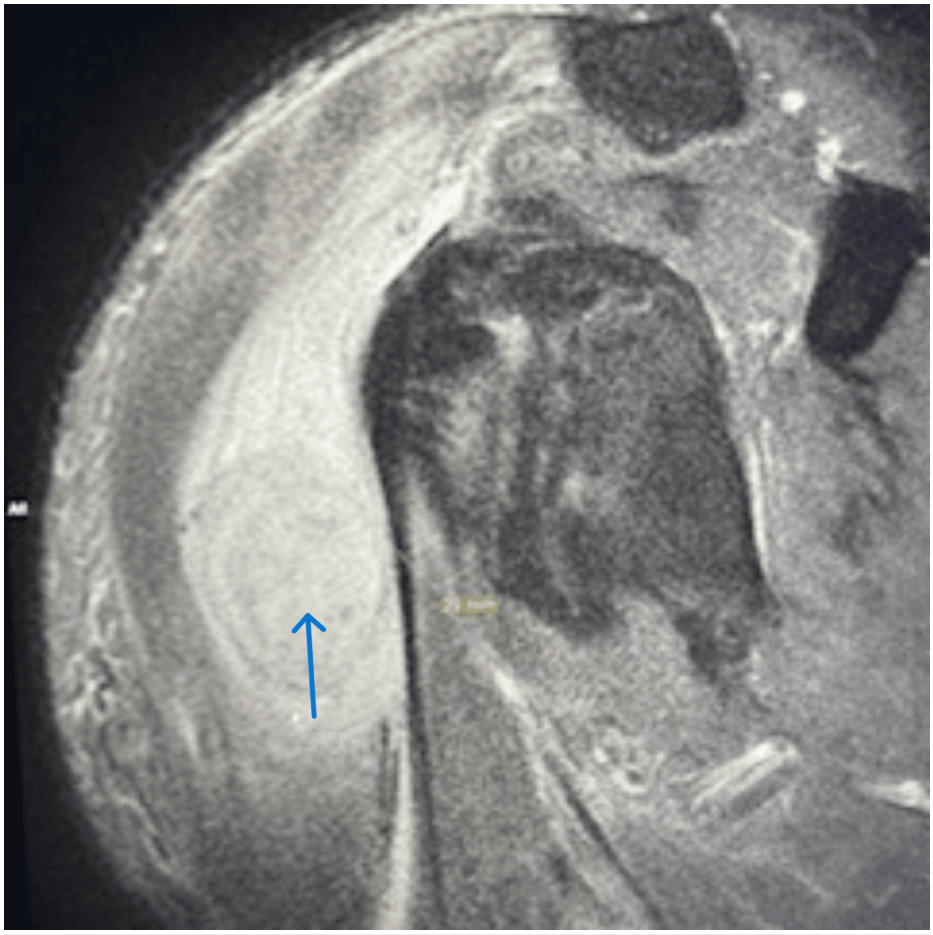


FIGURE 2: MRI left shoulder with contrast showing a 3.4 cm intramuscular collection within the pectoralis musculature (blue arrow)

Orthopedic teams evaluated the patient, who underwent debridement and irrigation (D&I) and washout of the left shoulder; surgical cultures were sent. These later became positive for *N. farcinica*, and susceptibility was requested. While the conventional blood cultures remained negative, plasma metagenomic next-generation sequencing (mNGS/Karius) also identified *N. farcinica*. Transplant infectious disease was consulted, and the patient was started on an antibiotic regimen consisting of IV ceftriaxone (2 grams daily) and oral trimethoprim-sulfamethoxazole (TMP-SMX) one tablet twice daily. Despite the absence of focal neurologic deficits, the known predilection of *N. farcinica* for the CNS prompted a brain MRI to be performed, and this revealed multiple peripherally enhancing intra-axial lesions in the left frontal lobe and both temporo-occipital regions consistent with abscesses, confirming "silent" intracranial abscesses (Figure *3*).

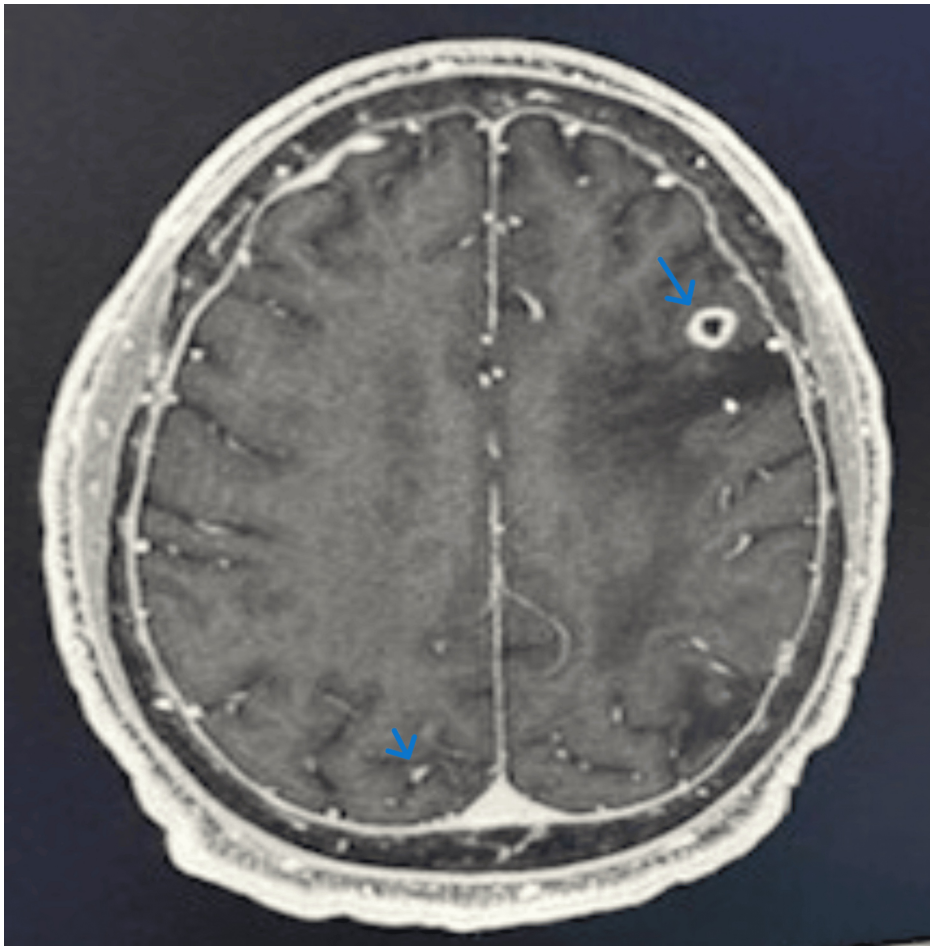


FIGURE 3: MRI brain with contrast showing multiple peripherally enhancing intra-axial lesions in the left frontal lobe and both temporo-occipital regions suspicious for brain abscesses (blue arrows)

With the presence of brain lesions, a third antibiotic, linezolid 600 mg twice daily, was added. The transplant nephrology team modified immunosuppression by holding Envarsus and reducing the dose of mycophenolate mofetil from 540 mg twice daily to 180 mg twice daily. His current antibiotic regimen was continued, and he was later discharged home on the three antibiotics with plans to follow up with infectious disease, as susceptibility results were still not available at the time of discharge.

Post-discharge, he was doing well initially, but his recovery course was complicated by two significant adverse drug reactions. Approximately 18 days after discharge, the patient was readmitted for a diffuse rash attributed to TMP-SMX hypersensitivity. The rash was refractory to diphenhydramine tablets and topical steroid cream. Simultaneously, the patient developed bicytopenia (Hgb 6.1 g/dl, platelets 103 x 10^9) presumed to be secondary to linezolid, given the known side effects of the medication. He received a blood transfusion of two units of packed red blood cells. Infectious Diseases were reconsulted, and by this time the susceptibility report was now available 22 days after collection (Table *2*). Based on the susceptibility report, the antibiotic regimen was discontinued and changed to a new oral salvage regimen consisting of moxifloxacin 400 mg daily, Augmentin 875 mg twice daily, and tedizolid 200 mg daily (the latter an oxazolidinone chosen for its improved hematologic safety profile compared to linezolid). Tedizolid susceptibilities were extrapolated from the results for linezolid, as testing was not available for it. This regimen was selected based on previous adverse reactions and anticipated tolerability of the available susceptible agents (Table *2*).

Antimicrobial	Reported MIC	Susceptibility Interpretation		
		Susceptible	Intermediate	Resistant
Amikacin	0.5 µg/mL	X		
Amoxicillin-clavulanate	8/4 µg/mL	X		
Ceftriaxone	32.0 µg/mL		X	
Ciprofloxacin	0.25 µg/mL	X		
Clarithromycin	>16.0 µg/mL			X
Doxycycline	8.0 µg/mL			X
Imipenem	16.0 µg/mL		X	
Linezolid	2.0 µg/mL	X		
Minocycline	4.0 µg/mL		X	
Moxifloxacin	0.12 µg/mL	X		
Tobramycin	8.0 µg/mL		X	
Trimethoprim-sulfamethoxazole	0.5/9.5 µg/mL	X		

TABLE 2: Nocardia farcinica susceptibility report

Due to the considerable risk of relapses associated with *N. farcinica* and the patient's ongoing iatrogenic immunosuppression, a 12-month duration was planned for the oral antibiotics. The patient's clinical status significantly improved, and he was discharged in a stable condition. At a post-admission clinic visit three months later, while still on this antibiotic regimen, pancytopenia resolved (Hgb 12.4 g/dl, platelets 210 x 10^9, and WBC 8.23 x 10^9). At both the six- and 12-month clinic visits, he remained free of symptoms and side effects of antimicrobials, and he successfully completed his treatment course. Long-term management will involve serial head and chest imaging to ensure sustained resolution of previously seen lesions and monitor for recurrence for the next one to two years or more.

Discussion

Nocardiosis remains a serious opportunistic infection in renal transplant recipients, with disease often occurring months to years after transplantation [2,4]. *N. farcinica* is consistently associated with higher rates of antimicrobial resistance and dissemination compared to other species [1].

Pulmonary involvement is the most common manifestation and can have various findings on imaging, including mass-like opacities that can mimic malignancy. Other unusual sites of infection include musculoskeletal and spinal cord involvement, etc. [5]. *Nocardia* is known to have a peculiar predilection for the brain, although it could be clinically silent [6]. Overall, misdiagnosis is common due to the nonspecific clinical and imaging findings regardless of site [7]. The site involved also plays a role in antimicrobial selection, especially for pulmonary and CNS involvement, and may require the use of alternative regimens [8,9].

Review of the literature showed only a few more case reports of intramuscular nocardiosis, and these occurred in immunocompromised patients too [10,11]. There have been cases reported in healthy immunocompetent patients, although not as frequent as is seen in the immunocompromised population [12,13]. Clinicians should maintain a low threshold for routine neuroimaging once nocardiosis is suspected, as this case confirms CNS involvement with no clinical symptoms but significantly alters the prognosis and treatment duration [5,6].

Nocardiosis typically tends to have an indolent course, with symptoms being subacute or even chronic and may span from weeks to months before a diagnosis is established. For this reason, diagnostic delays remain a recurring theme in the literature due to their fastidious nature [3]. It is also important to know that infection can present acutely with rapid progression of disease in both immunocompetent and immunocompromised populations [14]. The use of mNGS in this case allowed for rapid species-level identification, facilitating earlier targeted therapy while awaiting traditional culture testing. Chen Y, Fu H, et al. reported greater diagnostic sensitivity for mNGS than conventional culture in their 19-patient cohort [7].

The therapeutic challenge in this case underscores the difficulty of managing disseminated *N. farcinica* in patients who cannot tolerate the "gold standard" of TMP-SMX and linezolid. Accurate species-level identification and antimicrobial susceptibility testing are essential for guiding individualized antimicrobial therapy, particularly in patients requiring alternative treatment regimens [4]. Although there are no clear guidelines showing the use of tedizolid as an alternative regimen for nocardiosis, there have been case reports that have reported successful use of long-term tedizolid as a potential alternative when conventional treatment is poorly tolerated [8,9]. In this patient, transition to tedizolid was used as an emerging strategy to provide oxazolidinone coverage with a lower risk of myelosuppression, as the organism was susceptible to linezolid. Tedizolid has been shown to have in vitro activity against most *Nocardia* species, as was demonstrated in a study that looked at 286 *Nocardia* isolates [15]. However, currently there are no clear breakpoints noted by Center for Disease Control (CDC) or Clinical and Laboratory Standards Institute (CLSI) [16] and therefore its use in nocardiosis relies solely on the discretion of the treating team. For our patient, susceptibility testing for tedizolid was not available in our laboratory and reference laboratory used by our facility.

Conclusions

This case underscores the broad clinical spectrum of disseminated *N. farcinica* and reinforces the need for heightened suspicion, early comprehensive imaging (including the brain even in asymptomatic patients), and the integration of molecular diagnostics. Successful management requires a multidisciplinary balance between aggressive antimicrobial therapy and monitoring of side effects of medications and the careful adjustment of immunosuppression. This case adds to the few cases of successful use of a second-generation oxazolidinone as an alternative in patients who are unable to tolerate the recommended first-generation oxazolidinone, linezolid, due to its myelosuppressive side effects with prolonged use.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Chinelo Animalu

Acquisition, analysis, or interpretation of data: Chinelo Animalu

Drafting of the manuscript: Chinelo Animalu

Critical review of the manuscript for important intellectual content: Chinelo Animalu

Supervision: Chinelo Animalu

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors have declared that no AI tools were used during the work or in the drafting of this article. The authors take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Mustafa Alhashimi F, Salim S, Iqbal A, Balila M, Chishti MK: Disseminated Nocardia farcinica infection in a renal transplant patient: a case report. Cureus. 2024, 16:e54963. 10.7759/cureus.54963
2. Bhandari G, Tiwari V, Gupta A, et al.: Nocardiosis in renal transplantation: case series from India . Indian J Nephrol. 2023, 33:456-8. 10.4103/ijn.ijn_205_22
3. Lin Y, Xu M, Genis H, Andany N, Chen L: The diagnostic challenges of disseminated nocardiosis in a post-renal transplant patient: a case report. Reports (MDPI). 2025, 8:111. 10.3390/reports8030111
4. Bini Viotti J, Simkins J, Reynolds JM, Ciancio G, Guerra G, Abbo L, Anjan S: Nocardiosis in solid organ transplant recipients: 10-year single center experience and review of literature. Microorganisms. 2024, 12:1156. 10.3390/microorganisms12061156
5. Wu J, Li X, Zhang T, Lin X, Chen YC: Disseminated Nocardia farcinica involves the spinal cord: a case report and review of the literature. BMC Infect Dis. 2021, 21:1224. 10.1186/s12879-021-06905-y
6. Song J, Dong L, Ding Y, Zhou J: A case report of brain abscess caused by Nocardia farcinica . Eur J Med Res. 2021, 26:83. 10.1186/s40001-021-00562-2
7. Chen Y, Fu H, Zhu Q, Ren Y, Liu J, Wu Y, Xu J: Clinical features of pulmonary nocardiosis and diagnostic

value of metagenomic next-generation sequencing: a retrospective study. Pathogens. 2025, 14:10.3390/pathogens14070656

8. Matin A, Sharma S, Mathur P, Apewokin SK: Myelosuppression-sparing treatment of central nervous system nocardiosis in a multiple myeloma patient utilizing a tedizolid-based regimen: a case report. Int J Antimicrob Agents. 2017, 49:488-92. 10.1016/j.ijantimicag.2016.11.032
9. Chomei Y, Nishimura S, Iwata K: Long-term use of tedizolid for pulmonary nocardiosis . J Infect Chemother. 2022, 28:1172-6. 10.1016/j.jiac.2022.03.020
10. Spangenberg G, Aftab S, Juneja R, Gordon DL, Li JY: Nocardia infection presented as intramuscular abscess in a kidney transplant recipient: case report and literature review. Case Rep Nephrol. 2025, 2025:9102520. 10.1155/crin/9102520
11. Veerappan Kandasamy V, Nagabandi A, Horowitz EA, Vivekanandan R: Multidrug-resistant Nocardia pseudobrasiliensis presenting as multiple muscle abscesses. BMJ Case Rep. 2015, 2015: 10.1136/bcr-2014-205262
12. Khatri A, Esposito MJ, Koshy R: Unusual presentation of Nocardia abscessus infection in an immunocompetent patient. Access Microbiol. 2022, 4:000308. 10.1099/acmi.0.000308
13. Lee R., Razia D., Ledterman H, et al.: Rapidly progressive severe pulmonary nocardiosis in an immunocompetent host. Chest. 2024, 166:A1375-6. 10.1016/j.chest.2024.06.883
14. Chen K, Wang Y, Dong J, Wu PS, Yang J, Ai GP: Case report: from oral infection to life-threatening pneumonia: clinical considerations in Nocardia infection from a case. Front Med (Lausanne). 2024, 11:1424564. 10.3389/fmed.2024.1424564
15. Toyokawa M, Ohana N, Tanno D, et al.: In vitro activity of tedizolid against 43 species of Nocardia species . Sci Rep. 2024, 14:5342. 10.1038/s41598-024-55916-7
16. Dutarte M, Genestet C, Lina G, Dumitrescu O, Hodille E: Nocardia antimicrobial susceptibility testing by microdilution: evaluation of ready-to-use plate including tedizolid and meropenem. Eur J Clin Microbiol Infect Dis. 2026, 45:3103-15. 10.1007/s10096-026-05588-0