

CASE REPORT

***Nocardia farcinica*, as an uncommon cause of subcutaneous infection – Case report and literature review**

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Abstract

Introduction: *Nocardia* is a rare group of opportunistic bacteria that normally causes infections in immunocompromised patients. It can involve multiple organ systems: most frequently the lungs, followed by the skin, central nervous system, bones, and joints. Within this group, *Nocardia farcinica* is among the most commonly identified pathogenic species.

Case presentation: We report the case of a 45-year-old male patient with a history of sensory-motor polyradiculoneuropathy of demyelinating origin who presented to the emergency department with an extensive soft tissue infection in the right popliteal region. The patient required multiple surgical procedures and multidisciplinary care. The patient also received prolonged, targeted antibiotic therapy, which resulted in complete recovery.

Conclusions: *Nocardia farcinica* is a rare opportunistic pathogen that mainly affects immunocompromised individuals and can cause severe systemic or localized infections. Early identification is crucial, as prompt surgical intervention combined with targeted antibiotic therapy is essential for favorable outcomes and helps prevent dissemination and other serious complications.

Keywords: *Nocardia farcinica*, trimethoprim/sulfamethoxazole, polyradiculopathy

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Introduction

The *Nocardia* genus comprises aerobic, Gram-positive, weakly acid-fast bacteria. It was first described in animals in 1888; since then, 251 species have been identified, of which 54 can cause infection in humans. *Nocardia* species are commonly found in soil and sand, and infections occur via inhalation, ingestion, or direct contact [1,2]. Pulmonary involvement is most frequent, potentially leading to pleuritis and abscess formation. Extrapulmonary manifestations primarily affect the central nervous system (abscesses, meningitis) and the skin (cellulitis, abscesses) [3]. Although vascular dissemination to other organs is rare, it has been reported. Immunocompromised patients - particularly transplant recipients, individuals with HIV infection, or those receiving chronic corticosteroid therapy - are at the highest risk for infection [4].

Diagnosis is typically established through microbiological culture, Gram staining, polymerase chain reaction, and histopathological examination. Due to the slow growth of *Nocardia*, the incubation period can extend to 3 weeks,

although results are most commonly available within 3–5 days. Imaging studies are indicated when internal organ involvement or dissemination is suspected, particularly in immunocompromised patients [3].

Management varies depending on the affected organ but generally includes targeted antibiotic therapy and, in selected cases, surgical intervention such as incision and drainage of the collections. Since no standardized antibiotic regimen exists, treatment should target the *Nocardia* species, local epidemiology, and case-specific factors. [4]. The most used antibiotics include trimethoprim-sulfamethoxazole, amikacin, imipenem, and linezolid. The optimal duration of therapy is not fully established; in immunocompromised patients, treatment typically ranges from 6 to 12 months and may be prolonged in cases involving the central nervous system [5].

Case presentation

A 45-year-old male patient with a medical history notable for chronic tobacco and alcohol consumption presented to our department as an emergency admission with complaints of a progressively enlarging mass located in the right popliteal region. The onset of symptoms occurred ap-

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proximately 3 weeks prior to presentation, following blunt contusion trauma contaminated with soil. The patient reported no associated fever or chills.

Local physical examination demonstrated a large, fluctuant, erythematous, and tender mass extending from the distal third of the posterior thigh to the distal third of the leg. Two cutaneous fistulous openings were identified, with spontaneous drainage of copious purulent material of grayish–orange–yellowish coloration. Neuromuscular examination of the right lower limb revealed hypotonia, hypokinesia, and mild hypotrophy, compared with the contralateral limb.

According to the medical history, 2 years prior to the current admission, the patient had sustained a similar blunt contusion injury to the left lower limb, which subsequently evolved into an abscess. Within approximately 3 weeks, he developed gait disturbances and ascending paresthesias affecting the lower limbs, accompanied by muscular hypotonia and flaccid tetraparesis (Medical Research Council grade 4/5 in the upper limbs and grade 3/5 in the lower limbs), as well as diminished osteotendinous reflexes in the lower extremities.

The patient was subsequently admitted to a neurology department, where the diagnosis of acute sensorimotor polyradiculoneuropathy with a primary demyelinating mechanism was established, associated with flaccid tetraparesis with predominant paraparesis, severe sensory ataxia, and a superinfected wound caused by *Streptococcus pyogenes*. Following neurological management, as well as physiotherapy and kinesiotherapy, the patient recovered motor function and deep tendon reflexes, with partial restoration of sensory function and persistent muscular hypotonia.

On this admission, the patient underwent emergency surgical intervention under total intravenous anesthesia using a target-site infusion technique. The procedure comprised multiple serial, staged, and counter incisions, followed by extensive lavage with antiseptic solutions, manual drainage, and sterile dressing application. During surgery, pathological material was collected for bacteriological analysis. Intraoperatively, a subcutaneous lesion was identified, extending from the distal third of the posterior thigh to the distal third of the posterior leg, tracking deeply between the superficial and deep muscle compartments of the leg, without evidence of bone involvement.

Postoperatively, conservative management was initiated, including correction of fluid, electrolyte, and acid–base imbalances; analgesic and anti-inflammatory therapy; anticoagulation; gastric protection; antiemetics; and empiric antibiotic therapy with cefuroxime 1.5 g thrice daily.

On the second postoperative day, the patient underwent a second surgical intervention performed by a multidisciplinary team, including plastic surgery. The procedure comprised extension of the previous incisions, debridement, lavage with antiseptic solutions, manual drainage, dressing application, and immobilization of the limb in

30° flexion with a splint.

Purulent secretion obtained from the patient was subjected to microbiological analysis. Microscopic examination following Gram staining revealed Gram-positive branching bacilli. After 48 hours of incubation under aerobic conditions at 35 °C, small colonies were observed on a blood agar plate. These colonies, upon further growth, exhibited colony morphology characteristics consistent with those of typical *Nocardia* species (chalk-white colonies). Microbial identification was performed using matrix-assisted laser desorption/ionization–time-of-flight mass spectrometry (MALDI-TOF MS) with the Vitek MS Prime system (bioMérieux), which identified the isolate as *Nocardia farcinica*. Following an infectious disease consultation, antibiotic therapy was adjusted: cefuroxime was discontinued, and treatment was initiated with ceftriaxone 2 g twice daily, in combination with trimethoprim-sulfamethoxazole 400/80 mg, two tablets twice daily orally. Subsequently, a contrast-enhanced CT scan of the chest, abdomen, pelvis, and lower extremities (angiography) was performed to exclude disseminated disease; however, no acute pathological findings were identified.

Following surgery, the patient received daily wound care, performed under local anesthesia and adjunctive analgesic medication. The wound was thoroughly irrigated with oxygenated water, Betadine, and chlorinated solution. Gauze dressings were carefully layered between the tissue planes, and the limb was kept immobilized for 4 days post-operatively.

The wound showed a favorable evolution, with progressive tissue granulation, resolution of purulent discharge, and reduction of edema and erythema. Consequently, on the 15th day after the initial surgery, secondary closure was attempted; however, complete closure of the wound in the popliteal region was impossible due to a skin defect measuring approximately 8 × 8 cm with a depth of 6 cm. This area was debrided and irrigated daily with antiseptic solutions. The general and plastic surgery teams subsequently performed skin grafting of the popliteal region, using an autograft harvested from the anterior aspect of the right thigh.

The patient's postoperative course was uneventful, and was discharged 30 days after admission. The surgical wound was clean, progressing appropriately toward healing, with no local complications observed.

During hospitalization, the patient was repeatedly evaluated by the infectious disease specialist. On day 21, ceftriaxone therapy was discontinued, while trimethoprim-sulfamethoxazole treatment was continued and maintained after discharge.

The patient returned for follow-up 15 days post-discharge, when the wound demonstrated near-complete healing without complications. However, full knee extension was unachievable, prompting an orthopedic evaluation. The examination revealed arthrosis secondary to immobilization, and the patient subsequently commenced

physiotherapy. Following guidance from the infectious disease specialist, antibiotic therapy was maintained for 6

months, without adverse effects, ultimately achieving complete recovery.



Fig. 1. The chronological evolution of the lesion. 1 - The second day after surgery. 2 - The condition before secondary suture. 3 - The day of discharge. 4 - The 15-day follow-up.

Discussion and Literature Review

Nocardia farcinica is an opportunistic pathogen whose management is challenging due to frequent antibiotic resistance. Clinical manifestations are often atypical and lack characteristic features, which may delay diagnosis and the initiation of appropriate therapy [6]. In immunocompromised patients, impaired immune function contributes to an insidious and prolonged course of infection. Diagnosis relies on Gram staining, direct inoculation, and bacterial culture obtained from the infected site, while blood cultures are frequently negative [7].

Based on published studies, *Nocardia farcinica* exerts its pathogenic effects primarily through intracellular survival and modulation of the host immune response. After being phagocytosed by macrophages, the bacterium inhibits phagosome–lysosome fusion, enabling it to avoid intracellular killing. Its mycolic acid-rich cell wall, along with antioxidant enzymes such as catalase and superoxide dismutase, protects it from oxidative burst-mediated damage. Additionally, specific virulence factors (e.g., the Nfa34810 protein) promote host cell adhesion and invasion and activate inflammatory signaling pathways, including TLR4-dependent NF- κ B and MAPK pathways. This leads to increased production of proinflammatory cytokines such as TNF- α . The resulting strong but often insufficient cellular immune response contributes to granuloma formation, tissue damage, and, in severe cases, hematogenous dissemination, particularly to the central nervous system [8].

A literature search was conducted in PubMed to iden-

tify published cases of *Nocardia farcinica* infection between January 2003 and December 2025. The search strategy included the terms “*Nocardia farcinica*”, “muscle infection”, “soft tissue infection”, “subcutaneous infection”, and “intramuscular abscess”. Inclusion criteria were microbiologically confirmed *N. farcinica* infection with documented muscle and/or subcutaneous tissue involvement. Cases limited exclusively to other organ systems (e.g., pulmonary, cerebral, or osseous infections) without soft tissue involvement were excluded. Reference lists of relevant publications were also screened to identify additional eligible cases.

According to the available literature, 24 cases of *Nocardia*-associated muscle and soft tissue infections have been reported to date. Among these, *Nocardia farcinica* was the most frequently identified pathogen (nine cases), followed by *N. asteroides* and *N. pseudobrasiliensis*. [9]. The thigh musculature was the most affected site, with subsequent involvement of the back, hip, and upper extremities [10]. In this context, the present report represents the 10th documented case of *Nocardia farcinica*-related soft tissue and muscle infection. Importantly, most previously published cases described disseminated disease with multisystem involvement, whereas in our case, the infection remained localized exclusively to the lower extremity, highlighting its clinical rarity [11].

An immunocompromising condition was identified in approximately 78% (n=7) of reported cases, while two cases occurred in patients with no detectable immunodeficiency.

Table 1. Cases of muscle/subcutaneous infection caused by *Nocardia farcinica* identified through a PubMed search (2003–2025) [6, 7, 10, 12–17].

Author	Study year	Age/Sex	Immunocompromising condition	Muscle site affected	Other organs affected	Antibiotic therapy	Surgical management
Smit L. H., et al.	2003	42 / M	None	Psoas	None	TMP-SMX	Drainage
Malani A. K. et al.	2006	65 / F	Chemotherapy Hodgkin's Lymphoma	Thigh	None	TMP-SMX	Drainage
Agterof M. J. et al.	2007	76 / M	Polymyalgia rheumatic Prednisolone	Iliac fossa	Brain, lung	TMP-SMX	Debridement
Daniel J. H. et al.	2007	22 / F	Lupus nephritis Prednisone, Cyclophosphamide	Paraspinal	Lungs, heart, kidneys, brain	TMP-SMX, Amikacin, Imipenem, Ciprofloxacin	None
Noh J. Y. et al.	2011	61 / F	Lupus nephritis Prednisolone	Psoas	Lungs	TMP-SMX Ciprofloxacin Ceftriaxone	Drainage
Ukai Y. et al.	2012	59 / F	Autoimmune haemolytic anaemia Prednisolone	Thigh	Lung, brain	TMP-SMX, Amikacin Imipenem	Drainage
Acuner B. et al.	2021	37 / M	None	Forearm	None	Sulbactam/ Ampicillin, changed to TMP-SMX	Drainage and debridement
Palomba E. et al.	2022	61 / M	Bladder cancer, chronic renal failure Methylprednisolone Prednisolone Mycophenolate	Biceps brachii	Lungs	TMP-SMX	Drainage
Thakur A. et al.	2023	78 / F	Myasthenia gravis Prednisone Mycophenolate	Shoulder	Lungs, bursa, joints	Meropenem TMP-SMX	Debridement

Several case reports in the literature indicate that *Nocardia farcinica* predominantly affects immunocompromised individuals, with conditions such as long-term corticosteroid therapy, malignancy, organ transplantation, and advanced HIV infection representing well-established risk factors [7]. Conversely, infection in immunocompetent patients is considered uncommon. Although sporadic cases have been documented in individuals without identifiable immune deficits, these occurrences remain rare and are generally reported as exceptional findings. The low incidence in immunocompetent hosts underscores the pathogen's opportunistic nature and highlights the unusual clinical significance of such presentations when they arise [8,12].

Surgical management was necessary in almost all instances, predominantly comprising drainage and surgical debridement. Trimethoprim-sulfamethoxazole was the cornerstone of antimicrobial therapy in all cases and was combined with additional agents when disseminated infection with multiorgan involvement was present [9].

Nocardia species are responsible for approximately 0.7–3.0% of infections among solid organ transplant recipients, including kidney, liver, and heart transplant patients. In reported cases, antimicrobial therapy is generally administered for 6 months; however, treatment must be extended to at least 12 months in the presence of central nervous system involvement or in cases of suboptimal response to antibiotic therapy. To date, no consensus has been established regarding the optimal duration of treatment for organ-specific *Nocardia farcinica* infections [9].

The currently available literature on this pathogen is highly heterogeneous in terms of clinical presentation, diagnostic approaches, and therapeutic management. Re-

ported cases vary considerably regarding the anatomical location of infection, severity of disease, and associated complications, which makes it difficult to establish a unified clinical pattern or standardized management strategy. Consequently, interpretation of the existing evidence must be approached with caution, as most conclusions are derived from highly individualized clinical scenarios rather than comparable datasets. A further important limitation is that the literature is predominantly composed of isolated case reports and small case series, often lacking long-term follow-up data. This significantly weakens the level of evidence and limits the ability to draw robust conclusions regarding disease progression, optimal diagnostic workup, and treatment efficacy. In addition, publication bias may also influence the available evidence, as unusual or severe cases are more likely to be reported, potentially distorting the overall clinical picture of the disease. Another key issue is the absence of standardized diagnostic criteria and universally accepted therapeutic protocols. Most published reports describe individualized diagnostic pathways, typically combining imaging, microbiological testing, and empirical clinical judgment, while treatment strategies also vary widely with respect to antimicrobial selection, surgical management, and treatment duration. Despite this variability, some recurring clinical patterns can still be identified across cases, particularly the frequent delay in diagnosis, which is often related to the nonspecific clinical presentation and the initial assumption of more common pathogens. In many instances, the correct diagnosis is only established after failure of empirical therapy or following extended microbiological and imaging investigations. This underscores the importance of maintaining a broad differ-

ential diagnosis in atypical or non-resolving infections. In comparison with the existing literature, the present case contributes additional insight by providing a detailed description of the diagnostic and therapeutic approach, thereby supporting the need for individualized management in the absence of standardized guidelines. Furthermore, it reinforces previously reported observations while also adding practical clinical details that may be useful for the management of future cases.

Conclusion

Cutaneous infections caused by *Nocardia farcinica* are uncommon, often leading to delayed diagnosis and complicating management. Optimal treatment requires a case-specific approach, typically Cutaneous infections caused by *Nocardia farcinica* are uncommon, often leading to delayed diagnosis and complicating management. Individualized management requires a case-specific approach, typically encompassing targeted antibiotic therapy, most frequently trimethoprim-sulfamethoxazole, in conjunction with surgical intervention. In extensive skin infections, surgical planning must carefully consider the post-healing outcome, which may be challenging due to substantial skin loss. Overall management often requires 6–12 months of therapy with meticulous follow-up to ensure complete clinical resolution.

The presented case is distinguished by the absence of an underlying immunocompromised status, which is typically considered a major predisposing factor for infection. Notably, the patient was immunocompetent, making the disease occurrence particularly uncommon. Furthermore, the infection remained strictly localized, with no evidence of hematogenous dissemination of bacterial cells or involvement of distant organs.

This clinical course contrasts with the more frequently reported patterns, in which systemic spread - especially to the central nervous system - is observed. The combination of an immunocompetent host and a confined infectious process underscores the atypical nature of this case and highlights its clinical relevance.

Authors' contributions

GSSA (Conceptualization, Data curation, Writing – original draft)

MMG (Visualization, Writing – review & editing)

KBI (Resources)

MR (Resources)

SM (Project administration, supervision)

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Conflict of interest

None to declare.

Ethical statement

Written informed consent was obtained from the patient of this case report.

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