

Nocardia farcinica septic bursitis – a case report and scoping literature review

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Summary

BACKGROUND: Septic bursitis is mainly caused by *Staphylococcus aureus* and *Streptococcus* spp. However, patients with immunosuppression may develop infections caused by rare pathogens. Here, we describe a case of septic olecranon bursitis caused by *Nocardia farcinica* and provide a literature review of musculoskeletal infections caused by this pathogen.

CASE PRESENTATION: A male patient in his mid-70s with seronegative rheumatoid arthritis who was receiving methotrexate, low-dose prednisone, and golimumab experienced trauma to the elbow, followed by progressive painful swelling, slight erythema, and the formation of a fistula draining cloudy fluid over the subsequent two weeks. Culture of the aspirated bursal fluid yielded slow-growing yellow-pigmented colonies identified as *Nocardia farcinica*. Whole-genome sequencing confirmed the species without detecting any known virulence genes or resistance mutations. Clinical and radiological evaluation showed no signs of systemic dissemination. The patient underwent surgical bursectomy and received targeted antimicrobial therapy with oral trimethoprim/sulfamethoxazole. After 3 weeks, the patient developed a systemic allergic reaction, necessitating a switch to susceptibility-guided amoxicillin/clavulanate. Treatment was continued for 3 months, ultimately resulting in the complete and successful resolution of the bursitis.

CONCLUSION: This report highlights that it is important for clinicians to consider rare pathogens in immunosuppressed patients with bursitis, especially in those presenting with atypical courses. Localised musculoskeletal infections caused by *Nocardia* are mostly treated with a combined surgical and antibiotic approach. Antibiotics are typically administered for one to six months. In cases of surgical removal, a shorter antibiotic treatment duration seems sufficient. A multidisciplinary approach involving infectious diseases specialists, microbiologists, and surgeons is critical for achieving favourable outcomes.

Introduction

Bursitis is a common musculoskeletal condition. The olecranon bursa and prepatellar bursa are the most commonly affected sites because of their superficial anatomical location, which makes them more susceptible to trauma and infection [1]. Approximately one-third of cases of olecranon or prepatellar bursitis have a septic origin, with an annual incidence of 1 in 10,000 individuals [2, 3]. Septic olecranon or prepatellar bursitis can account for up to 0.1% of all hospital admissions [2]. In approximately 80% of septic bursitis cases, *Staphylococcus aureus* is identified as the causative pathogen [2]. However, opportunistic pathogens can also play a significant role, particularly in immunocompromised patients [4, 5]. The treatment of bursitis generally involves a combination of surgical intervention and antibiotic therapy [6]. There is no standardised approach to antibiotic therapy or the timing of surgical treatment. In current clinical practice, a multidisciplinary approach is often preferred. To facilitate targeted antibiotic therapy, intraoperative samples are routinely collected for microbiological analysis. This strategy allows for personalised treatment, improving patient outcomes while minimising the risks associated with broad-spectrum antibiotic use.

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Here, we present a rare case of septic bursitis with *Nocardia farcinica* in an immunocompromised patient and provide a literature review of reported cases.

Although musculoskeletal nocardiosis has been described, isolated septic bursitis caused by *Nocardia farcinica* remains exceedingly rare. In contrast to most reported musculoskeletal cases, which frequently involve dissemination or severe systemic illness, our patient presented with a localised infection despite ongoing immunosuppressive therapy. Moreover, molecular characterisation was performed using whole-genome sequencing to further assess resistance and virulence determinants, providing additional microbiological insight. This case therefore adds clinically relevant information regarding the diagnostic evaluation and management of atypical bursitis in immunosuppressed patients.

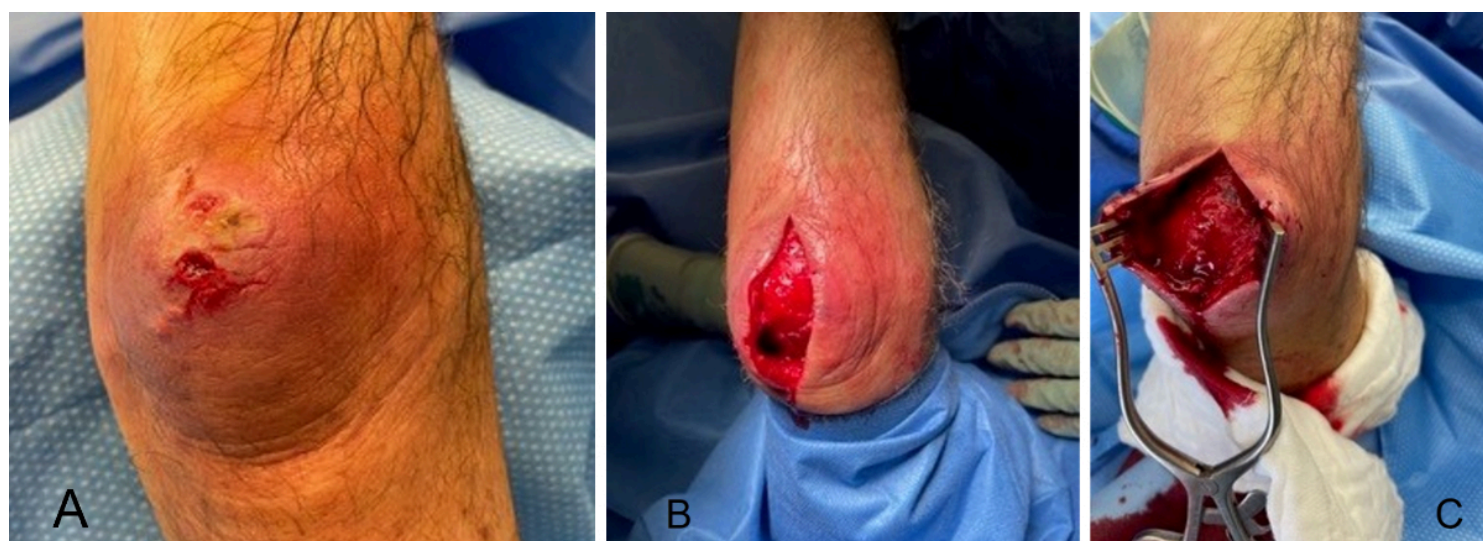
Case presentation

The following case description outlines the chronological course of events from the initial symptoms through diagnosis, treatment, and follow-up.

A male patient in his mid-70s presented to his primary care physician with community-acquired left-sided traumatic olecranon bursitis that developed following a fall on the stairs outside his home. The patient reported progressive local pain and swelling over two weeks, with increasing discomfort during elbow movement. Initially, the overlying skin remained intact, but the lesion subsequently opened and began draining cloudy fluid (figure 1A). He denied fever, chills, or other systemic symptoms, but the skin lesion had progressively worsened. Examination showed local warmth and redness, with no involvement of other joints. The patient had a medical history of seronegative rheumatoid arthritis managed with immunosuppressive therapy consisting of methotrexate (10 mg weekly), golimumab (50 mg subcutaneously monthly), and low-dose prednisone (2.5 mg daily). He had no relevant family history of infectious or autoimmune diseases and no known genetic disorders. The patient reported no psychosocial factors or environmental exposures of clinical relevance.

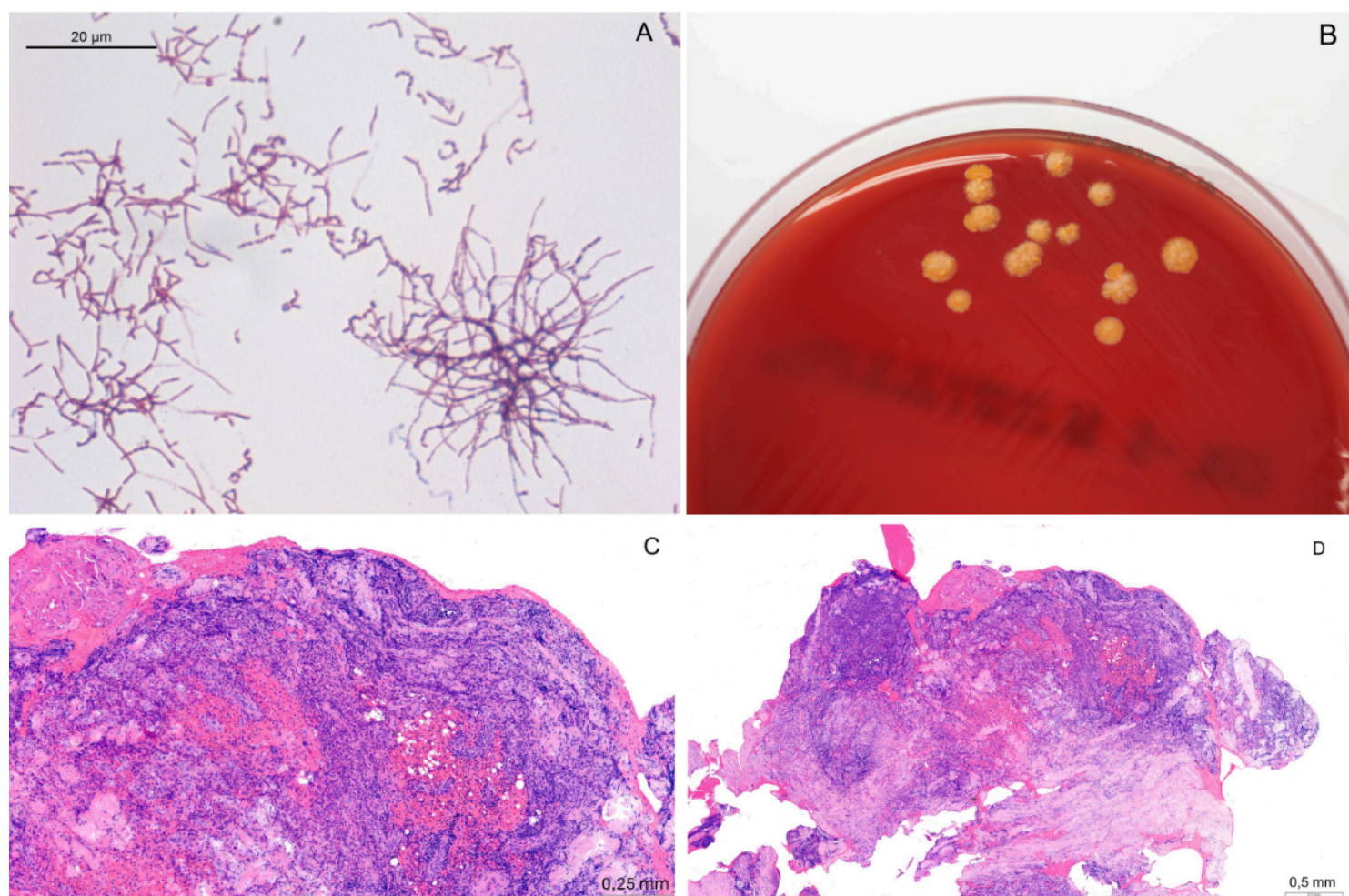
Cultures of a bursal puncture obtained in the outpatient setting revealed *Nocardia farcinica*, prompting referral to our clinic for further evaluation, and surgical bursectomy was indicated. Pre-operative laboratory tests were within normal limits. Surgery included excision of the macerated skin around the tip of the olecranon and removal of the affected bursa. Intraoperatively, the bursa appeared markedly inflamed and enlarged, resembling a pannus (figure 1B, C). Following excision, extensive irrigation, and haemostasis, the skin was closed in a standard manner.

Figure 1: Clinical presentation and intraoperative findings. A: Visible fistula over the olecranon region. B, C: Intraoperative views showing macerated skin and the inflamed bursa.



Gram stain revealed the characteristic star-shaped growth and branching patterns of *Nocardia* (figure 2A). The colonies presented the typical yellow pigmentation and dry colony morphology (figure 2B). Matrix-assisted laser desorption ionisation–time-of-flight (MALDI-TOF) confirmed the species to be *Nocardia farcinica*. Histological sections of the biopsy displayed a nonspecific, florid inflammatory response marked by haemorrhage and a dense infiltrate of neutrophils (figure 2 C/D). Furthermore, whole-genome sequencing (WGS) of the isolate was performed to gain more molecular insight into this rare case. WGS also identified the isolate as *Nocardia farcinica* based on the digital DNA–DNA hybridisation values (figures S1 and S2 in the appendix). However, no previously reported virulence genes were detected, potentially underscoring the relevance of host immunosuppression, rather than pathogen-derived virulence factors, to the development of infection caused by this pathogen. A chest CT as well as a cranial MRI performed to check for systemic spread and potential pulmonary or cranial nocardial lesions were unremarkable.

Figure 2: Microbiological and histopathological findings. A: Gram stain showing filamentous, branching structures typical of *Nocardia* spp. B: *Nocardia* colonies on blood agar. C, D: Histology of a retrieved biopsy demonstrating a florid inflammatory response with a dense neutrophilic infiltrate.



After surgery, empirical intravenous imipenem/cilastatin (500 mg four times daily) was initiated. After exclusion of disseminated infection and confirmation of antimicrobial susceptibility results, therapy was de-escalated after four days to oral trimethoprim/sulfamethoxazole (1600/320 mg twice daily). The wound healed well, and the patient was discharged home on the sixth day. After two weeks, the patient developed dyspnoea, fever, and a generalised rash, prompting re-hospitalisation. Elevated inflammatory markers and findings indicating hepatopathy suggested a drug reaction, which was supported by the findings of a skin biopsy of the exanthema. The treatment was

switched to oral amoxicillin/clavulanic acid (875/125 mg three times daily), guided by susceptibility testing. The temporal relationship to trimethoprim/sulfamethoxazole exposure and clinical improvement after discontinuation further supported a probable adverse drug reaction.

Antibiotic treatment was stopped after three months. At the one-year follow-up (figure 3), the patient remained asymptomatic with full functional recovery of the elbow and no signs of recurrence.

Figure 3: Postoperative clinical course. A, B: Two weeks post-surgery, showing progressive wound healing. C: Six weeks post-surgery, with resolution of local inflammation.



Literature review

Nocardia bursitis – five cases reported to date

We conducted a systematic literature search using the keywords "*Nocardia*" and "bursitis" in PubMed. We identified only five case reports and compared patient characteristics and treatment approaches (table S1 in the appendix). The locations of the bursitis included olecranon, prepatellar, and malleolar sites. Interestingly, none of the patients were immunosuppressed (information was missing for two patients), and no cases of systemic infection were observed. Antibiotic treatment mostly consisted of a sulfonamide, and the treatment duration varied from one to six months. Two patients were managed conservatively without surgery, yet all achieved a good outcome.

Nocardia farcinica musculoskeletal infections

To further evaluate the relevance of the subspecies *Nocardia farcinica* as identified in our case, we conducted an additional literature review focusing on musculoskeletal infections caused by *Nocardia farcinica*. A PubMed search using the keyword "*Nocardia farcinica*" yielded 503 papers. Of these, 12 papers primarily involved musculoskeletal manifestations (see figure S3 in the appendix for the selection workflow). Table S2 in the appendix summarises the screened papers.

These case reports illustrate the diverse manifestations of *Nocardia farcinica* musculoskeletal infections, which often occur in immunocompromised patients with comorbidities such as diabetes, chronic lung disease, lymphoma, or conditions requiring steroid therapy. The infection affected multiple anatomical sites in seven of the twelve patients, including the lungs, limbs, joints, and soft tissues.

Most cases required a combination of surgical intervention and long-term antibiotic treatment, with trimethoprim-sulfamethoxazole being the most commonly used antibiotic. In cases of adverse reactions, alternative antibiotics such as imipenem, moxifloxacin, and amoxicillin-clavulanic acid were successfully used. The reports highlight successful outcomes in the majority of pa-

tients treated using a multidisciplinary approach including both surgery and prolonged antibiotic treatment. However, patients with severe underlying disease sometimes experienced adverse outcomes or fatal complications, highlighting the important contribution of comorbidities to prognosis.

Discussion

Here, we presented a rare case of *Nocardia* bursitis of the olecranon in an immunosuppressed patient. The combined surgical and targeted antibiotic treatment led to complete resolution of the infection. The literature review identified only five other cases of *Nocardia* bursitis, none of which showed documented immunosuppression or systemic spread. On the other hand, the more general literature review covering all types of musculoskeletal infections caused by *Nocardia farcinica* showed that these infections mostly occur in immunocompromised patients, with a propensity for systemic spread.

Nocardia are aerobic, Gram-positive bacteria commonly found in soil and organic matter [7–9]. They primarily cause opportunistic infections in immunocompromised hosts, with the potential for haematogenous dissemination, particularly to the central nervous system. However, more than one-third of reported cases have been documented in immunocompetent patients, suggesting that the pathogen can overcome certain host defences in healthy individuals as well [5, 10]. *Nocardia* most commonly causes localised infections at the primary site of entry, typically the lungs or skin, where it can produce granulomatous lesions resembling tuberculosis. *Nocardia* is capable of spreading haematogenously to distant organs, causing disseminated infection that may involve the central nervous system, kidneys, or joints. This dissemination often occurs in immunosuppressed patients but has also been observed in otherwise healthy individuals, underscoring the pathogen's potential for systemic involvement [11, 12].

Among the numerous *Nocardia* species, *Nocardia farcinica* is one of the more frequently isolated species in clinical infections and is known for its high virulence and multidrug resistance [13, 14]. *Nocardia farcinica* exhibits increased virulence compared to other *Nocardia* species, driven by genetic factors such as the virulence genes *relA*, *icl*, and *mbtH*, which enhance survival and proliferation within the host. Resistance-related genes such as *RbpA*, *mtrA*, *FAR-1*, *blaFAR-1*, and *rox* further complicate treatment by conferring resistance to multiple antibiotics [15]. In our isolate, whole-genome sequencing revealed the presence of the intrinsic *FAR-1* and *rox* genes, while no acquired resistance mutations were detected. This supports the clinical observation of a good treatment response and indicates that the infection was driven primarily by host immunosuppression rather than by enhanced bacterial resistance.

Diagnosing nocardial infections is challenging due to the slow-growing nature of the organism. Standard diagnostic methods include culture and microscopy, using Gram and modified Ziehl-Neelsen stains. Advanced molecular techniques, such as MALDI-TOF mass spectrometry and 16S rRNA sequencing, have improved the speed and accuracy of *Nocardia* identification [11, 16]. In cases in which culture fails to yield results, whole-genome sequencing can provide or confirm a definitive diagnosis [5, 17].

Nocardia infections typically require prolonged antibiotic therapy, often lasting 6–12 months depending on the severity and extent of the infection. However, in cases of localised infections without systemic dissemination, shorter treatment durations may also be effective and should be considered on a case-by-case basis. In this case, the decision to continue antibiotic therapy for three months was based on an individualised risk–benefit assessment, taking into account the patient's immunosuppression and the known virulence of *Nocardia farcinica*. Shorter durations (e.g., 4–8 weeks) may be sufficient after complete surgical excision [8]. The duration was defined according to the clinical course, and therapy was discontinued after three months in the absence of signs of relapse. Although trimethoprim-sulfamethoxazole remains the first-line therapy, alternative agents such as imipenem, moxifloxacin, linezolid, or amoxicillin-clavulanic acid may be required in cases of resistance or adverse drug reactions. Surgical intervention, such as abscess drainage or debridement, may be appropriate depending on the location, especially in severe cases. Interdisciplinary management involving infectious disease specialists and surgeons is crucial to optimise outcomes.

The clinical presentation of our patient was atypical, lacking prominent systemic signs such as fever or elevated inflammatory markers, which are commonly observed in classic bacterial bursitis caused by pathogens such as *Staphylococcus aureus*. In immunosuppressed individuals, this constellation should prompt consideration of unusual or opportunistic pathogens such as mycobacteria, fungal organisms, or other rare bacteria (e.g. *Actinomyces* or *Rhodococcus*).

Conclusion

In cases of non-staphylococcal or non-streptococcal musculoskeletal infections, particularly in immunosuppressed patients, clinicians should consider rare or opportunistic pathogens and involve infectious diseases specialists early to guide targeted microbiological diagnostics and antimicrobial management. Given the potential for resistance and adverse drug reactions, individualised treatment strategies guided by microbiological data are essential. Multidisciplinary collaboration involving infectious disease specialists, microbiologists, and surgeons plays a pivotal role in achieving favourable clinical outcomes.

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Potential competing interests

All authors have completed and submitted the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest. No potential conflict of interest related to the content of this manuscript was disclosed.

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Appendix

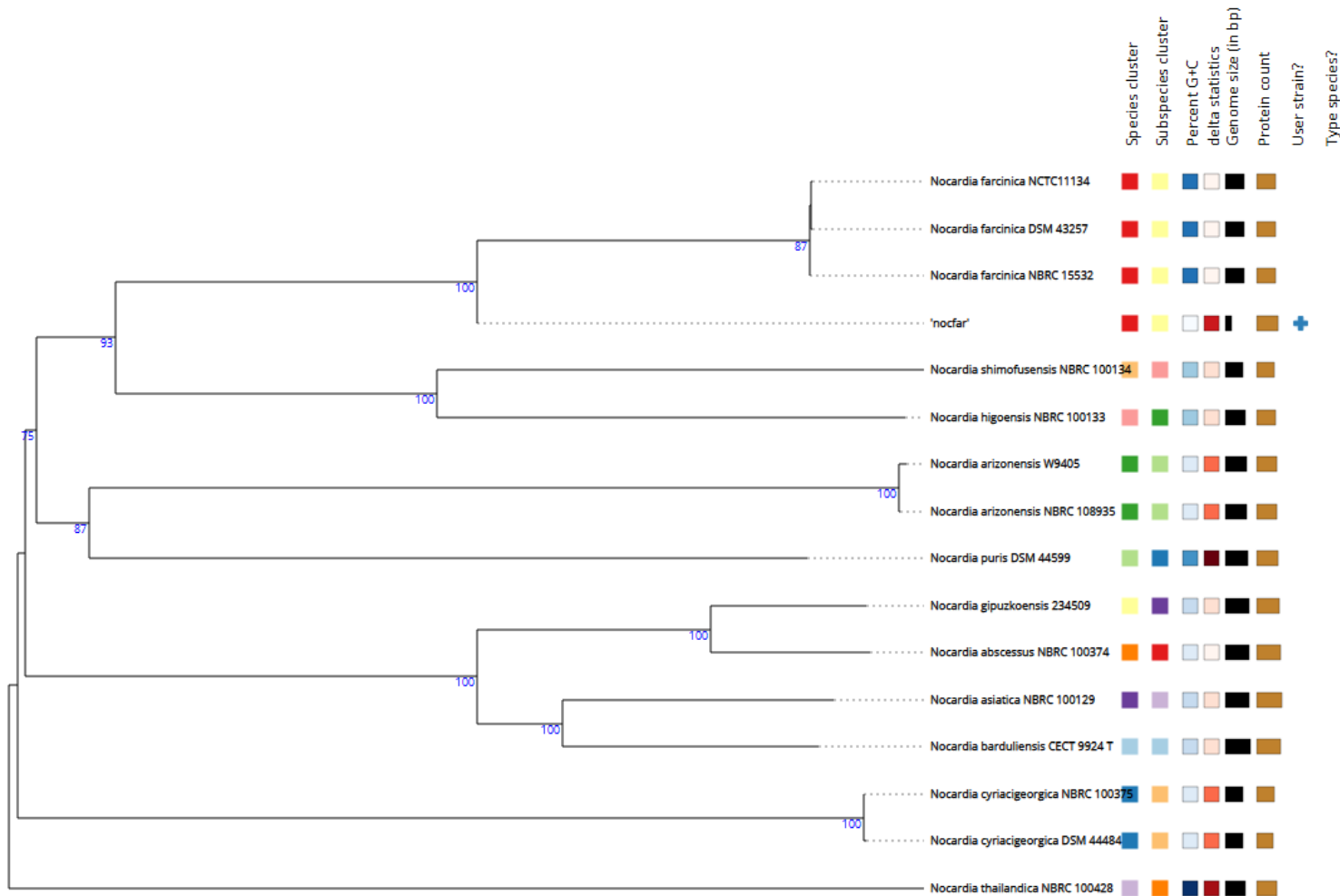


Figure S1. Whole-genome-based phylogram. Phylogenetic tree showing *Nocardia farcinica* isolate from this case in comparison with reference strains based on whole-genome sequencing.

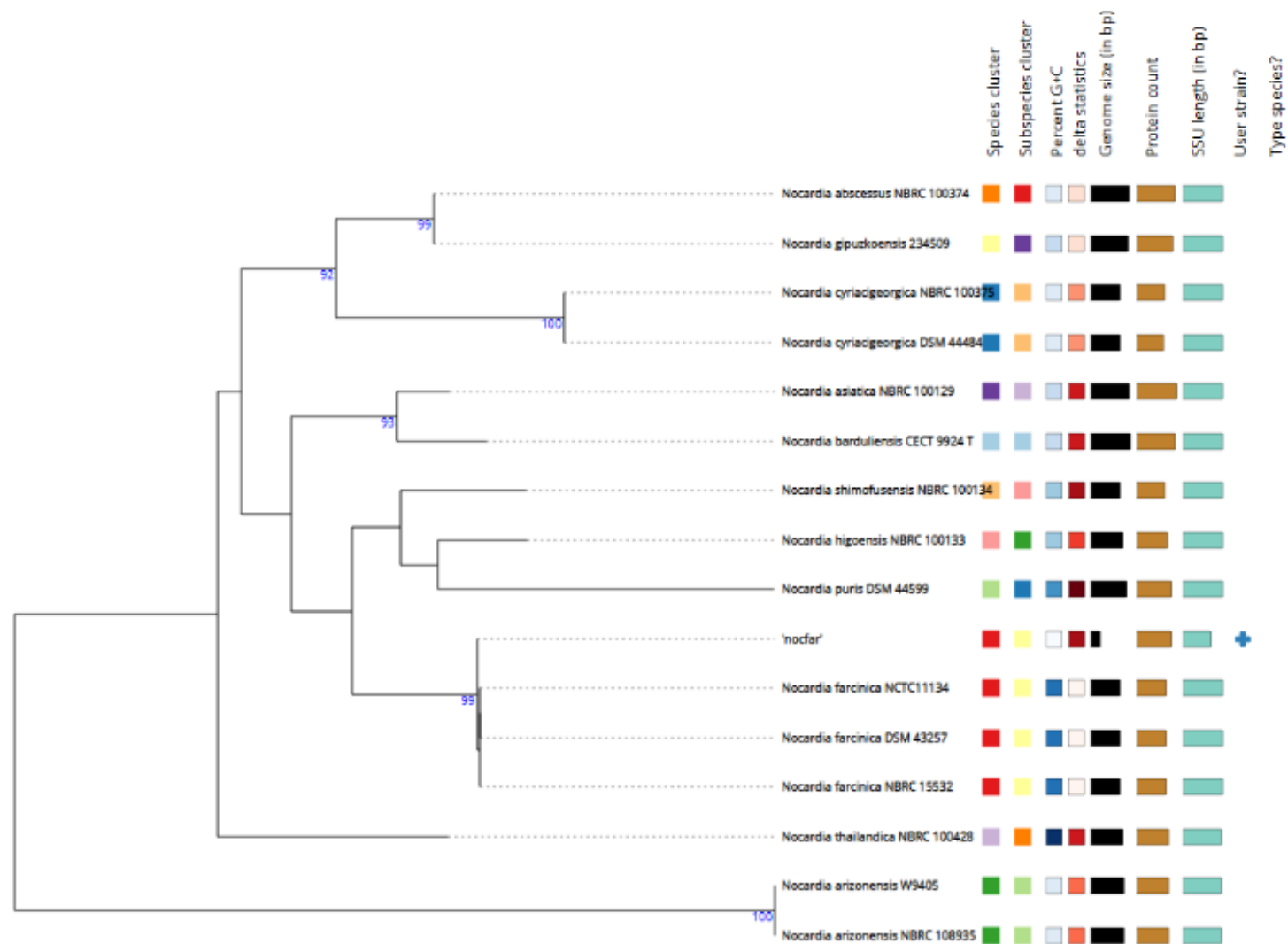


Figure S2. 16S rDNA gene based phylogram. Phylogenetic relationship of the clinical isolate to other *Nocardia* species based on 16S rDNA gene sequence analysis.

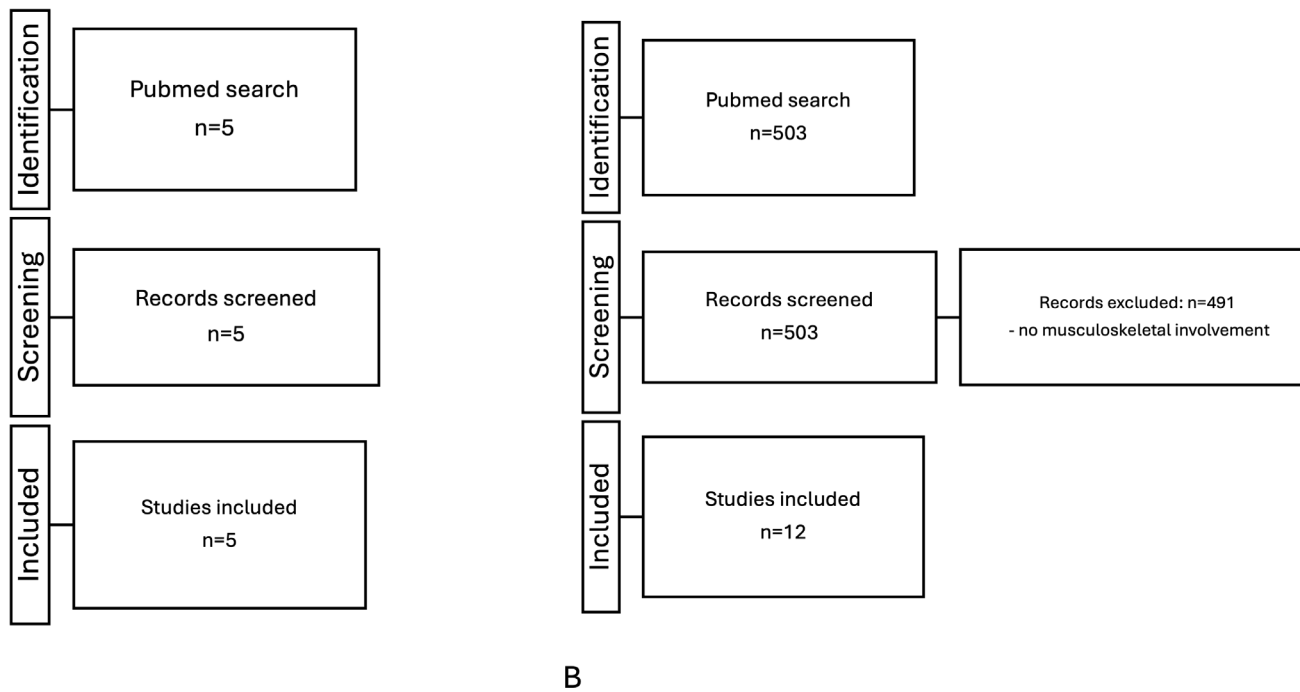


Figure S3. Flowchart of the literature search. A: database search for “Nocardia” AND “Bursitis”, B: database search for “Nocardia farcinica”

Table S1: Paper identified reporting cases with *Nocardia bursitis*. Overview of previously reported cases of bursitis caused by *Nocardia* species, including patient characteristics, causative strain, treatment, and outcomes.
Abbreviations: n.a., not available.

Paper	Age, sex	Location	<i>Nocardia</i> species	Dissemination	Disease/immunosuppression	Antibiotic agent	Treatment duration	Outcome	Intervention
Khodadadi et al. (USA, 2024) (1)	57, M	prepatellar	<i>Nocardia nova</i>	n.a.	n.a.	trimethoprim-sulfamethoxazole	89 days	good	none
Anazonwu et al. (USA, 2013) (2)	49, F	prepatellar	<i>Nocardia nova</i>	none	diabetes, fibromyalgia	trimethoprim-sulfamethoxazole	6 months	good	bursectomy
Chowdahary et al. (USA, 1988) (3)	n.a.	olecranon	<i>Nocardia asteroides</i>	n.a.	n.a.	sulfonamide	n.a.	good	None
Leitner et al. (Austria, 2013) (4)	66, M	olecranon	<i>Nocardia asiatica</i>	none	n.a. / no immunosuppression	linezolid	1 month	good	bursectomy
Park et al. (Korea, 2014) (5)	67, M	bursitis malleolus lateralis	<i>Nocardia farcinica</i>	none	diabetes, hypertension / no immunosuppression	cefoperazone cepodoxime	1 month	good	bursectomy
This case	75, M	olecranon	<i>Nocardia farcinica</i>	none	rheumatoid arthritis/ methotrexate, prednisone, golimumab	trimethoprim-sulfamethoxazole, amoxicillin-clavulanate	3 months	good	bursectomy

Table 2: Overview of musculoskeletal infections caused by *Nocardia farcinica*. Summary of published cases describing musculoskeletal manifestations of *N. farcinica*, including underlying diseases, therapeutic approaches, and outcomes.

Abbreviations: d, diagnostic; t, therapeutic; n.a., not available.

Paper	Age, Sex	Location / Manifestation	Dissemination	Disease / Immunosuppression	Antibiotic agent	Treatment duration	Outcome	Intervention (d) = diagnostic (t) = therapeutic
Schiff et al. (USA, 1992) (6)	54, M	face / cutaneous abscess, osteomyelitis	none	none / none	trimethoprim-sulfamethoxazole, amikacin	8 weeks	good	surgical debridement (t)
Torres et al. (Spain, 2000) (7)	85, M	clavicle, supraclavicular / abscess	pulmonary	lymphoma, tuberculosis / steroids, cyclophosphamide	cefotaxime	20 days	lethal	skin biopsy (d)
Audenaert et al. (Belgium, 2004) (8)	68, M	knee / arthritis	pulmonary	diabetes, COPD / steroids	trimethoprim-sulfamethoxazole	6 months	good	knee puncture (d,t)
Malani et al. (USA, 2006) (9)	65, F	thigh / abscess	none	Hodgkin lymphoma / methotrexate, vinblastine, bleomycin	trimethoprim-sulfamethoxazole	3 months	good	incision and drainage (t)
Budzik et al. (USA, 2012) (10)	78, M	knee / arthritis	pulmonary	none / none	trimethoprim-sulfamethoxazole	unclear	lethal	knee aspiration (d,t)
De Nardo et al. (Italy, 2013) (11)	54, M	thigh / phlegmon	none	diabetes, leprosy / none	trimethoprim-sulfamethoxazole, imipenem	1 month	good	incision and drainage (t)
Park et al. (Korea, 2014) (5)	67, M	malleolus lateralis / bursitis	none	diabetes, hypertension / none	cefoperazone, cefodoxime	1 month	good	bursectomy (t)
Ishiguro et al. (Japan, 2017) (12)	82, M	knee / arthritis	pulmonary	diabetes / None	Imipenem, levofloxacin	5 months	good	knee puncture and continuous drainage (d,t)
Garofalo et al. (Italy, 2021) (13)	70, M	lower limb / abscess	none	minimal change disease / prednisone	Imipenem, moxifloxacin	6 months	good	surgical drainage (t)
Acuner et al. (Turkey, 2021) (14)	37, M	forearm / abscess	none	none / none	trimethoprim-sulfamethoxazole, imipenem, amikacin	7 months	good	drainage and debridement (t)

Zhang et al. (China, 2021) (15)	51, M	forearm, leg / abscess	pulmonary	eosinophilic granulomatosis / steroids	sulfamethoxazole, linezolid, moxifloxacin, minocycline	13 months	good	incision and drainage (t)
Smit et al. (Netherlands, 2022) (16)	42, M	musculus psoas / abscess	none	none / none	trimethoprim-sulfamethoxazole	10 months	good	drainage (t)
Wang et al. (China, 2024) (17)	56, M	abdomen / abscess	pulmonary	diabetes, gout / prednisolone	ceftriaxone, imipenem	6-12 months	good	puncture (d)
This case	75, M	olecranon / bursitis	none	rheumatoid arthritis / methotrexate, prednisone, golimumab	trimethoprim-sulfamethoxazole, amoxicillin-clavulanate	3 months	good	bursectomy (t)

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