IDCases 29 (2022) e01590

Contents lists available at ScienceDirect

IDCases

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Case report

Septic arthritis due to *Nocardia brasiliensis* and a review of nocardiosis as a cause of arthritis

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ARTICLE INFO

Keywords:
Nocardia brasiliensis
Septic arthritis
Immunocompromised
Disseminated nocardiosis

ABSTRACT

Bacteria of the genus *Nocardia* are implicated in several disease processes but are a rare cause of septic arthritis. Typically, the cause of *Nocardia* septic arthritis is dissemination from a pulmonary infection in an immunocompromised host. Herein we present a case of a 64-year-old male who had received a long course of prednisone for membranous nephropathy and developed a septic arthritis due to *Nocardia brasiliensis*. He was treated sequentially with trimethoprim-sulfamethoxazole and amoxicillin-clavulanate, linezolid and amoxicillin-clavulanate, tigecycline and amoxicillin-clavulanate, and omadacycline and amoxicillin-clavulanate. To our knowledge, only two prior cases of *Nocardia brasiliensis* septic arthritis without antecedent trauma to the joint or local skin breakdown have been reported. A review of the literature identified 19 other cases of *Nocardia* septic arthritis. This case reinforces the need to consider *Nocardia* infection in the differential diagnosis in the immunocompromised patient with concurrent pulmonary infection and septic arthritis.

Introduction

Bacteria of the genus *Nocardia* are rod-shaped Gram-positive bacteria that are ubiquitous in the environment. To date, 119 species of *Nocardia* have been described, with 40 of these being pathogenic in humans. The primary infection sites in humans are the lungs and the skin. Most cases of human nocardiosis have been reported in the immunocompromised, with diminished host immune response contributing to the dissemination of the organism [1]. Septic arthritis secondary to *Nocardia spp.* may arise in the setting of disseminated infection or cutaneous inoculation [2,3]. We report a rare case of *N. brasiliensis* septic arthritis and a literature review was conducted of the reported cases of septic arthritis due to *Nocardia* species.

Case

A 64-year-old male, presented with severe right knee pain for 10 days, with inability to ambulate because of the pain. His co-morbidities included gout, coronary artery disease, membranous nephropathy, with resultant stage 3 chronic kidney disease, type 2 diabetes, and hypertension. He had been treated with prednisone for his renal disease for four months prior to his presentation; his current dose was 20 mg/day, decreased from 60 mg/day that he had received for the prior month. He reported smoking marijuana but denied other illicit drug use. He worked as a landscaper and house painter but denied a history of penetrating trauma to the knee. He had no prior history of knee pain.

On admission, his WBC was 19.2 K/μL (reference range (RR) 4–10 K/μL), with 95.3 % neutrophils, 1.9 % lymphocytes, absolute lymphocyte count 365/μL (RR 900–3600/μL); and 1.6 % monocytes. His hemoglobin level was 10.8 g/dL (RR 11.5–14.9 g/dL), platelets 240 K/μL (RR 150–400 K/μL), and creatinine at baseline and on presentation was 1.44 mg/dL (RR 0.50–1.10 mg/dL). A urine protein/creatinine ratio performed a week before his presentation was 3.8 (> 3.5 is nephrotic range proteinuria), which had been improving with corticosteroid use. On physical exam, he was noted to have tenderness, erythema, and swelling of his right knee; an X-ray showed a moderate-sized joint effusion with diffuse soft tissue swelling and moderate-to-severe osteoarthritic change. An aspiration of the right knee was performed, and the synovial

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https://doi.org/10.1016/j.idcr.2022.e01590

Received 21 May 2022; Received in revised form 19 July 2022; Accepted 27 July 2022

Available online 28 July 2022

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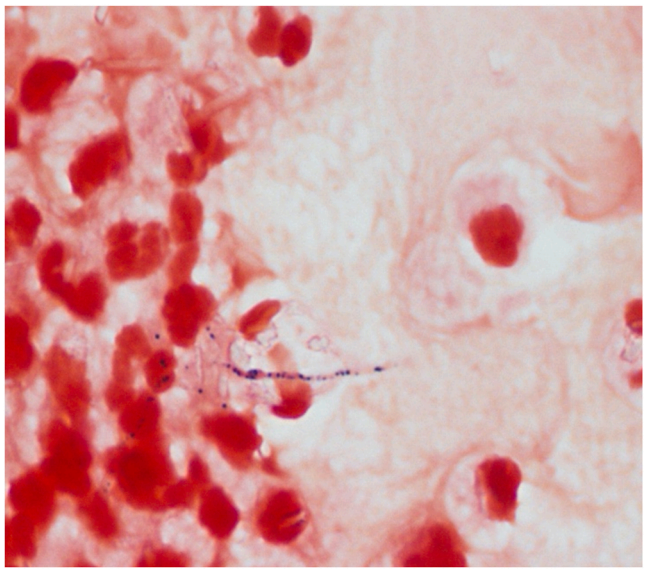

Fig. 1. Photomicrograph of the Gram stain of the synovial fluid showing Gram-positive filamentous rods (1000-×).

Table 1
Susceptibility testing of *Nocardia brasiliensis* isolate of case patient.

Antibiotic	MIC, μg/mL	Interpretation
Amikacin	2	Susceptible
Amoxicillin + Clavulanic acid	12	Susceptible
Cefepime	> 32	Resistant
Ceftriaxone	> 64	Resistant
Ciprofloxacin	> 4	Resistant
Clarithromycin	8	Resistant
Doxycycline	4	Intermediate
Imipenem	16	Resistant
Linezolid	2	Susceptible
Minocycline	2	Intermediate
Moxifloxacin	4	Resistant
Tobramycin	1	Susceptible
Trimethoprim + Sulfamethoxazole	5	Susceptible
Tigecycline	0.12	No interpretive breakpoint

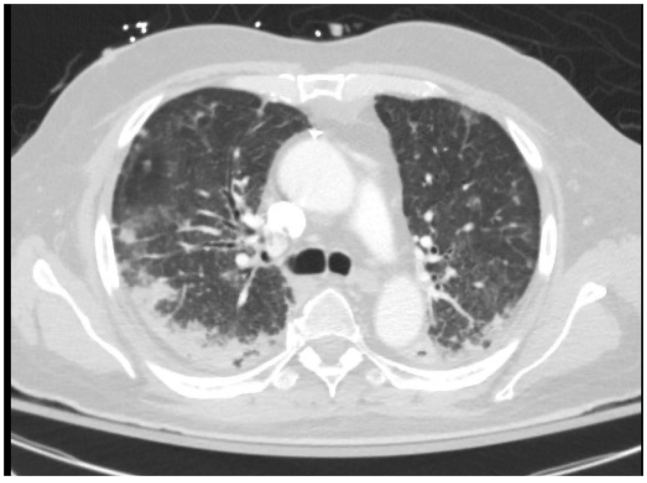

Fig. 2. Initial computerized tomograph of the lungs showing nodular and diffuse infiltrates.

fluid showed 64,000 white blood cells/mm^3, with a 78 % polymorphonuclear predominance, and calcium pyrophosphate crystals. A Gram stain of the synovial fluid showed gram-positive filamentous rods (Fig. 1). With the diagnosis of septic arthritis, a right knee arthrotomy was performed. Intraoperatively, the joint capsule was excised, and copious purulent material was evacuated.

A culture of the synovial fluid aspirate grew *Nocardia brasiliensis* in seven days; the isolate's susceptibility testing results are noted in Table 1. He was initiated on linezolid (600 mg orally twice a day) and amoxicillin-clavulanate (875–125 mg twice a day). After two weeks of therapy with linezolid, susceptibilities of this *Nocardia brasiliensis* isolate were available and therapy was changed to trimethoprim-sulfamethoxazole (800–160 mg twice a day) and amoxicillin-clavulanate.

Given the patient's dyspnea, a computerized tomograph (CT) of the chest was obtained, which showed diffuse bilateral patchy ground glass and nodular opacities. (Fig. 2). With the recognized neurotropism of *Nocardia* species [2], magnetic resonance imaging of the brain was performed, which was negative.

Following 21 days of therapy with trimethoprim-sulfamethoxazole and amoxicillin-clavulanate combination therapy, he developed acute kidney injury with creatinine increasing from 0.8 mg/dL to 1.35 mg/dL and he was transitioned to linezolid and amoxicillin-clavulanate for two weeks. Thereafter, given concern for linezolid toxicity with a longer duration of use, he was transitioned to intravenous tigecycline and amoxicillin-clavulanate for an additional six weeks. Given that the tigecycline minimum inhibitory concentration (0.12 μg/mL) was likely to correlate with susceptibility to omadacycline, treatment was continued with omadacycline 300 mg once daily and amoxicillin-clavulanate to provide a completely oral regimen. The patient completed a total of nine months of therapy. Six weeks after discontinuing antibiotics, and 10.5 months following his initial diagnosis, he was seen in clinic. There was no evidence of relapse and full recovery of knee function. The patient's dyspnea had significantly improved but had not fully resolved. A repeat chest CT showed significant interval improvement of the multiple ground glass and nodular opacities, with decreased reticulation from interlobular septal thickening. Residual nodular opacities were still evident.

Methodology

A PubMed search from Jan 1980 to March 2021 was performed using the search terms '*Nocardia*' or '*Nocardia brasiliensis*', and 'septic arthritis' or 'pulmonary infection', 'bacteremia' or 'disseminated infection' or 'extrapulmonary'. Additional references were obtained by Google Scholar searches with identical keywords. References published in English were reviewed. A total of 28 cases of *Nocardia* septic arthritis have been reported in the adults from disseminated infection originating in the lung, or from cutaneous inoculation through trauma; one case occurred in a child [8]. Among the adults with *Nocardia* septic arthritis, cases of disseminated nocardiosis occurring after skin breakdown were excluded. This review presents the twenty cases of non-traumatic septic arthritis due to infection by various *Nocardia* species. Only two of the previously reported *Nocardia* septic arthritis cases were attributed to *N. brasiliensis.*

Discussion

Forty species of *Nocardia* have been implicated as human pathogens [1]. Based on their biochemical properties and antimicrobial susceptibility patterns, the nocardiae are classified into various species complexes [3]. The *Nocardia asteroides* complex is the group clinically known to cause the vast majority of human *Nocardia* infections [4,5]. Distinct from this group is *Nocardia brasiliensis*, previously classified in the genera *Streptothrix*, *Oospora*, and *Actinomyces*. This species is distinct based on certain biochemical reactions, including nitrate reduction, urea hydrolysis, casein and tyrosine decomposition, and variable decomposition of hypoxanthine. Its distinctive drug susceptibility pattern is that of typical resistance to ciprofloxacin and clarithromycin,

Table 2
Summary of previously reported cases of non-traumatic *Nocardia* septic arthritis.

Nocardia species [Ref.]	Age (yrs)/ Sex	Risk factors	Joint	Other sites involved	Antimicrobial treatment/duration of therapy	Clinical outcome
asteroides [15]	30/F	Renal transplant, on immuno-suppressives	Knee	None	TMP-SMX/12 mos	Cure
asteroides [16]	46/M	Autoimmune disease, on corticosteroids	Wrist	None	TMP-SMX/unspecified Duration	Unknown
asteroides [17]	46/M	HIV, IVDU	Knee	Pneumonia	TMP-SMZ for 3 weeks, then minocycline/unspecified duration	Cure
asteroides [18]	50/F	Heart transplant	Hip	None	TMP-SMX/> 30 mos	Unknown
asteroides [19]	52/M	Renal transplant	Knee	Abscess on back	TMP-SMX/6 mos	Cure
asteroides [20]	56/F	DM, temporal arteritis, on corticosteroids	Knee	Pneumonia, pustules, tongue	TMP-SMX/2 weeks	Cure
asteroids [21]	64/M	CLL, with immune suppression	Knee	None	Imipenem/cilastatin + TMP-SMX/unspecified duration	Death due to other cause
asteroides [22]	82/M	corticosteroid to the joint, gout, osteoarthritis	Knee	None	TMP-SMX/6–12 mos	Unknown
farcinica [23]	55/M	HSCT, steroid-dependent chronic GVHD; DM, chronic renal failure	Knee	None	Ceftriaxone/levoflox, 7 days; meropenem/amikacin, 15 days; meropenem/levoflox, 10 days, + linezolid, days 35–52; levoflox/minocycline, 5 mos; clinical failure; then ticarcillin-clavulanate/TMP-SMX until day 172, cefuroxime/TMP-SMX for 6 mos, then addition of doxycycline	Death due to other cause
farcinica [24]	68/M	DM, COPD, on corticosteroid	Knee	Pneumonia	TMP-SMX/6 mos	Cure
farcinica [25]	78/M	Not specified	Knee	Pneumonia, pleural effusions	TMP-SMZ/11 days	Death from respiratory failure
farcinica [26]	82/M	DM, Pneumoconiosis	Knee	Right empyema	Levoflox/6 mos	Cure
brasiliensis [27]	4/F	None	Proximal IP joint	Cutaneous vesicles	TMP-SMX/6 mos	Cure
brasiliensis [7]	36/M	Astrocytoma, on dexamethasone	Knee	Pneumonia	TMP-SMZ/amikacin/20 days	Death from respiratory failure
brasiliensis [this case]	64/M	Membranous nephropathy, on corticosteroids; DM; gout	Knee	Pneumonia	TMP-SMX/amox-clav, 21 days; linezolid/amox-clav, 14 days; tigecycline/amox-clav, 42 days; omadacycline/amox-clav, 6 mos	Cure
cyriacige-orgica [28]	38/F	SLE, on immuno-suppressives	Knee	None	TMP-SMX/12 mos	Cure
cyriacige-orgica [23]	60/M	HSCT, acute GVHD	Knee	None	Imipenem/amikacin, 1 mo; cefuroxime/doxycycline, 1 year	Cure
pseudo brasiliensis [29]	86/M	Aortic valve replacement; pacemaker; stress fracture of the leg	Knee	None	TMP-SMX/ciprofloxacin/unspecified duration	Cure
caviae [29]	75/M	Osteoarthritis, DM	Knee	None	TMP-SMX/amox-clav for 1.5 mos, then amox-clav for 3 mos	Cure
nova [22]	64/M	DM, lymphoma in remission, myasthenia gravis, on immune suppression	Knee	None	Imipenem for 15 days, followed by imipenem/ceftriaxone until day 39, then ceftriaxone/TMP-SMX until day 80, followed by amoxicillin/clarithromycin until death.	Death due to other cause

Abbreviations: Amox-clav, amoxicillin-clavulanate; CLL, chronic lymphoid leukemia; DM, diabetes mellitus; GVHD, graft versus host disease; HSCT, hematopoietic stem cell transplant; IP, interphalangeal; levoflox, levofloxacin; SLE, systemic lupus erythematosus; TMP-SMX, trimethoprim-sulfamethoxazole; IVDU, Intravenous drug use.

and susceptibility to amoxicillin-clavulanate, trimethoprim-sulfamethoxazole, and minocycline [5], although resistant strains may occur. *Nocardia asteroides* is the most geographically widespread of the genus, with most American cases of *N. brasiliensis* infection occurring in the southeastern and southwestern USA [6]. *Nocardia brasiliensis* has been rarely reported as a cause of septic arthritis [7].

Pulmonary involvement is the most common manifestation of nocardiosis with underlying lung disease play a predisposing role [5,8,9]. Our case patient likely had pulmonary nocardiosis resulting from inhalation, which manifested as chronic dyspnea and radiographic abnormalities.

Bacteremia and disseminated nocardiosis arise from primary pulmonary infection in most cases. Defective cell-mediated immunity increases susceptibility to *Nocardia* bacteremia and disseminated infection. This is typically seen in the setting of organ transplantation, leukemia, the acquired immunodeficiency syndrome (AIDS), or long-term therapy with cytotoxic agents and/or corticosteroids [9]. A low mean lymphocyte percentage $\leq$ 7.8 % (RR 20–40 %) of the total leukocyte count has been shown to be a predisposing factor for nocardiosis [10]. This patient had a lymphocyte percentage of 1.3 % due to the use of high-dose prednisone for four months. We suspect that the pulmonary infection in our immunocompromised patient eventually resulted in dissemination to a joint thereafter. Although no specific exposure risk factors were identified in our patient, as a landscaper, he was exposed to dust. It is also stipulated that marijuana smoking may have played a role in the development of the nocardiosis. Marijuana may be contaminated by environmental organisms and previously immunocompromised patients have developed respiratory infections from marijuana exposure, including aspergillosis and nocardiosis [11–13]. Marijuana smoking also impairs the microbicidal function of alveolar macrophages [14] which may potentially predispose to infection.

Table 2 provides a review of reported cases of *Nocardia* septic arthritis without antecedent trauma. Of the 20 cases of *Nocardia* arthritis without joint trauma, 40 % were due to *N. asteroides*, 20 % to *N. farcinica,* 15 % to *N. brasiliensis,* 10 % to *N. cyriacige-orgica,* and single cases ascribed to *N. pseudo brasiliensis, N. caviae,* and *N. nova*. This

showcases the rarity of *N. brasiliensis* as a cause of septic arthritis. The cases were reported in patients 30–86 years of age; 75 % occurred in men. Seventy-five percent of the involved patients had underlying immunosuppression, either from corticosteroid use (as in this case), organ transplantation, or HIV infection. Seventeen of the 20 cases involved the knee joint with involvement of the wrist, the hip, and small joints of the hand reported as well. Six of these cases had concomitant pulmonary involvement, indicating the need to assess other sites for *Nocardia* infection if there is joint involvement, as was noted in our case. In terms of therapy, trimethoprim-sulfamethoxazole was the mainstay of treatment; in 90% of cases this agent was employed as monotherapy or in combination with other antibiotics. Forty percent of case patients received 8–12 months of therapy; our patient received 9 months. In one case, clinical cure was documented after only two weeks of therapy [20]. Cure was achieved in 55 % of the cases (11 out of 20 cases, including this case), with death due to other causes occurring in 25 % of cases prior to treatment completion; no outcome was reported in 20 % of the cases. No deaths were ascribed to nocardiosis.

Increasing levels of resistance are known for *N. farcinica* and resistance to TMP-SMX and sulfonamides has been reported across all *Nocardia* species [31]. Minimum inhibitory concentrations of tigecycline are noted to be generally 1–2 dilutions within the 100 % inhibition omadacycline minimum inhibitory concentration values for non-tuberculous mycobacteria, indicating that this could be an alternative regimen for treatment [32], prompting its use in our patient.

Nocardia infections are known in literature to primarily occur in immunocompromised individuals. *Nocardia* species known to disseminate to various body sites, including the central nervous system. However, there have been infrequent instances of isolated *Nocardia* joint infections, without a previous history of penetrating trauma to the joint, or local cutaneous disruption. We highlight the importance of considering this diagnosis, especially in the setting of an immunocompromised patient. It is imperative to follow cultures until finalization, as diagnostic yield is significantly increased with longer incubation times, even up to 4 weeks [30]. A high degree of suspicion for nocardiosis is indicated if there is initial Gram stain evidence of filamentous gram-positive rods in a clinical specimen.

Funding

This research was funded by the Department of Veterans Affairs and the University of Texas Health San Antonio.

CRediT authorship contribution statement

Divya Chandramohan: patient care, data analysis, Writing – original draft, Writing – review & editing. **Heat Javeri:** patient care, data analysis, Writing – review & editing, Funding acquisition. **Gregory Anstead:** data analysis, Writing – review & editing, Funding acquisition. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare that they have no conflict of interest regarding the publication of this paper.

Acknowledgments

Sponsors did not play any role in the preparation of this manuscript.

Institutional Review Board Statement

Ethical review and approval were waived for this study because the patient received standard of care only and written informed consent was obtained from the patient.

Ethical approval

The patient was treated with the standard of care. No research studies were conducted. Thus, the study does not require Institutional Review Board Approval.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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