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Severe Cutaneous Dissemination of Nocardia niigatensis in a Patient Receiving Immunosuppression for Myasthenia Gravis: A Case Report

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Abstract

Nocardia niigatensis is a rare pathogen whose infections can be difficult to recognize in patients receiving immunosuppressive therapy for myasthenia gravis (MG). The clinical presentation may mimic other infectious or inflammatory conditions, delaying diagnosis.

A 36-year-old man receiving high-dose prednisolone for MG presented with loss of consciousness, fever, and progressive lower-limb swelling after months of dysphagia and muscle weakness. He required urgent resuscitation and intensive care. Examination showed widespread erythematous, scaly lesions and a large fluctuant collection in the right leg. Incision and drainage yielded 2.5 L of pus. Microscopy demonstrated branching filamentous gram-positive bacilli, and culture yielded chalky colonies. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and 16S ribosomal RNA gene sequencing identified the organism as *N. niigatensis*. During prolonged trimethoprim-sulfamethoxazole therapy, the skin lesions improved noticeably within one month.

This case highlights the need to consider nocardiosis in immunosuppressed patients with atypical cutaneous or systemic infections. Early molecular identification at the species level and targeted antimicrobial therapy are essential for optimal outcomes.

Categories: Neurology, Infectious Disease
Keywords: 16s rrna sequencing, cutaneous nocardiosis, immunosuppression, maldi tof identification, myasthenia gravis, nocardia niigatensis, opportunistic infection, prednisolone therapy

Introduction

Nocardia species are filamentous Gram-positive environmental bacteria that can cause serious opportunistic infections, especially in individuals with impaired immunity [1]. More than 30 species are known to infect humans [2,3]; *Nocardia cyriacigeorgica*, *Nocardia farcinica*, and *Nocardia brasiliensis* are the most common. *Nocardia niigatensis* is one of the rarer of these.

To our knowledge, this is the second reported human infection with *N. niigatensis* in the setting of immunosuppression [4] and the first reported in a patient with myasthenia gravis (MG). The species was first identified in human clinical specimens from Japan in 2004 [5]; the only previously reported human infection was a cutaneous case in a renal transplant recipient, confirmed by 16S ribosomal RNA (rRNA) sequencing [4]. A review of *Nocardia* species spanning 2006 to 2021 identified no additional cases, underscoring the rarity of this pathogen [6].

Although nocardiosis itself is uncommon, it has particular relevance in patients with MG. Fewer than 30 cases have been described, nearly all in individuals receiving corticosteroids or other immunosuppressive therapy [2]. In patients with MG, aspiration risk, thymic-related T-cell dysfunction, and chronic corticosteroid exposure may increase susceptibility [2].

Cutaneous nocardiosis can resemble fungal or mycobacterial infection, delaying recognition [4,7]. Because the skin findings can vary widely and do not reliably indicate systemic involvement [8], accurate species-level identification is important. Matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry and 16S rRNA gene sequencing improve diagnostic precision, although uncommon species may still be misidentified [6,9,10].

Additional case reports are valuable because of the extreme rarity of human infection caused by *N. niigatensis* and the lack of data on its clinical manifestations, optimal management, and microbiological

identification. This report expands the clinical spectrum of *N. niigatensis* infection and highlights diagnostic considerations that may assist in the identification of this rare pathogen in immunosuppressed patients.

Case Presentation

A 36-year-old man from Bangladesh with a history of MG presented in February 2025 with fever, bilateral lower-limb swelling and redness for 10 days, and loss of consciousness for one day. Three months before admission, he developed dysphagia and proximal muscle weakness, which progressively worsened over time to generalized weakness and fatigue in the days leading up to his admission. Ten days before admission, he complained of high-grade fever, bilateral lower-limb swelling, and redness, during which he was taking a course of oral Augmentin from another clinic without any notable improvement. On the day of admission, he deteriorated further with loss of consciousness, for which he was brought to the hospital emergency department. He was receiving prednisolone 60 mg orally once daily and pyridostigmine 60 mg orally twice daily for MG. His social and occupational histories were unknown. Examination revealed a temperature of 40°C, hypotension, tachycardia, tachypnea, pallor, and generalized edema. The lower limbs showed redness, tenderness, swelling, and fluctuance, particularly on the right side. Scattered skin lesions were present throughout the body, predominantly on the limbs and neck. The lesions were erythematous, hyperpigmented, and scaly (Figure *1A*). Many resembled tinea, with peripheral hyperpigmentation and central fading (Figure *1B*). In the emergency department, he sustained two cardiac arrests and was resuscitated, intubated, and mechanically ventilated. He received fluids and inotropic support with adrenaline, noradrenaline, and vasopressin before immediate transfer to the intensive care unit. He underwent incision and drainage, and multiple abscesses extending from the right thigh to the medial malleolus were evacuated. Drainage yielded 2,500 mL (2.5 L) of pus, and tissue and pus samples were sent to the microbiology laboratory. Initial differential diagnoses included septic shock with severe soft tissue infection, cellulitis, necrotizing fasciitis, toxic shock syndrome, particularly *Streptococcus pyogenes* infection, disseminated nocardiosis, atypical mycobacteria, invasive fungal infection (e.g., invasive mold disease or disseminated tinea), sporotrichosis, and leishmaniasis.

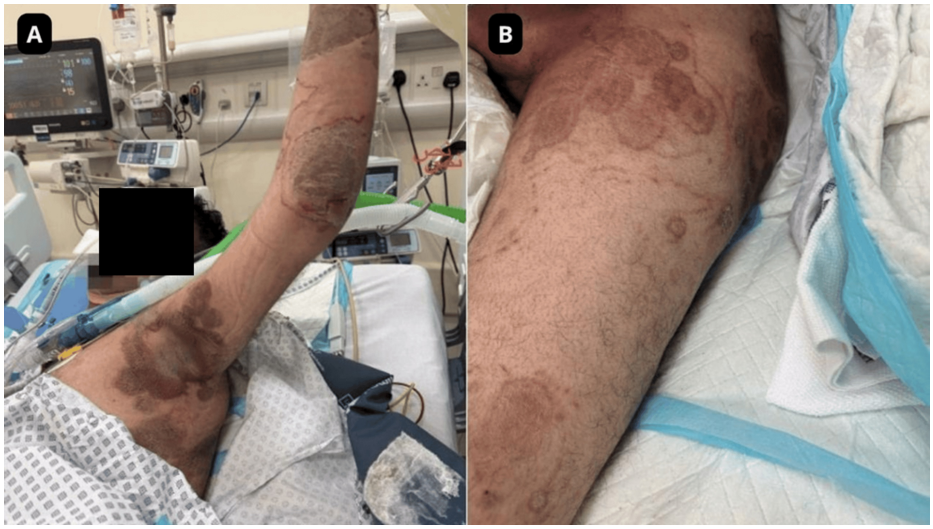


FIGURE 1: Skin lesions before treatment

A) Erythematous, hyperpigmented, scaly lesions; B) Tinea-like lesions with peripheral hyperpigmentation and central fading.

The patient provided written and signed consent, allowing publication of this identifiable facial image in an open-access journal.

Laboratory testing showed an elevated white blood cell count and C-reactive protein, acute kidney injury, elevated alanine aminotransferase and alkaline phosphatase, and an abnormal coagulation profile, consistent with an acute-phase response (Table *1*).

Test		Result	Reference range
CBC	WBC	38.33 × 10^3/µL	4.0-11.0 × 10^3/µL
	Absolute neutrophils	36 × 10^3/µL	2.0-7.5 × 10^3/µL
	Hemoglobin	10 g/dL	12-16 g/dL (female)/13.5-17.5 g/dL (male)
	Platelets	167 × 10^3/µL	150-400 × 10^3/µL
CRP		365 mg/L	<5 mg/L
eGFR		51 mL/min/1.73 m^2	>90 mL/min/1.73 m^2
LFT	Total bilirubin in serum/plasma	7.38 µmol/L	3-21 µmol/L
	Total protein in serum/plasma	48.57 g/L	64-80 g/L
	ALT	318.31 U/L	0-41 U/L
	ALP	171 U/L	40-129 U/L
	Albumin	25 g/L	35-52 g/L
Coagulation profile	Prothrombin time	17.7 s	9-14 s
	INR	1.59	0.8–1.1
	Activated partial thromboplastin time	28.5 s	20-30 s
	Thrombin time	18.1 s	12-23 s
	Fibrinogen	5.60 g/L	1.5-4 g/L

TABLE 1: Complete blood count and blood biochemical findings

CBC: complete blood count; WBC: white blood cells; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; LFT: liver function tests; ALT: alanine aminotransferase; ALP: alkaline phosphatase; INR: international normalized ratio

The sepsis workup included cultures of blood, sputum, and urine, together with HIV screening. Blood cultures showed no growth after five days of aerobic and anaerobic incubation. Sputum culture grew normal respiratory flora, and there was no growth in urine culture. HIV serology screening (enzyme-linked immunosorbent assay (ELISA)) was non-reactive. The drained tissue and pus were incubated aerobically at 37°C and under anaerobic conditions. Microscopy of the tissue and pus revealed branching filamentous Gram-positive bacilli (Figure *2A*). After 48 hours of aerobic incubation, both tissue and pus cultures showed growth on blood and chocolate agar. The colonies were dry and chalky, with white and orange pigmentation (Figures *2B*-*2C*).

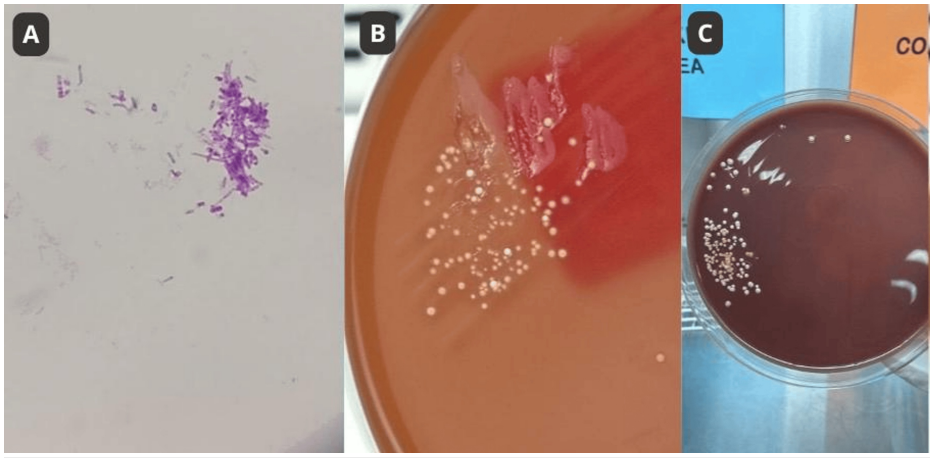


FIGURE 2: Microscopy and culture findings

A) Branching filamentous Gram-positive bacilli; B) Dry, chalky colonies on blood agar; C) Dry, chalky colonies on chocolate agar. The colonies showed white and orange pigmentation.

MALDI-TOF analysis using the MALDI Biotyper (MBT Compass 4.1.100; Bruker Daltonics GmbH, Bremen, Germany) initially identified the isolate as *N. niigatensis*. A reference laboratory confirmed the species by 16S rRNA gene sequencing. After DNA extraction, the 16S rRNA gene was amplified by polymerase chain reaction (PCR) and sequenced using the universal primers 27F and 519R with AmpliTaq Gold™ 360 Master Mix (Applied Biosystems™, Tokyo, Japan) (Table *2*).

Primer	Primer sequence (5’ to 3’)	Amplicon size	Reference
27F	AGAGTTTGATCMTGGCTCAG	~ 500 bp	1
519R	GWATTACCGCGGCKGCTG	-	1

TABLE 2: Universal primers for 16S rRNA amplification and sequencing

rRNA: ribosomal RNA

Partial 16S rRNA sequencing (464 bp) demonstrated 100% homology with *N. niigatensis*, with a query coverage of 99% and the closest reference accession being NR_117402. Similarity to related species was as follows: *Nocardia acididurans* (97.84%), *Nocardia tengchongensis* (97.41%), and *Nocardia acidivorans* (97.41%). The consensus sequence was compared with sequences in the NCBI (National Center for Biotechnology Information) database using 16S rRNA BLASTn (Basic Local Alignment Search Tool for nucleotide sequences) [11]. Both the NCBI and Greengenes databases returned a 100% match to *N. niigatensis*. The microbiological findings are summarized in Table *3*.

Test/panel	Finding
HIV serology screen (ELISA)	Nonreactive
Blood culture (aerobic and anaerobic)	No growth after 5 days
Sputum culture	Normal respiratory flora
Urine culture	No growth
Tissue and pus microscopy	Branching filamentous gram-positive bacilli
Culture on blood and chocolate agar	Dry, chalky, white and orange colonies
MALDI-TOF mass spectrometry	*Nocardia niigatensis*
16S rRNA sequencing	*Nocardia niigatensis*

TABLE 3: Microbiological findings

HIV: human immunodeficiency virus; ELISA: enzyme-linked immunosorbent assay; MALDI-TOF: matrix-assisted laser desorption/ionization time-of-flight; rRNA: ribosomal RNA; NCBI: National Center for Biotechnology Information

Antimicrobial susceptibility testing (AST) was unavailable. Repeated cultures of endotracheal secretions yielded no *Nocardia*. Chest radiography showed bilateral opacities, more pronounced on the right (Figure *3A*). The findings were likely suggestive of aspiration pneumonia, consistent with the recently increasing muscular weakness and correlating with the respiratory culture results. Chest computed tomography (CT) was not performed because of clinical instability and financial constraints. Brain CT showed diffuse edema and loss of gray-white matter differentiation, consistent with hypoxic-ischemic encephalopathy, without cerebral abscesses (Figure *3B*). Brain magnetic resonance imaging (MRI) showed findings similar to those on CT (Figure *3C*).

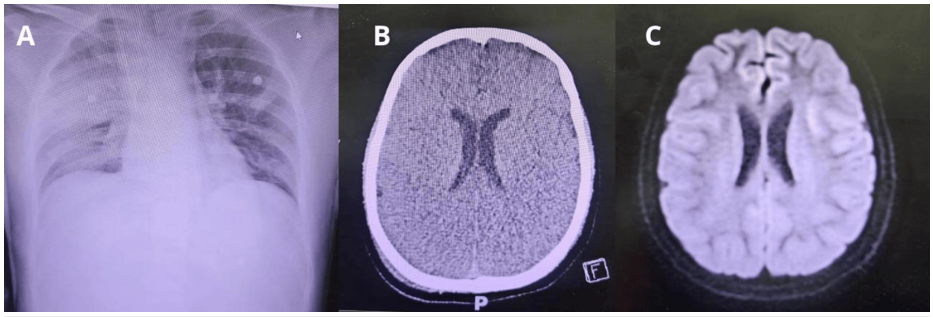


FIGURE 3: Chest and brain imaging

A) Chest radiograph; B) Brain computed tomography (CT); C) Brain magnetic resonance imaging (MRI).

The patient initially received intravenous meropenem 2 g three times daily, vancomycin 1 g twice daily, and clindamycin 900 mg three times daily. Intravenous immunoglobulin (30 g once daily for four days) was subsequently added because of concern for necrotizing fasciitis and group A streptococcal (GAS) toxic shock syndrome. Antifungal therapy was also considered; however, it was not initiated empirically. After *Nocardia* was identified, vancomycin and clindamycin were discontinued. Intravenous trimethoprim-sulfamethoxazole (TMP-SMX) 15 mg/kg/day (TMP component) IV in two divided doses was started, and meropenem was continued for one month. After one month of treatment, the lesions showed reduced erythema, scaling, swelling, and hyperpigmentation (Figures *4A*-*4C*). Subsequently, the patient completed five months of oral TMP-SMX monotherapy 5 mg/kg/day (TMP component) in two divided doses for a total treatment duration of six months of TMP-SMX, during which the skin lesions improved. Cutaneous improvement continued throughout the six-month treatment course. However, the patient sustained a permanent hypoxic-ischemic brain injury secondary to cardiac arrest. Consequently, his neurological status remained unchanged throughout hospitalization, with a Glasgow Coma Scale score of 8/15 (E4V1M3). He remained bedridden, dependent on enteral feeding via a nasogastric tube, tracheostomy care, and minimal ventilatory support until discharge. No further follow-up was available, as the patient returned to his home country after six months.

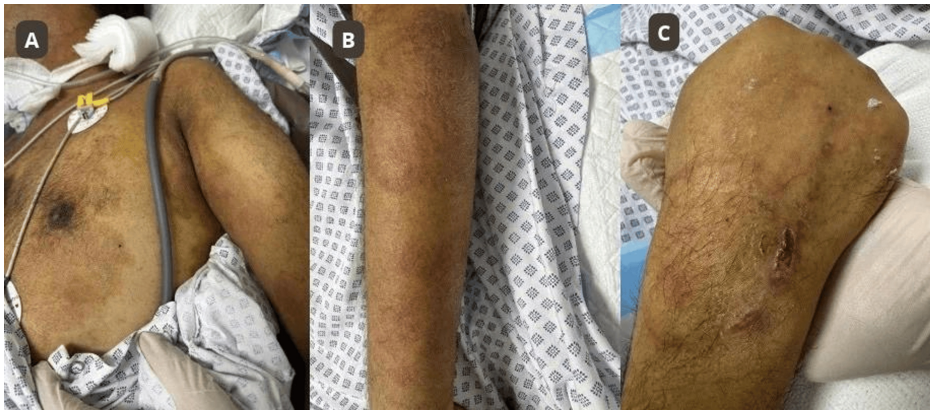


FIGURE 4: Skin lesions after 1 month of treatment

A) Left photograph of the post-treatment skin lesions; B) Middle photograph of the post-treatment skin lesions; C) Right photograph of the post-treatment skin lesions. Hyperpigmentation and erythema were reduced.

The patient provided written and signed consent, allowing publication of this identifiable facial image in an open-access journal.

Discussion

N. niigatensis remains rarely reported in clinical practice despite its identification in 2004 [5,12]. As the name indicates, Niigata is a city in Japan where this strain was first isolated [5]. Only one previous human infection with *N. niigatensis* has been reported. In South Korea, a 57-year-old renal transplant recipient developed multiple cutaneous abscesses and rapidly expanding fungating masses on her leg. The diagnosis was confirmed by 16S rRNA PCR, and TMP-SMX was effective over six months [4]. A systematic review of *Nocardia* species from 2006 to 2021 identified no further cases involving this species [6].

Fewer than 30 cases of nocardiosis in patients with MG have been reported. This opportunistic infection occurs in immunosuppressed individuals, including those receiving corticosteroids or other immunosuppressants [5,13,14]. Its prevalence is unknown because the available evidence consists solely of case reports.

In nature, *Nocardia* species are widespread in decayed plant material, soil, and dust grains [1,5,15,16]. Infection is most commonly acquired through inhalation of aerosolized bacteria from these environments [1,4]. Direct cutaneous inoculation after injury is less common. This route has been documented after trauma involving contaminated soil, penetrating wounds, insect or wasp stings, and surgical wounds [7]. Muralidhar Reddy et al. analyzed 21 cases of nocardiosis in patients with MG. Potential contributors to susceptibility in patients with MG include pulmonary aspiration caused by progressive respiratory and bulbar muscle weakness; autoimmune and thymic T-cell dysfunction; prolonged immunosuppressant use, particularly prednisolone at doses above 10 mg daily; and immunomodulatory therapy. Age above 50 years, male sex, and chronic lung disease further increase risk. The most frequent pathogens in the previous study were *N. farcinica*, *Nocardia asteroides*, and *N. brasiliensis* [2].

The lungs are the most common site of infection, followed by the skin, brain, muscles, bones, and eyes. Cutaneous disease may result from disseminated infection or arise as a primary infection after direct inoculation through the skin [17]. Cutaneous nocardiosis can present in several forms: pustules, plaques, granulomatous or fungating nodules, or ulcers [4,7]. These lesions can mimic fungal infection, tuberculosis, atypical mycobacterial infection, syphilis, sporotrichosis, or leishmaniasis [4,7]. The varied presentations complicate diagnosis and may delay treatment [7]. In our patient, the ringworm-like lesions closely resembled disseminated tinea (dermatophytosis). Recognition of these varied presentations supports prompt diagnosis and appropriate treatment.

The genus *Nocardia* now includes approximately 100 species, more than 30 of which are linked to human infections. *N. asteroides*, *N. brasiliensis*, *Nocardia otitidiscaviarum*, and *Nocardia transvalensis* are responsible for the vast majority of infections [18]. *Nocardia* are branching filamentous gram-positive rods that stain partially acid-fast because their cell walls contain mycolic acids. The colonies are dry and chalky and may be white, orange, red, or gray. They grow aerobically and require prolonged incubation of two to three weeks. Recovery of *Nocardia* species may be enhanced by inoculation onto selective media, such as modified Thayer-Martin medium and buffered charcoal-yeast extract [13,18,19]. *N. niigatensis* grew within 48 hours in our patient, as in the previously reported case [4].

Definitive diagnosis relies on culture of *Nocardia* from clinical samples and microscopic examination,

supported by molecular identification [2,20].

Antimicrobial susceptibility varies markedly among *Nocardia* species; some, including *N. farcinica*, are intrinsically resistant to multiple β-lactams [3,21]. Under the CLSI (Clinical and Laboratory Standards Institute) M24 standard, *Nocardia* susceptibility is assessed by broth microdilution to determine minimum inhibitory concentrations [9,22].

Clinically relevant *Nocardia* isolates should ideally undergo species-level identification and AST to guide treatment [3,17,19]. Molecular species identification can inform an assessment of the species' typical antimicrobial susceptibility and intrinsic resistance patterns reported in the literature [9], particularly when standardized CLSI testing is unavailable at the treating hospital or a reference laboratory.

Molecular biology includes a variety of tests for species-level identification. These include sequencing of the 16S rRNA, secA1, DNA gyrase subunit B (gyrB), rpoB, hsp65, and sodA genes; whole-genome sequencing; and multilocus sequence typing, with sodA sequencing mainly supported by CLSI MM18 [6,9,17,23,24]. Of these methods, 16S rRNA gene sequencing is the most widely used for species identification and phylogenetic analysis [6]. Full-length or near-full-length 16S rRNA gene sequences are recommended for reliable identification [6,24,25]. However, 16S rRNA sequencing may not distinguish closely related taxa, such as *Nocardia abscessus* versus the *Nocardia nova* complex [6,26]. Proteomic methods such as MALDI-TOF are widely used with favorable results. Their reliability depends on database coverage, and uncommon species may be misidentified [6,9,10]. Restriction fragment length polymorphism analysis, DNA probes, ribotyping, randomly amplified polymorphic DNA analysis, and biochemical or serological methods are not recommended for *Nocardia* speciation because of their major limitations [6].

A limitation of this report is that species identification relied on partial 16S rRNA gene sequencing rather than multilocus or whole-genome analysis. However, the sequence showed complete 100% identity with the *N. niigatensis* reference strain, 99% query coverage with the *N. niigatensis* reference sequence (NR_117402), and clear separation from the closest phylogenetically related species, all of which demonstrated less than or equal to 97.84% sequence similarity. This represents a divergence of approximately 2.2% to 2.6% from the nearest related species over a highly conserved gene. No competing species showed 99%-100% identity in the analyzed region, and the chromatograms were of high quality, with no ambiguous bases. The concordant results obtained from independent reference databases (NCBI and Greengenes) further strengthen the assignment. Taken together, these findings, in agreement with the MALDI-TOF result, provide strong molecular evidence supporting the identification of the isolate as *N. niigatensis*.

Luu et al. conducted a case series and population-level analysis of six cases of cutaneous nocardiosis. Grocott methenamine silver staining demonstrated filamentous bacteria in all cases. Histology also showed pseudoepitheliomatous hyperplasia, neutrophilic infiltrates, necrotizing granulomas, sinus formation, and neutrophilic panniculitis [7].

Cutaneous nocardiosis in immunosuppressed patients may be accompanied by systemic involvement. The appearance and severity of skin lesions do not reliably indicate internal organ involvement. Some cases with systemic involvement have few cutaneous lesions or only subcutaneous nodules [8]. Accordingly, chest CT and brain MRI or contrast-enhanced CT are important for evaluating possible systemic involvement, particularly of the brain and lungs [7,8,17].

For disseminated nocardiosis or brain or severe lung involvement, empirical treatment generally combines TMP-SMX with a carbapenem; amikacin, linezolid, or a cephalosporin may also be included. Treatment can then be tailored to susceptibility results and the known susceptibility profile of the species [17]. No species-specific antibiotic regimen for *N. niigatensis* has been established in the available literature. In the only previously reported case, *N. niigatensis* abscesses were successfully treated with intravenous TMP-SMX for six months, initially combined with ampicillin-sulbactam for 10 days [4]. Our patient showed marked improvement after one month of intravenous TMP-SMX plus meropenem, followed by oral TMP-SMX monotherapy, for a total treatment course of six months. Reducing or temporarily withholding immunosuppressive agents until the infection resolves is also important [17]. Some antibiotics and other drugs can exacerbate myasthenic crises and should be avoided. Examples include fluoroquinolones, aminoglycosides, macrolides, and tetracyclines [2,27].

Host immunity and disease severity influence prognosis. Estimated cure rates are approximately 100% for cutaneous and soft tissue disease, 90% for pulmonary infection, and as low as 50% for brain involvement or disseminated disease [2].

Conclusions

This case adds to the limited literature available on human infection caused by *N. niigatensis* and, to our knowledge, is the first reported case in a patient with MG. It points to the possibility of an opportunistic nocardial infection in the setting of prolonged immunosuppression and shows the benefit of combining molecular methods with conventional microbiological findings for the correct identification of the species.

Targeted antimicrobial therapy and multidisciplinary management resulted in a favorable clinical outcome.

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: Amira Al Dhabbari, Amal Al Jabri, Mubarak Al Yaqoobi, Hala Al Hinai, Mustafa Al Shaaibi, Azza Al Rashdi

Acquisition, analysis, or interpretation of data: Amira Al Dhabbari, Amal Al Jabri, Amal Al Kalbani

Drafting of the manuscript: Amira Al Dhabbari, Amal Al Jabri, Mubarak Al Yaqoobi, Hala Al Hinai, Mustafa Al Shaaibi, Azza Al Rashdi, Amal Al Kalbani

Critical review of the manuscript for important intellectual content: Amira Al Dhabbari, Amal Al Jabri, Mubarak Al Yaqoobi, Hala Al Hinai, Mustafa Al Shaaibi, Amal Al Kalbani

Supervision: Amira Al Dhabbari, Amal Al Jabri, Mubarak Al Yaqoobi, Mustafa Al Shaaibi, Azza Al Rashdi, Amal Al Kalbani

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **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.

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