SYNTHĒSIS · CASE REPORT

Disseminated Nocardiosis – A rare case of Nocardia kroppenstedtii in a Ghanaian adult – A case report

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Abstract

Aims: The aim of this report is to present a rare case of disseminated Nocardia kroppenstedtii infection in an immunocompromised patient with Systemic Lupus Erythematosus (SLE), highlighting the diagnostic challenges and management complexities with a resource-limited setting.

Case presentation: A 43-year-old Ghanaian woman with a history of SLE and Lupus nephritis on long-term corticosteroids presented with respiratory distress, fever, and cutaneous ulcers. Clinical evaluation revealed sepsis from infected cutaneous ulcers and bilateral pneumonia, acute kidney injury, and features of lupus myocarditis. Cultures from her leg ulcers and blood cultures isolated Nocardia kroppenstedtii. Imaging later confirmed a perinephric abscess, and bilateral pneumonia after which disseminated nocardiosis was suspected. She was treated with a prolonged course of trimethoprim-sulphamethoxazole, amikacin and meropenem, alongside management of lupus myocarditis and AKI. Despite limited diagnostic tools, a multidisciplinary approach facilitated recovery back to her baseline.

Conclusion: This case underscores the importance of maintaining high clinical suspicion for nocardiosis in immunocompromised patients, especially where laboratory capabilities are limited. Early culture sampling and tailored antimicrobial therapy are key to survival in disseminated disease.

Introduction

Nocardia is a genus of aerobic, filamentous, variably acid-fast, gram-positive bacteria that can cause systemic or localised infections. They cause mainly opportunistic infections, but about a third of infections occur in immunocompetent individuals. The most commonly pathogenic Nocardia spp. are Nocardia asteroides complex and Nocardia brasiliensis, among others. Nocardia is found in all regions of the world. Its incidence in the United States in the 1970s was approximately 500-1000 cases per year [Beaman et al., 1976], but this number has likely increased due to the increase in population size and the higher prevalence of immunosuppression. The bacteria naturally reside in soil, decaying vegetation, salt- and fresh-water environments, and has even been isolated from house dust and tap water [Goodfellow et al., 1983]. It is usually transmitted to humans via inhalation or inoculation by penetrating trauma.

The most common risk factor for infection is Immunocompromising conditions such as Glucocorticoid and other Immunosuppressive medications, organ transplant recipients, HIV infections, and primary immune deficiencies [Ledermen et al., 2004]. Treatment with glucocorticoids is the most frequently reported risk factor. Other immunosuppressive

medications associated with Nocardia infection are calcineurin inhibitors (tacrolimus, cyclosporin), monoclonal antibodies to B-cells, T-cells, and tumour necrosis factor-alpha (rituximab, alemtuzumab, infliximab) [Peleg et al., 2007]. One condition where many of these medications are used is Systemic Lupus Erythematosus (SLE). SLE patients have T lymphocyte immune dysfunction due to the disease itself, and also take some immunosuppressant medications listed above [Gil-Rodríguez et al., 2025].

Most often, Nocardia causes a localised infection at the site of entry (lungs, skin), but can disseminate to other organs. The most common site of dissemination is the brain [Beaman et al., 1976]. The infection typically has a subacute course that progresses for several weeks and results in focal symptoms at the site of entry. Disseminated disease is more common in immunocompromised individuals, with rates as high as 43% in solid organ transplant recipients. Lung infections can present with fever, cough, night sweats, fatigue, weight loss, dyspnoea, haemoptysis, pleuritic chest pain and it can also progress to acute respiratory failure in severe cases [Lerner et al., 1996]. Pulmonary Nocardiosis can not only disseminate to the brain but also to adjacent structures, causing empyema thoraces, mediastinitis, pericarditis, intra-abdominal abscesses and superior vena cava syndrome. CNS involvement is typically due to dissemination from the lungs and presents with brain abscess or meningitis, but may also rarely involve the spinal cord.

Case Report

A 43-year-old female with a known history of Systemic Lupus Erythematosus (SLE) with Lupus Nephritis, diagnosed in 2020, for which she was taking prednisolone, hydroxychloroquine and mycophenolate mofetil. She presented to the Emergency room on account of gradually worsening difficulty breathing of 5 days and cough productive of sputum with streaks of blood of 2 weeks duration. She also described that 1 week prior to all this she noticed painful deep-seated boils (one on each leg and two at her back), which without any manipulation ulcerated after a few days and discharged pus. This was associated with intermittent high grade fever.

At presentation, she looked acutely ill and in respiratory distress. She was also mildly pale, warm to touch with axillary lymphadenopathy and bipedal edema. She was tachypnoeic with a respiratory rate of 52 cycles per minute and had bronchial breath sounds and coarse crepitations in the entire right hemithorax and left middle and lower zones. Cardiovascular examination showed tachycardia (PR=124bpm) and slightly laterally deviated apical beat. Patient was alert and fully oriented to time, place and person and had no CNS abnormalities on examination. She also had three ulcers on her lower limbs, one on the posterior aspect of the left leg measuring 3x2cm and two on the anterior aspects of both legs each, measuring 2x2cm, with yellowish discharge. Ulcers were noted to have deep tunnelling, however grossly sparing the underlying bone.

Investigations showed anaemia (Haemoglobin 8.1g/dL), Leukocytosis (15.23x109/L), with neutrophilia (14.09x109/L). Kidney Function tests (KFT) showed an elevated urea and creatinine, with reduced estimated GFR of 53mL/min/1.73 m2. Urine examination showed moderate proteinuria and C-reactive protein was elevated to 341mg/L. A chest X-ray done showed bilateral patchy (almost ground-glass) opacification spanning the entire right hemithorax especially concentrated around the upper and hilar regions and lower and middle zones on the left. A chest CT scan was also done to that effect which showed multiple heterogeneously enhancing lesions of bilateral lung fields predominantly in the

peribronchovascular distribution with enlarged lymph nodes suggestive of infective etiology, with differentials of an atypical pneumonia. An Abdominal USG showed an enlarged left kidney with a hypoechoic collection in the lower pole, that was suspected to be a perinephric abscess. This collection was drained via ultrasound guidance - a total of 100mls of pus. An initial diagnosis of “Sepsis secondary to infected ulcers ?cause to rule out Cutaneous Tuberculosis (Scrofuloderma) and a bilateral pneumonia to rule out Pulmonary Tuberculosis/Disseminated Tuberculosis complicated by Acute kidney injury in a known SLE patient with Lupus Nephritis” was made.

She was started on broad-spectrum empiric antibiotic therapy and urine outputs were being monitored closely as her kidney function gradually continued to deteriorate and patient was now in septic shock. Blood and wound swab cultures isolated Nocardia Kroppenstedtii after 12 days of incubation. With this new development her diagnosis coupled with the chest CT findings and the history of immunosuppressive drugs use, brought about the possibility of Disseminated Nocardiosis to the forefront of the differential diagnosis. Diagnosis was reviewed to “Septic shock secondary to Disseminated nocardiosis (cutaneous, abdominal and lung involvement) complicated by Acute kidney injury” and her antibiotics were reviewed to IV meropenem, amikacin and oral trimethoprim-sulphamethoxazole. This revised treatment was done for a total of 6 weeks. During this time a contrast-enhanced brain CT was also done to rule out CNS involvement of the disease, which came back unremarkable.

As her treatment progressed, her respiratory distress lessened as evidenced by a reduction in her tachypnoea and need for supplementary O2 to maintain adequate SpO2. She was also gradually weaned off O2 and started on chest physiotherapy. Her renal function improved back to her baseline and repeat chest imaging showed radiographic improvement. She was eventually discharged on oral trimethoprim-sulphamethoxazole to complete a whole 12 months of therapy.

Discussion

Nocardia spp. is a rare pathogenic organism mainly affecting immunocompromised individuals. The relative rarity of this organism and the lack of definitive laboratory testing for nocardia in a large part of the country makes it a difficult clinical problem to diagnose (Lucas et al., 1994). Secondly, due to the high prevalence of Pulmonary Tuberculosis infections in both Immunocompromised and immunocompetent individuals, and the relative similarity in clinical presentation, there is a high likelihood of misdiagnosis of pulmonary Nocardiosis as Pulmonary TB (Kandi et al., 2015).

In the index case, a 43-year-old Ghanaian female with SLE and lupus nephritis on long-term systemic corticosteroids and other immunosuppressive medications presented with cough, shortness of breath and deep-seated ulcers with tunnelling sinus tracts. Through imaging and culture of specimens from multiple relevant sites, Nocardia kroppenstedtii was isolated which led to the diagnosis of Disseminated nocardiosis.

Nocardia kroppenstedtti is a very rare species and per our research there are only 6 other documented cases globally. There has been no definitive published case study of Nocardia infection in Ghana, but a study done in 2018 regarding the prevalence of Mycobacterium tuberculosis and Nocardia spp. in patients suspected of Pulmonary TB showed a prevalence of 18.3% for Nocardia infection. A majority of Nocardia patients also had HIV infection (90.9%), and of the total population studied, 16.7% had Nocardia and HIV co-infection.

A case report in Morocco defines a 22-year-old man, with a history of Pulmonary TB and subsequent relapse, that was treated, presented with chronic cough, hepatosplenomegaly and purulent subcutaneous swellings. Chest CT scan showed alveolar condensations with pleurisy. Bacteriological analysis confirmed Nocardia infection. He was treated with antibiotics and discharged when healthy (Rhofir et al., 2017).

Another case study in China details a 61-year-old man with macroglobulinemia on prednisolone who presented with recurrent productive cough, repeated fever, dyspnoea, joint pain, and headache. CT showed multiple nodular shadows in the left lung, with axillary and mediastinal lymphadenopathy. He also developed scalp ulcers and a brain abscess. Blood culture, sputum smear, bronchial washing fluid and scalp smear revealed Nocardia spp. and Gene sequencing confirmed Nocardia vulneris. He was treated successfully with Trimethoprim-Sulphamethoxazole, Linezolid and Amikacin (Qiu et al., 2022).

Management of Disseminated Nocardiosis involves prompt recognition and a high index of suspicion especially in the immunocompromised population, as this variant of disease has potential to progress very quickly leading to prolonged hospital stays and worsening prognosis. Upon early suspicion of the disease, it is imperative to notify relevant laboratories about this so that adequate incubation conditions and time can be allowed (3-21 days of incubation) before isolating the organism. Upon isolation, PCR or mass spectrometry is used to arrive at the offending strain after which sensitivities are also assessed. Antibiotic sensitivities vary for the various species depending on the individual virulence of the strain. However widely used and sensitive antibiotics for disseminated disease include trimethoprim-sulphamethoxazole, imipenem, amikacin and linezolid.

Conclusion

This case highlights the diagnostic and therapeutic challenges posed by Nocardia kroppenstedtii (a rare species of an already uncommon pathogen) especially in an immunocompromised host in a resource-limited setting. The clinical presentation mimicked more common infections such as pulmonary tuberculosis, and the delay in culture growth further complicated the diagnosis. Despite these hurdles, a high index of clinical suspicion, early empiric broad-spectrum antibiotic coverage, and a multidisciplinary approach were crucial in identifying and successfully managing disseminated nocardiosis. This case also underscores the importance of considering nocardial infections in the differential diagnosis of sepsis or pneumonia in immunocompromised patients. Timely microbiological sampling and tailored, prolonged antimicrobial therapy can significantly improve outcomes, even in the absence of advanced diagnostic tools.

Consent

All authors declare that written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editorial office of this journal.

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