

Challenges in an Immunocompromised Host: A Case of Disseminated Nocardiosis

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Introduction

Nocardia farcinica is an aerobic, gram-positive bacteria from the genus *Nocardia*. It is one of the most clinically relevant species in the genus, and it is known for its pathogenic potential in humans, particularly in immunocompromised individuals such as those with underlying conditions, including HIV/AIDS, organ transplant recipients, and those on immunosuppressive medication.

We present a case of disseminated nocardiosis caused by a difficult-to-treat *N.farcinica* in a 68-year-old woman with a known history of systemic lupus erythematosus with concurrent use of high-dose prednisone, antiphospholipid antibody syndrome, and right renal vein thrombosis.

Case presentation

The patient presented to our hospital in November 2023 with a 2-week history of fever and dry cough. At admission, her laboratory exam revealed a white blood cell count of $14 \times 10^3/\mu\text{L}$ (CD4 count was 69 cells/ μL) and elevated C-reactive protein. Computed tomography imaging revealed diffuse and inhomogeneous

thickening of the pulmonary interstitium with areas of parenchymal consolidation and multiple nodular formations (**Fig.A**).

Sputum cultures and other microbiological test, including legionella and streptococcal urine antigen, acid-fast bacilli stains, PCR for *Mycobacterium tuberculosis*, galactomannan antigen, and 1,3- β -D-glucan of bronchoalveolar lavage, all returned negative results.

Blood cultures showed the presence of gram-positive bacilli, subsequently identified as *N.farcinica*. Treatment was initiated with sulfamethoxazole-trimethoprim (SMX-TMP) and amikacin (AMK), leading to clinical improvement. However, after 14 days of therapy, the patient developed leukopenia and anaemia, suspected to be myelotoxicity from the antibiotics; SMX-TMP was discontinued, and imipenem-cilastatin (IPM) was started, resulting in progressive resolution of leukopenia and anaemia. After 3 weeks of therapy, the patient's clinical and radiological conditions improved (**Fig.B**).

Given her recent myelosuppression and pharmacological interactions with other drugs, she continued moxifloxacin as a consolidative therapy after discharge.

Following discharge, the patient remained asymptomatic for 8 weeks before being readmitted due to fever and elevated inflammatory markers. Blood cultures once again revealed *N.farcinica* but with a modified resistance pattern, resulting in it being sensitive only to AMK, IPM, and linezolid. Based on the microbiological results, IPM and linezolid.

Based on the microbiological results, IPM and AMK treatment was restarted. After 4 weeks and negative blood cultures, the patient was discharged with a prolonged course of IPM and ongoing monitoring for any recurrence or complications related to *N.farcinica* infection.

Conclusion

In conclusion, this case highlights the challenges faced in diagnosing and treating disseminated nocardiosis, particularly when complicated by antimicrobial resistance with limited therapeutic options. Prompt identification of atypical pathogens, such as *N.farcinica*, and vigilant monitoring is crucial for optimal management and improved outcomes.

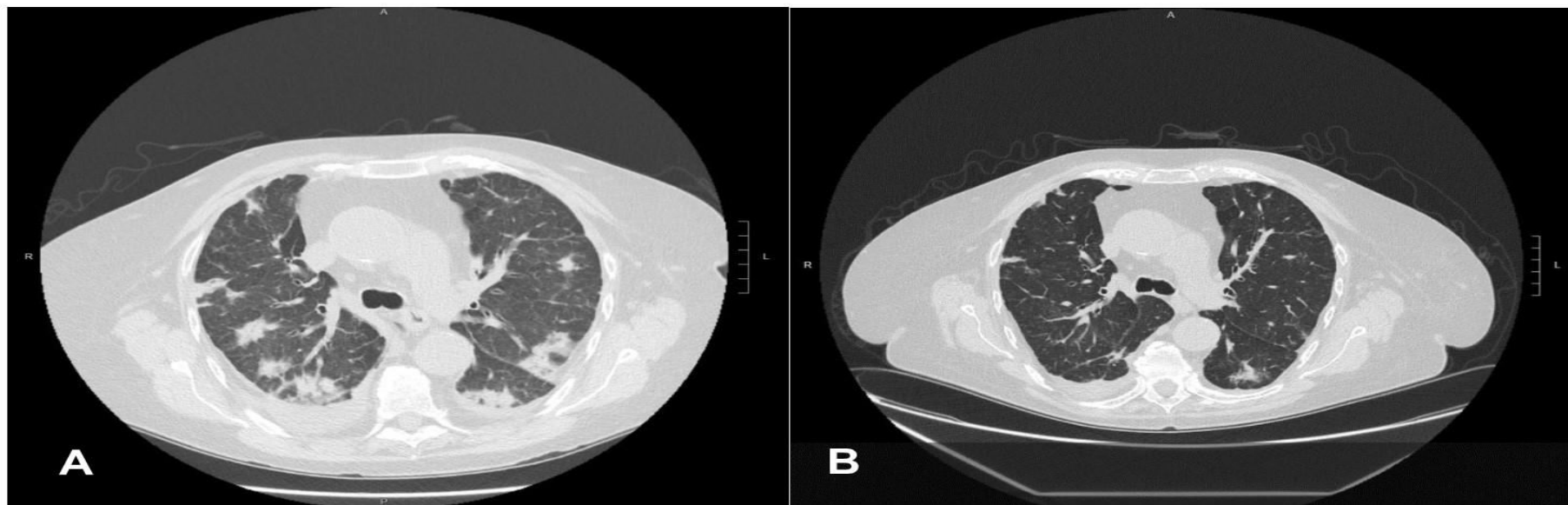


Figure A-B. Radiological finding in pulmonary nocardiosis: CT of the chest at the basal (**A**) and after XXI days of specific antibiotic therapy (**B**).

References

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