Background
Nocardiosis is an uncommon bacterial infection, often requiring prolonged therapy with multiple antimicrobials. Antibiotic allergies and side effects may complicate management.

Clinical Features
A 79-year-old Caucasian male presented with abdominal pain and constipation. Relevant past medical history included non-Hodgkin’s Lymphoma, in remission after completing chemotherapy six months earlier. He had also previously experienced an allergy to trimethoprim/sulfamethoxazole (TMP/SMX), resulting in a rash, with no features of Steven Johnson’s syndrome. Whilst in hospital he developed ataxia and monocular visual disturbance. Magnetic Resonance Imaging (MRI) Brain and CT Chest demonstrated multiple lesions. Lung biopsy samples grew Nocardia cyriacigeorgica, and he was assessed as having disseminated Nocardiosis of the lungs and brain.

Interventions & Case Progress
Induction therapy was commenced; amikacin 15mg/kg intravenously daily, meropenem 2g intravenously three times daily, and TMP/SMX 320/1600mg orally three times daily. Due to the known allergy, he underwent desensitisation prior to commencing treatment, guided by the Immunologist and Infectious Diseases (ID) pharmacist (Figure 1). Therapeutic drug monitoring (TDM) for amikacin was conducted by the ID pharmacist. TDM analysis demonstrated adequate exposure but inadequate clearance of the medication, which was managed by increasing the dosing frequency from 24 to 36 hours (Figure 2). Nocardia sensitivities returned on day 11 of treatment, showing resistance to meropenem. Based on these sensitivities, meropenem was replaced with ceftriaxone. Repeat imaging at four weeks revealed no change in the size of lesions, suggesting sub-optimal response to antibiotics. The lesions were not amenable to surgical intervention. As the patient was clinically unchanged, the decision was made to continue with active treatment. During treatment, the patient developed pancytopenia and folate deficiency (Figure 3), with concern for TMP/SMX contributing, in addition to underlying patient factors. Oral folic acid was recommended by the ID pharmacist to minimise toxicity, with further investigations into underlying aetiology.

Outcomes
Audiodiometric monitoring revealed hearing loss, and amikacin was ceased after six weeks of treatment. The patient was transferred to a country hospital to complete a further six weeks of IV ceftriaxone, with a plan to continue TMP/SMX for a minimum of 12 months. Our patient developed pancytopenia and folate deficiency after prolonged treatment with TMP/SMX. Long-term administration of TMP/SMX has been shown to cause pancytopenia and reduce serum folate levels (2), however this is thought to be relatively rare (3). TMP/SMX-induced pancytopenia and low folate has been shown to be responsive to folic acid. In this case, although the folate level improved with oral folic acid, the pancytopenia did not resolve. He subsequently underwent bone marrow biopsy which demonstrated hypocellular bone marrow, likely secondary to the previous chemotherapy.

Conclusion
Multiple pharmacy interventions were required to optimize therapy and minimise adverse effects of antimicrobial therapy. This case has shown that clinical pharmacists can aid in the management of complex infections, such as disseminated Nocardiosis.

References

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**Clinical Pharmacist Involvement in the Management of Disseminated Nocardiosis**

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