

Severe Opportunistic Infection in SLE: A Case of Disseminated Nocardiosis Mimicking Tuberculosis Post-Rituximab

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Introduction:

Patients with autoimmune diseases especially those on immunosuppression are susceptible to opportunistic infections.

Nocardiosis is a rare opportunistic infection which is commonly seen in immunocompromised patients such as solid organ transplant recipients, hematopoietic stem cell transplantation, cancer and immunosuppression for autoimmune diseases.

Common sites of infection are the lungs, central nervous system (CNS) and skin. We hereby report a case of active SLE with Disseminated Nocardiosis.

Report:

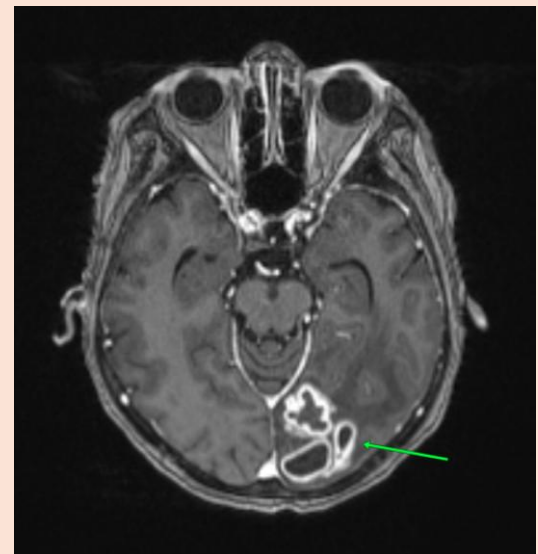
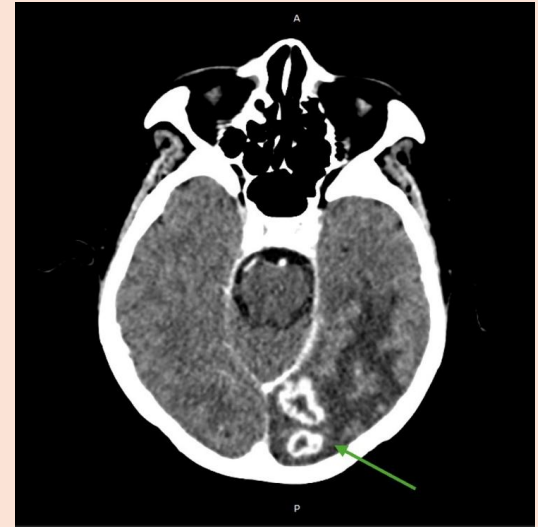
This case report describes a 61-year-old woman with a long history of Systemic Lupus Erythematosus (SLE) diagnosed in 2008, with the manifestations of lupoid hepatitis, autoimmune haemolytic anaemia (AIHA), thrombocytopenia, low C4 and positive autoantibodies (ANA, anti-dsDNA, anti-RNP, anti-Ro and anti-La). Over the years, she experienced recurrent flares of AIHA, necessitating escalation of immunosuppressive therapy from Azathioprine to a combination of Mycophenolate Mofetil (MMF) and Cyclosporin A.

The patient remained stable on MMF maintenance until three years later when she experienced another severe AIHA flare (Hemoglobin 5.1 g/dL), leucopenia and lymphopenia despite optimized treatment and pulse IV Methylprednisolone. IV Rituximab 1g was administered. Before the 2nd dose of Rituximab dose at week 2 was given, the patient was admitted for *E.coli* septicaemia, bronchopneumonia with right upper lobe cavitating lesion and disseminated abscess over left upper back, right popliteal fossa and right kidney. TB workup and pus culture were negative. Broncho-alveolar lavage did not reveal any positive results for opportunistic infection. She was subsequently treated with IV Unasyn for 2 weeks and was discharged with oral Unasyn planned for another 4-6 weeks.

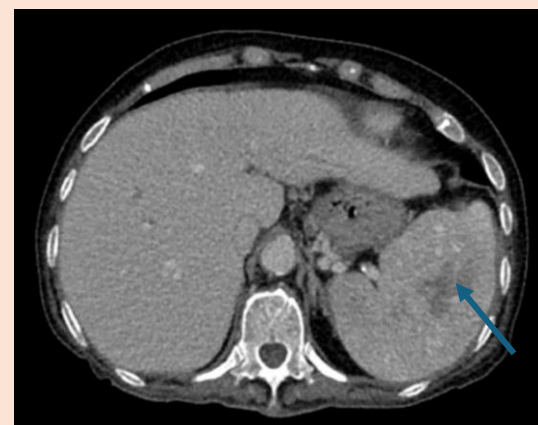
After discharge, the patient developed fever and chills, prompting readmission for recurrence of abscesses over the left posterior back and left popliteal fossa extending to distal thigh. Draining was done and IV Tazocin was initiated. The hospital stay was further complicated by status epilepticus secondary to multiloculated left occipital pyogenic abscess as shown in the CECT and MRI brain. Further investigations revealed AIHA exacerbation with post-Rituximab hypogammaglobulinemia, requiring IV immunoglobulin. Initial septic workup indicated acid-fast bacilli in the pus, raising suspicion of tuberculosis. However, cultures later identified *Nocardia farcinica*, leading to the diagnosis of disseminated nocardiosis, involving the brain, lungs, soft tissues, and kidney. The patient was treated with IV Meropenem for 6 weeks, followed by a year-long oral Bactrim.

Conclusion:

Dissemination of *Nocardia* spp. is favoured by auto-immune diseases, high dose corticosteroids and lymphopenia, as found in this patient. This case emphasizes the importance of vigilance in monitoring for infections in SLE patients, especially after immunosuppressive therapy, which can increase susceptibility to opportunistic infections. Managing such complex cases requires a delicate balance between carefully adjusting immunosuppressive therapy and minimizing the risk of infection.



Green arrow showing rim enhancing lesion in the left occipital lobe with perilesional oedema involving the occipito-parieto-temporal lobe



Blue arrow showing one of the largest hypodense lesion in the spleen causing splenomegaly