An unusual case of lung mass, bacteraemia and CNS disease in a renal transplant patient

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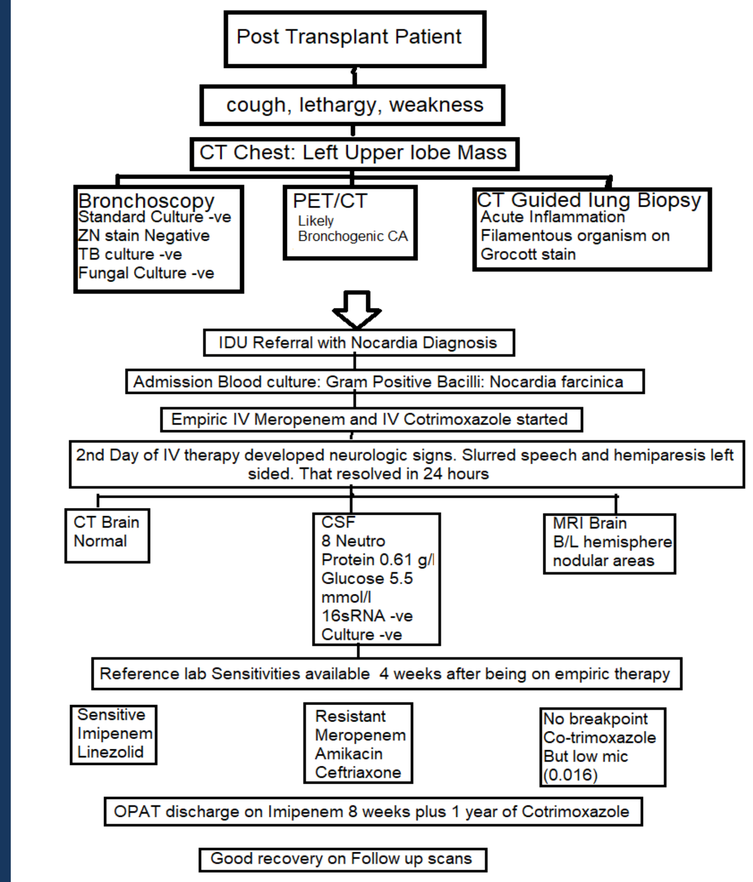
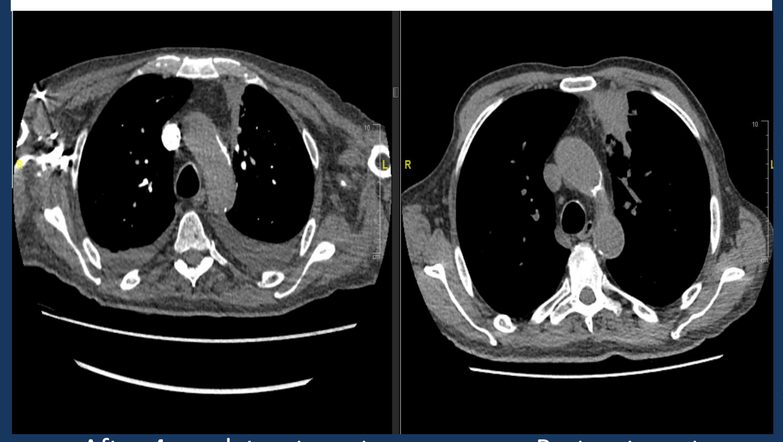
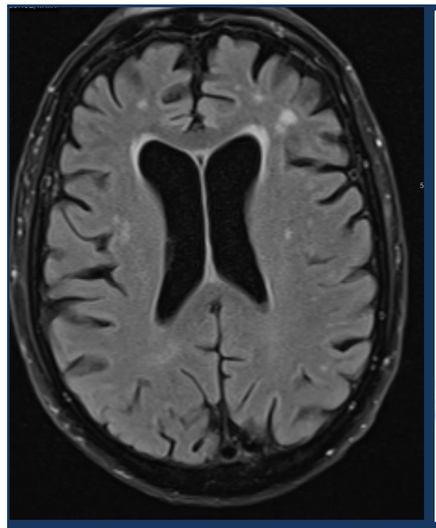
A 79 year old UK born Caucasian male
BG: renal transplant 2 years prior, type 2 diabetes mellitus and traumatic splenectomy, LTBI treatment Prior to Transplant
He had been experiencing
1)progressive lethargy, 2)weakness 3)mild cough for 3 months.
He was admitted under the Renal team for investigation and was noted to have a widespread vesicular rash. There were no night sweats and no phlegm production. Serum titres of cytomegalovirus were high (151,991 copies/ml), and he was commenced on ganciclovir.
CT Chest showed a left upper lobe mass, concerning for malignancy. Bronchoscopy did not show any malignant cells and was negative for acid fast bacilli, standard and mycobacterial culture. 6 weeks later he went on to have out-patient CT-guided lung biopsy. Histology showed acute inflammation with abscess material, with no granulomas or malignancy. ZN and modified ZN stain were negative, but filamentous organisms were noted on Grocott stain. Sub-culture grew a mucoid Pseudomonas and a Nocardia species, identified as *Nocardia farcinica* on MALDI. He was then referred to our infectious diseases unit. By the time of admission he was significantly frail, had lost weight with decreased mobility, and was febrile with raised inflammatory markers (CRP 219, WCC 23). Admission blood cultures showed a gram positive bacilli, subsequently confirmed as *Nocardia farcinica* by the Reference Laboratory. . He was empirically started on Meropenem and high dose IV co-trimoxazole (15mg/kg of trimethoprim) whilst awaiting sensitivities. 2 days into admission, he became confused and drowsy with slurred speech and left sided hemiparesis which resolved within 24 hours. CT Brain with contrast was normal. CSF sampling showed 8 white cells with mildly raised protein and normal glucose. CSF cytology showed active, chronic inflammatory cells. Microscopy, culture and 16S rDNA were negative. MRI brain showed multiple nodular areas of peripheral enhancement (2-3mm each) scattered throughout both hemispheres.
Subsequent tissue and blood culture MICs showed isolates to be sensitive to imipenem and linezolid, but resistant to meropenem, ceftriaxone, and amikacin. There were no co-trimoxazole breakpoints but MIC was low (0.016 mg/L). Meropenem was switched to imipenem, and oral co-trimoxazole continued. The patient made a good recovery. 4 weeks into treatment, CT chest showed improvement in the lung mass. He was discharged to complete 6-8 weeks of IV antibiotics via OPAT, followed by a minimum of 1 year of co-trimoxazole.

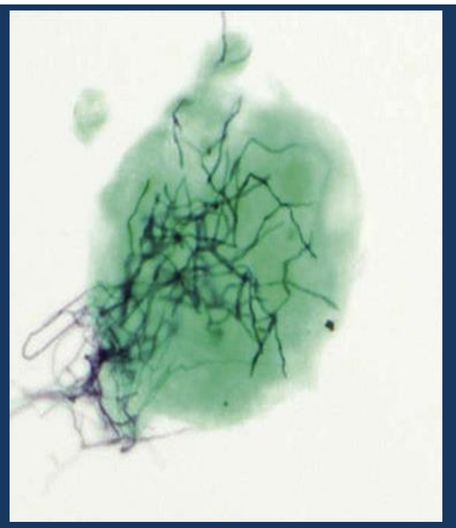




After 4 week treatment Pretreatment
CT Chest (Mediastinal window)



MRI Head (Flair)



Gorcottt Stain (Nocardia)

Learning points.
Nocardia is delicate gram positive branching rods that appear similar to actinomyces. It differs from actinomyces as it is aerobic and acid fast. Most common pathogenic species are N. brasiliensis, N. farcinica and N. cyriacigeorgica. Cell mediated immunity is crucial in containing Nocardia infection. Most common sites of infection are lung, CNS and skin. Lungs are primary site of nocardiosis in 60% of cases. CNS disease accounts for 20% of cases. When 2 or more non contiguous sites are involved then it is termed as disseminated nocardiosis, In routine aerobic culture, colonies appear as chalky white to yellow and brown and can take 5 to 21 days to grow. EUCAST has no recommendation on clinical breakpoints for Nocardia. CLSI recommend microdilution method for AST, while E-test has also been suggested by some experts. In Vitro AST results need to be interpreted with caution as no EUCAST breakpoints.This case also highlights that carabapenems may not always be interchangable for Nocardia treatment.

References

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