

CASE REPORT

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It never rains but pours: disseminated nocardiosis in a renal transplant patient from Nigeria – a case report

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Abstract

Background Nocardiosis is a rare opportunistic infection with high mortality and a tendency to relapse. The causative *Nocardia spp.* are filamentous Gram-positive aerobic bacteria. The lungs and brain are commonly affected and bacteraemia is rare, occurring in < 8% of cases. Solid organ transplant recipients receiving steroid immunosuppression are particularly at risk. The mainstay of treatment is prolonged antibiotic combination therapy, typically including trimethoprim-sulfamethoxazole.

Case presentation We present the case of a renal transplant recipient from Nigeria with disseminated *Nocardia cyriacigeorgica* infection with lung and brain abscesses and, unusually, bacteraemia. Most recommended antibiotic regimens were precluded by severe deterioration of graft function and anaemia whilst being unable to receive blood products due to religious beliefs. He was initially treated with intravenous ceftriaxone 2g twice a day and meropenem 1g twice a day before commencing continuation therapy with oral minocycline 100mg twice a day. Despite resolution of the acute cavitating complications of Nocardia infection with antibiotic therapy and controlled reduction of immunosuppression, graft function continued to deteriorate. Serum BK viral load was found to be very high at 6.40×10^7 copies/mL, prompting a graft biopsy which showed BK virus nephropathy with severe inflammation. The patient later recalled he had received pulsed methylprednisolone prior to travel for presumptive rejection.

Conclusions To the best of our knowledge, this is the first time concomitant BK virus nephropathy and *Nocardia cyriacigeorgica* bacteraemia have been described in the literature. The presence of bloodstream infection amenable to culture significantly aided in the diagnosis of nocardiosis and therefore in the prompt initiation of treatment. In our patient's case, intravenous meropenem and ceftriaxone then oral minocycline were effective in the treatment of disseminated *Nocardia cyriacigeorgica* with lung and cerebral abscesses. The rarity of bloodstream nocardiosis alongside florid BK virus nephropathy demonstrated clear overimmunosuppression and the importance of a 'for cause' graft biopsy before immunosuppression escalation. Whilst rare, the significant associated mortality makes

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nocardiosis an important differential to consider in transplant recipients with unexplained fever. Furthermore, as access to transplantation increases globally, access to quality post-transplant care must too.

Clinical trial number Not applicable.

Keywords *Nocardia*, Immunosuppression, Transplant, Bacteraemia, BK virus nephropathy

Background

Nocardia are ubiquitous Gram-positive, acid-fast bacteria found worldwide in soil and both fresh- and salt-water. The main route of infection is inhalation [1] but primary cutaneous infection can also occur following traumatic inoculation [2]. These environmental organisms were first described by French veterinarian Edmond Nocard [3] and >160 species have been identified so far [4] of which >40 can cause disease in humans [4], such as *Nocardia cyriacigeorgica* but more commonly *N. nova* and *N. farcinica* [1]. The taxonomy for *Nocardia* is complicated with several key revisions having been made; relevant here, *N. asteroides*, later *N. asteroides* complex, has been separated into several distinct species [5] including *N. cyriacigeorgica*.

People who are immunocompromised, including solid organ transplant (SOT) recipients -particularly lung and heart transplant recipients [1] - and individuals with HIV [6], are more likely to develop nocardiosis. Steroid use is a strong risk factor [7, 8]. Whilst the respiratory tract is most commonly involved, extrapulmonary dissemination occurs in >20% of cases [1] with the brain being the second most commonly affected organ [9] such that it is advised that brain imaging be performed on all patients recognised to have pulmonary disease [1]. Bacteraemia, however, is rare, occurring in only < 8% of cases [10]. It would appear that different species preferentially affect different systems, e.g., *N. brasiliensis* accounts for 80% of primary cutaneous disease [4] and *N. farcinica* may have a greater predilection for CNS spread [1]. Mortality rates are high, some studies reporting as high as 55% with cerebral infection [1] and standing at about 20% at 12-months for SOT recipients with disseminated disease [11], defined as involvement of two or more non-contiguous sites. As well as predisposing to infection in the first instance, organ transplantation and the intensification of its associated immunosuppression increases the risk of dissemination [12].

Though rare, the presence of bloodstream infection amenable to culture significantly aids in diagnosis and therefore in the prompt initiation of treatment. CNS involvement may resemble other conditions including CNS malignancy or toxoplasmosis, and similarly differentials for respiratory disease in SOT recipients include fungal pneumonias like *Pneumocystis jirovecii* pneumonia (PJP) although PJP tends to be more diffuse in radiological appearance. *Nocardia* spp. are especially

slow-growing and samples, particularly those from patients at risk of nocardiosis, should be incubated for up to 2 weeks [1, 13]. Traditional phenotypic methods for identifying *Nocardia* species are inadequate: sequencing of 16 S rRNA is the gold standard for speciating *Nocardia* isolates [4]. In the UK, the national reference laboratory processing *Nocardia* is AMHRAI (Antimicrobial Resistance and Healthcare Associated Infections), located in Colindale, with the unit's role in nocardiosis cases being antimicrobial susceptibility testing rather than speciation [14].

The mainstay of treatment for the disease is with antibiotic therapy, typically with trimethoprim-sulfamethoxazole (TMP-SMX) although cases may also require surgical intervention. Additionally, there is emerging evidence for the use of interferon γ in refractory disease [7]. For nocardiosis in SOT patients, the Infectious Diseases Community of Practice (IDCOP) of the American Society of Transplantation have provided guidelines [1] for empiric antibiotic treatment for nocardiosis based on the severity and distribution of disease with the suggested first-line empiric therapy for disseminated nocardiosis being TMP-SMX in combination with imipenem and amikacin.

When considering targeted therapy, European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints are part of a system for categorising microorganisms as susceptible (S and I) or resistant (R) to antimicrobial agents. However, some species/species groups including *Nocardia* lack numerical breakpoints to allow categorical interpretation to S, I or R. In such cases, EUCAST advice is to determine a minimum inhibitory concentration (MIC) and compare it with provided numerical values to assess the microbiological activity of the agent against the species. Agents for which the MIC is the same or lower can be considered for therapy [15]. In contrast, Clinical and Laboratory Standards Institute (CLSI) guidance, which is more widely used in the United States, does provide breakpoints for *Nocardia* for interpretation of MICs obtained from broth microdilution specifically [16].

The BK virus was first discovered in 1971 in a renal transplant recipient, patient BK, after whom the virus was named [17]. Clinically significant opportunistic infection can occur in kidney transplant recipients, leading to viraemia and nephropathy which in turn can cause allograft loss. There is a 3-year allograft survival of 79%

following a diagnosis compared to 90% in patients without virus associated nephropathy [18]. The intensity of immunosuppression is considered to be the most significant risk factor associated with BK viral replication [19]. A probable diagnosis of BK viral (BKV) nephropathy is considered in the setting of a serum BK viral load $\geq 10,000$ copies/mL [19] but kidney allograft biopsy is the gold standard for diagnosis. Histological findings include tubulitis visible on haematoxylin and eosin (H&E) staining and intranuclear inclusions in tubular epithelial cells visible on both H&E and immunohistochemistry (IHC) staining [19]. The reduction of immunosuppression is the mainstay of therapy for BK viraemia, starting with the withdrawal or reduction of antiproliferative agents such as mycophenolate [20].

Recognising the broadening range of geographical and resource settings in which solid organ transplantation is now being conducted, we report a case of *Nocardia cyriacigeorgica* bacteraemia with lung abscesses as well as concomitant BK virus nephropathy in a patient visiting from Nigeria within 12 months of a kidney transplant. To the best of our knowledge, this is the first time such co-infection with associated organ pathology has been reported in the literature.

Case presentation

Our patient, a 67-year-old man, presented initially to the Acute General Medicine take in our hospital feeling generally unwell after travelling from Lagos, Nigeria, where he was resident, to visit family in the Oxford area. Having been in the UK for a week, he began experiencing fevers and confusion, and also reported right-sided pleuritic chest pain. These symptoms had been going on for two days at the time of presentation. He had been self-medicating with oral ciprofloxacin 500mg twice a day as he was concerned about a probable infection.

The patient had a background of autosomal dominant polycystic kidney disease with end-stage renal failure for which he had received a right-sided kidney transplant in Lagos 12 months prior to this admission. This was from a live unrelated donor and unfortunately no further transplant history, including HLA and CMV status, was available from his transplant centre. His other comorbidities were hypertension, atrial fibrillation, and gastro-oesophageal reflux disease. He was a practising Jehovah's witness who confirmed he would not be able to accept blood products. His regular pre-hospital medications included prednisolone 5mg once a day, mycophenolate sodium 360mg twice a day, and tacrolimus (Prograf®) 0.5mg twice a day. He was not taking prophylactic anti-infective medications and it was unclear whether he had taken these in the immediate post-operative period. He had no drug allergies.

On examination, he had bibasal crepitations and no focal neurology. His temperature was measured to be 39.5 °C on admission. Otherwise, he was haemodynamically stable and did not require supplementary oxygen throughout his admission. His admission blood tests showed a C-reactive protein level of 142mg/L (normal range <5), serum creatinine of 330 μ mol/L (normal range 64–104; with no previous results from this patient for comparison of kidney function) and haemoglobin 72 g/L (male normal range 130–170). He was confirmed to be HIV Ab/Ag negative and a bloodborne virus screen was negative for hepatitis B (surface antigen) and C (antibody). A chest X-ray showed right upper zone consolidation, and he was discharged initially on treatment for a community-acquired pneumonia with oral amoxicillin 500mg three times a day and doxycycline 100mg twice a day for a 5 day course. Neither sputum nor whole blood were sent at that time for tuberculosis (acid fast bacteria staining and mycobacterial culture) or malaria (rapid diagnostic test and blood film) testing.

Blood cultures taken prior to discharge flagged 1 week later for *N. cyriacigeorgica*. He was recalled to the Ambulatory Assessment Unit where he had cross-sectional CT imaging of the lungs and brain, given that this is a known cavitating organism with a predilection for those organs, and was admitted to the Infectious Diseases ward under joint care of the Infectious Diseases and Nephrology teams after this showed multiple lung and intracranial abscesses (Fig. 1). MRI scans of brain and spine were conducted to rule out further cavitating disease not detected on CT. A transthoracic echocardiogram was also performed to rule out infective endocarditis and his blood culture isolate sent to AMRHAI reference laboratory for further work-up. A summary of admission investigations is given in Tables 1 and 2. Further blood cultures were taken upon admission to the ward and returned negative.

IV TMP-SMX 480mg twice a day in combination with IV meropenem 1 g twice a day were initially used to treat his disseminated and cerebral disease. This decision considered the IDCOP guidelines [1] as well as the MICs from our laboratory whilst awaiting results of further susceptibility testing from the AMRHAI reference lab (Table 2) along with the need for good central nervous system penetration (Table 3). At the same time, his immunosuppression was reduced with mycophenolate being stopped outright by the nephrology team in light of this serious infection (Fig. 3).

However, his graft function declined (with a creatinine rise from 280 to 330 μ mol/L) following commencement of IV antibiotic therapy (Fig. 3). Associated with this decline in graft function was severe renal anaemia with haemoglobin <70 g/L. He declined anaemia treatment with red blood cell transfusions and instead received subcutaneous erythropoietin (Retacrit®) 10,000 units

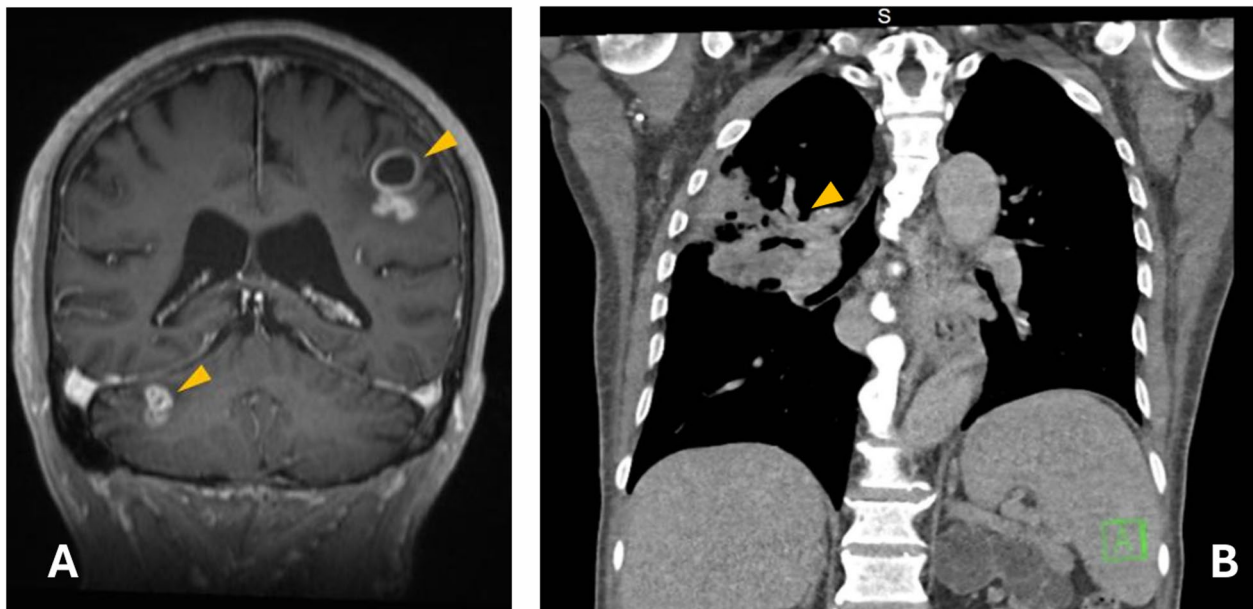


Fig. 1 Admission brain and chest cross-sectional imaging. **(A)** Initial brain MRI scan (T1 weighted post gadolinium) showing both supra- and infratentorial ring-enhancing lesions suggestive of abscesses (yellow arrows); **(B)** Initial chest CT with portal venous iodinated contrast showing abscesses and cavitation (yellow arrows)

Table 1 Summary of admission investigations

Investigation:	Result
HIV Ab/Ag	Negative
Hepatitis B surface antigen	Negative
Hepatitis C antibody	Negative
Chest radiograph	Multiple opacities throughout both lungs with some apparent cavitations
Chest CT with contrast (see Fig. 1)	Right-sided low attenuation lesions in keeping with pulmonary abscesses. No large cavities.
Brain CT with contrast	Multiple intracranial ring-enhancing lesions in the supra- and infratentorial compartments in keeping with abscesses.
Brain MRI with gadolinium contrast(see Fig. 1)	Multiple ring enhancing intracranial lesions in brain parenchyma, demonstrating central restricted diffusion and high T2/FLAIR signal abnormality, in keeping with abscesses.
Whole Spine MRI	No spinal abnormality demonstrated
Transthoracic Echocardiogram	No evidence of mass or vegetation. No significant valvular abnormalities noted. Preserved left ventricular systolic function with ejection fraction ~60%. Borderline dilated right atrium with preserved function

Table 2 Minimal inhibitory concentration listings from the Oxford University Hospitals (OUH) regional and AMRHAI (National) reference laboratories

Antibiotic	OUH ($\mu\text{g/mL}$)	AMRHAI (mg/L)	EUCAST Suitability for Therapy? (mg/L)	CLSI Breakpoints	
				Susceptible ($\mu\text{g/mL}$)	Intermediate ($\mu\text{g/mL}$)
Ceftriaxone	0.25	2	if ≤ 0.5	≤ 8	16–32
TMP-SMX	0.5	0.125	if ≤ 1	$\leq 2 / 38$	N/A
Imipenem	Not tested	0.5	if ≤ 2	≤ 4	8
Linezolid	1	4	if ≤ 2	≤ 8	N/A
Minocycline	Not tested	2	if ≤ 2	≤ 1	2–4
Meropenem	2	Not tested	if ≤ 2	N/A	N/A
Turnaround Time	7 days	14 days			

Table 3 Summary of factors affecting antibiotic choice

Case characteristic	Medical need/decision	Specific trade-off
Failing renal transplant	Immunosuppression Low eGFR Anaemia	Balance between graft preservation and infection risk Relative contraindication to TMP-SMX Relative contraindication to TMP-SMX, linezolid
Multiple cerebral abscesses	Limits antibiotic choice, extends duration of therapy	Antibiotics with good central nervous system penetration: meropenem, ceftriaxone, minocycline
Disseminated disease	Extends duration of therapy with multiple antibiotics	Combination therapy for extended period with potentially increased risk of adverse effects
Jehovah's Witness	No blood products	Relative contraindication to TMP-SMX, linezolid
Rare environmental pathogen	No EUCAST breakpoints No randomised control trials	Specialist laboratory turnaround time Limited evidence to support antibiotic selection

three times a week. The ultrasound scan of the transplant kidney showed no hydronephrosis and good perfusion. Given the decline in renal function without a clear anatomical cause and worsening anaemia, IV TMP-SMX was stopped and IV ceftriaxone 2 g twice a day commenced in its place (Table 3).

Following recovery of graft function and anaemia as well as his continued clinical stability (Fig. 3), he was discharged to his daughters' home after 23 days as an inpatient to continue IV ceftriaxone 2 g and IV meropenem 1 g for a total of eight weeks administered by his daughters, who are nurses, with training, support, and ongoing monitoring from the outpatient antibiotic therapy (OPAT) team. The patient and his family opted to not have formal nephrology outpatient care because of their concerns about cost.

However, his graft function was noted to have declined again by the OPAT team six weeks into IV antibiotic therapy and three weeks in the community (Fig. 3). Following discussion with the nephrology team, a BK viral titre was taken. His viral load returned as very high at 6.40×10^7 copies/mL and he therefore went on to have a renal transplant biopsy which confirmed florid BK virus nephropathy with viral inclusion bodies (Fig. 2). There was no evidence of transplant rejection. As such, his tacrolimus dose was reduced (Fig. 3) with the understanding that this would increase his risk of transplant rejection.

The patient and his family at this point recalled further immunosuppression had been previously administered on top of his usual maintenance. About two months prior to visiting the UK on this occasion, he had received a single pulse of IV methylprednisolone of unknown dose in Nigeria for a presumptive diagnosis of acute T-cell mediated rejection following declining graft function and findings on an ultrasound scan without a renal biopsy at that time to confirm this. Retrospectively, it is likely that declining graft function was driven by BK virus nephropathy which, if identified by for-cause biopsy, would have offered an opportunity to reduce immunosuppression rather than escalate it.

After a total of 51 days on effective IV antibiotic therapy (51 days with meropenem 1 g twice daily and 48 days with ceftriaxone 2 g twice daily; ~8 weeks from initial presentation), a repeat MRI head scan showed an improved appearance of the abscesses, with resolution of internal restricted diffusion within the left posterior frontal/parietal, left occipital and right cerebellar abscesses (Fig. 4). Given his clinical improvement and his imminent return to Lagos, the patient was changed after 58 days of treatment from IV meropenem 1 g and IV ceftriaxone 2 g both twice a day to oral minocycline 100mg twice a day to be continued for a total antibiotic course of 12 months. We have also recommended that the patient continue on secondary prophylaxis with oral TMP-SMX lifelong if tolerated after completion of minocycline. At the point of returning to Nigeria, the patient was on prednisolone 5mg once a day and tacrolimus 1mg twice a day; mycophenolate was not restarted with the view that it be permanently stopped (Fig. 3).

Discussion & conclusions

The rise in kidney transplantation globally reflects the increasing prevalence of end stage renal failure due to an ageing and progressively more co-morbid population [21]. Given the growing number of kidney transplants being performed and the significant immunosuppression associated, opportunistic infections are increasingly important to monitor for, prevent, and treat as part of high-quality post-transplant multidisciplinary care. We report what we believe to be the first case of concomitant *N. cyriacigeorgica* bacteraemia and BK virus nephropathy in the literature. Nocardial bacteraemia is rare, occurring in only < 8% cases [10], but *N. cyriacigeorgica* bacteraemia is especially uncommon even within this cohort with the majority of bloodstream disease cases described in the literature in the past 10 years having been due to *N. farcinica* [10].

A retrospective case series review of post-renal transplant care of 35 patients in Nigeria from 2002 to 2018 highlighted that the majority (77%) of transplants were performed abroad (mostly in India or Pakistan) [22] and

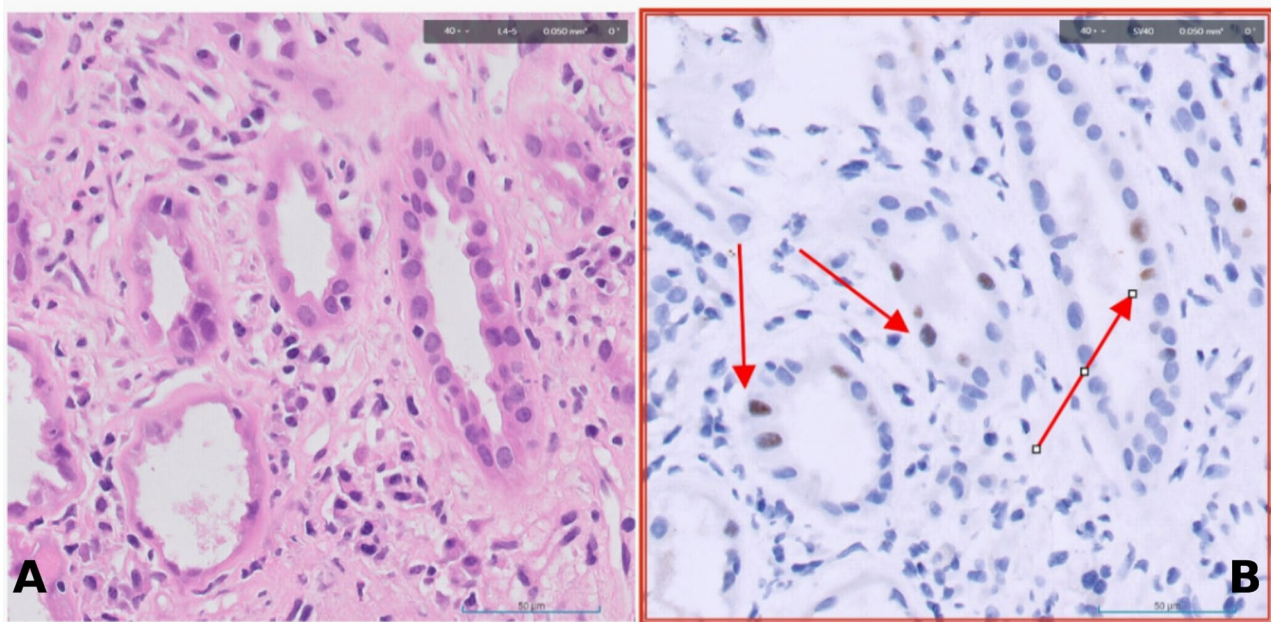


Fig. 2 Renal Transplant Histology following Further Deterioration of Kidney Function. **(A)** Haematoxylin and eosin staining; **(B)** Simian virus 40 polyomavirus immunohistochemistry with the red arrows indicating intranuclear viral inclusion bodies seen in BK virus nephropathy. Both at x400 magnification: the scale bars in the bottom right corner are both for 50 µm

that documentation was sparse, particularly regarding the donor. The most common post-transplant complication was bacterial infection in 57% of patients. Whilst high prices of typically imported drugs for aftercare were recognised as an obstacle, neither prophylaxis against nor education regarding opportunistic infections were explicitly discussed. In addition, as more than half (54.8%) of the recipients were lost to follow-up, it was not possible to establish rates of graft or patient survival. Similarly, whilst a recent retrospective review [23] of patients who underwent kidney transplantation at a single transplant centre in Nigeria recognised the need for long-term follow-up of patients, only early local post-operative outcomes were reviewed including delayed graft function and acute allograft rejection. Anti-infective prophylaxis was not discussed.

TMP-SMX therapy for nocardiosis has been reported since 1969 [24] and it has become the drug of choice for this disease following a 1983 retrospective cohort analysis [25] reviewing outcomes of patients who had received different antimicrobial regimens. To this day there have been no randomised control trials comparing treatments for nocardiosis. Three-drug regimens that include TMP-SMX are suggested by IDCOP for life-threatening nocardiosis while awaiting susceptibility testing [1], but again there is no evidence that such a combination provides improved outcomes. TMP-SMX is often used following SOT as prophylaxis against *Pneumocystis jirovecii* pneumonia and, given that it is first line for treatment, there also appears to be prophylactic benefit for nocardiosis.

A recent systematic review and individual patient data meta-analysis of 3 studies [8] ($n = 779$; 260 SOT recipients with nocardiosis vs. 519 uninfected controls) noted that TMP-SMX prophylaxis was independently associated with a significantly decreased risk of nocardiosis but prophylaxis had unclear benefit for preventing disseminated disease. This patient was not taking any post-transplant anti-infective prophylaxis prior to admission.

Ceftriaxone was a suitable empirical choice for *N. cyriacigeorgica* as this species is typically susceptible [1, 9]. That said, we recognise the variation between the local and reference laboratory MIC listings for ceftriaxone which were both based on gradient strip testing. We feel that combination therapy for this patient's severe disease as recommended by IDCOP [1] was additionally justified in view of the difficulty of accessing broth microdilution [16] within a clinically relevant time frame for definitive susceptibility testing to resolve the differences in results from the local and reference laboratories. At the same time, the isolate would have been considered susceptible to ceftriaxone based on MICs from both laboratories if CLSI breakpoints were used instead of EUCAST cut-offs (Table 2) [26].

The carbapenem imipenem is recommended by IDCOP [1] as part of empiric therapy for disseminated nocardiosis. However, in the OUH formulary, imipenem is available only as combination therapy for resistant Gram-negative infections (imipenem-cilastatin and imipenem-cilastatin-relebactam), not as a standalone antibiotic. There is both in vitro [27] and in vivo [28] evidence

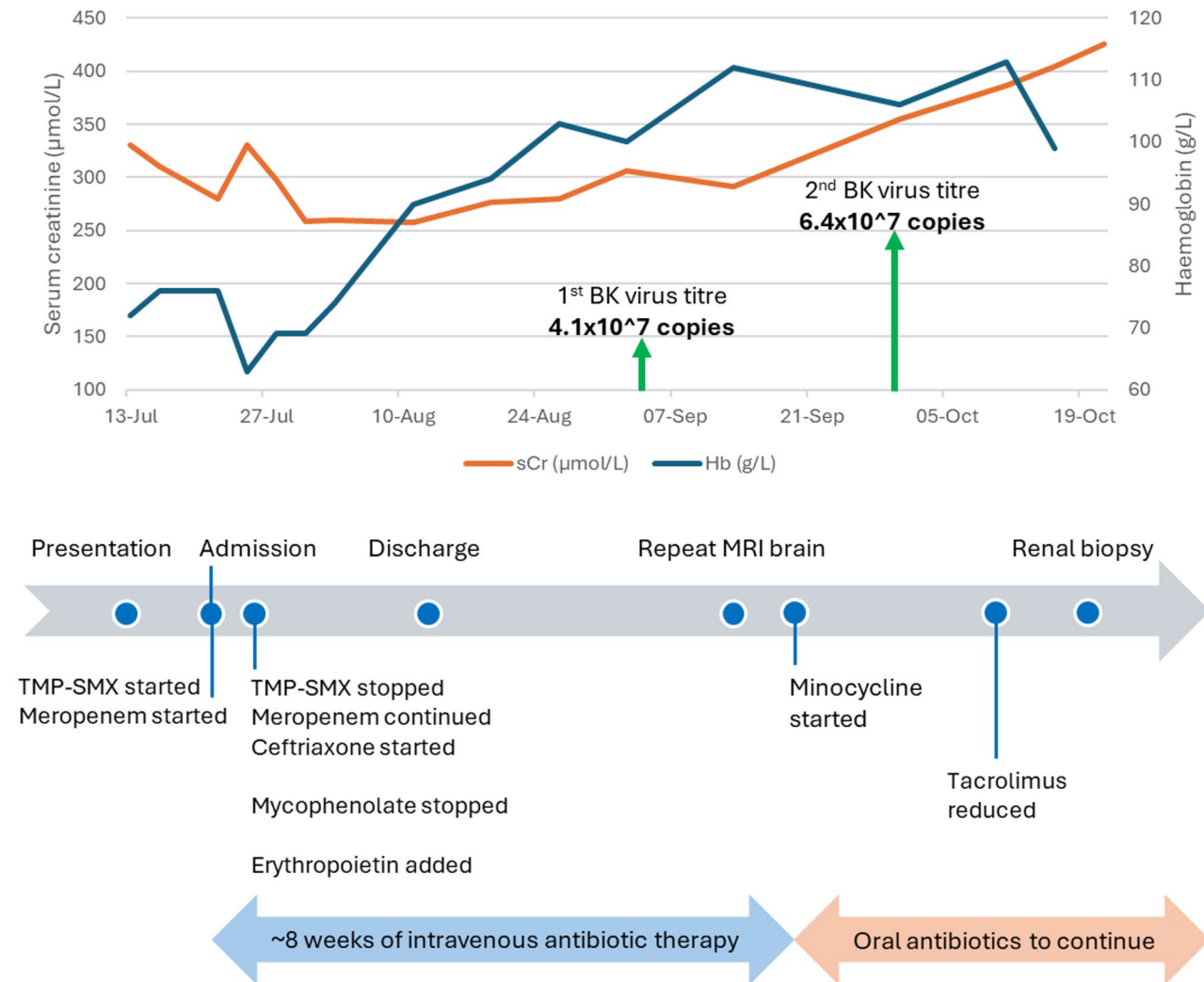


Fig. 3 Graft function (creatinine, $\mu\text{mol/L}$) and anaemia (haemoglobin, g/L) over time. The patient was discharged from the ward with outpatient follow-up on tacrolimus 0.5mg twice a day and prednisolone 5mg once a day. Mycophenolate was stopped and never restarted. Given low trough levels after discharge, the patient's tacrolimus dosing was initially increased to 1.5mg in the morning and 1mg in the evening. However, after the second BK viral titre returned as up-trending, the tacrolimus dose was reduced to 1mg twice a day

that *N. cyriacigeorgica* is typically susceptible to meropenem. For this case, susceptibility to meropenem was further supported by MIC results on the patient's isolate from our hospital laboratory (Table 2).

Minocycline, which our patient was eventually switched to from meropenem and ceftriaxone, has good CNS penetrance even when taken orally with a key historical case report from 1979 [29] showcasing its efficacy in treatment of intracranial *N. asteroides*. There is no evidence for the role of minocycline in prophylaxis for nocardiosis.

The resolution of signs and symptoms, such as fevers and confusion, and improvement on serial head imaging supported the efficacy of treatment. However, given the concomitant BK virus nephropathy and issues with balancing immunosuppression against disseminated nocardiosis infection, it is unclear for how long his transplant

kidney will continue to function. Moreover, immune reconstitution associated nephritis from the anti-BKV response itself may cause further decline in kidney function (though this in the hope of salvaging some longer-term function).

The reduction of immunosuppression is the mainstay of therapy for BK viraemia, starting with the withdrawal or reduction of antiproliferative agents (in this case, mycophenolate) [20]. IVIg at 2 g/kg is sometimes considered at select centres alongside controlled reduction of immunosuppression [30]. However, this is not part of routine treatment at our centre in accordance with the British Transplantation Society guidelines [31] due to a lack of randomised control trial evidence supporting its use in this context.

The presumed diagnosis of rejection without histology led to methylprednisolone pulsing, which often occurs in

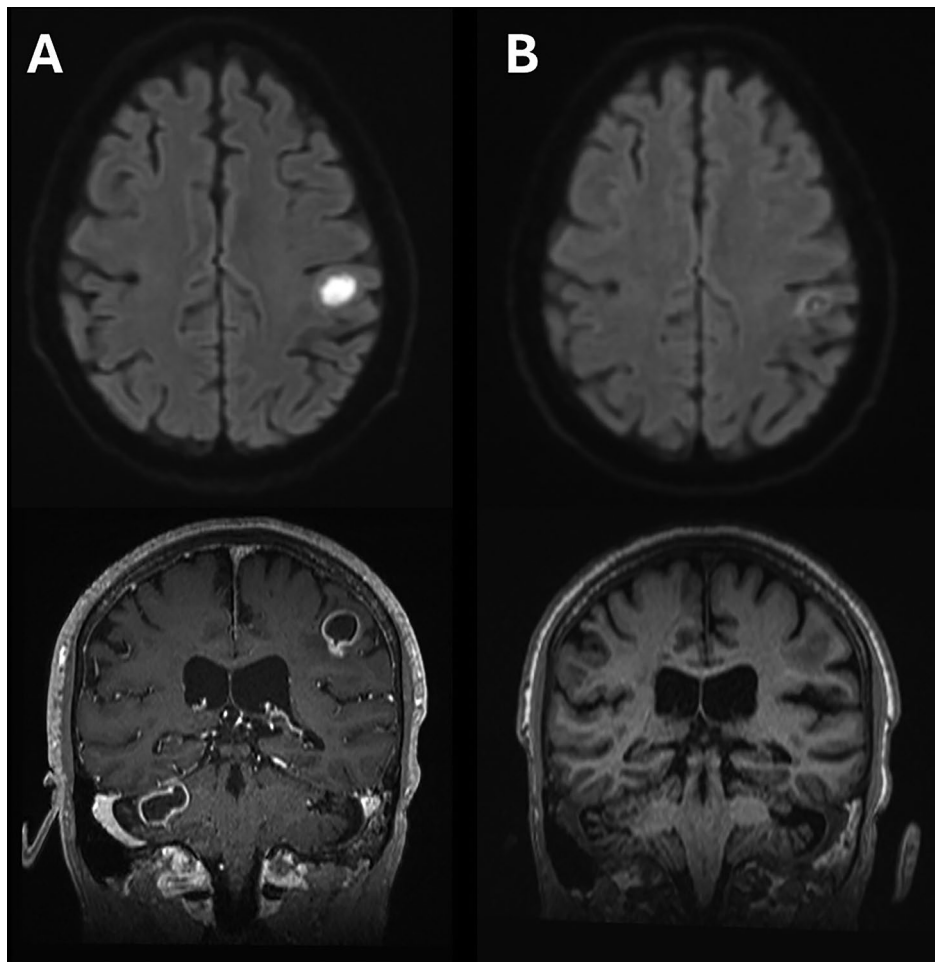


Fig. 4 Pre-treatment and follow-up MRI brain imaging. Pre-treatment (A) and follow-up (B) MRI brain imaging, 6 weeks apart. Contrast was not used in the follow-up imaging due to concerns about graft function. The axial images, top, are diffusion-weighted and show reduction of the diffusion restriction within the post-central gyrus abscess. The coronal images, bottom, are T1 weighted and show collapse of the right cerebellar and post-central gyrus abscesses

combination with an increase in maintenance immunosuppression. It is likely that this predisposed the patient to a more florid presentation of both diseases, noting his very high BK viral load and the rarity of *Nocardia* bacteraemia. Other risk factors for nocardiosis include HIV [6] and pulmonary alveolar proteinosis [9] (PAP) which is associated with granulocyte-macrophage colony-stimulating factor autoantibodies [9]. Our patient was HIV negative. We did not test for anti-GM-CSF Abs as we felt his presentation was already adequately explained by immunosuppression and he did not have other features of underlying PAP such as ‘crazy paving’ on CT thorax [32].

In summary, whilst nocardiosis is uncommon and bloodstream involvement especially rare, the high associated mortality makes it an important disease presentation to consider in SOT recipients. In particular, consideration of prophylaxis and regular monitoring for opportunistic infections including nocardiosis, as

well as the use of appropriate microbiological tests for *Nocardia* in SOT recipients with unexplained fever is recommended. Our patient’s experience underscores the importance of access to high-quality multi-disciplinary post-transplant care globally which should include monitoring for, preventing, and treating opportunistic infections alongside management of immunosuppression. Furthermore, the co-presentation of nocardiosis bacteraemia with BK virus nephropathy demonstrates clear over-immunosuppression and the importance of a for-cause graft biopsy before immunosuppression escalation. The prolonged duration of antibiotic therapy means SOT recipients are more likely to experience side effects and this subset of kidney transplant patients are at particular risk on account of their vulnerable filtration rate; as evidenced here, ceftriaxone, meropenem, and minocycline can be used to safely treat disseminated nocardiosis with cerebral disease in patients with renal transplants.

Patient perspective

The patient was keen for this case report to be published so that healthcare professionals globally could learn more about the risk factors and management of the rare form of nocardiosis that he presented with, and to raise awareness of this disease. Unfortunately, since returning to Nigeria, the patient's graft function continued to deteriorate such that he now requires haemodialysis.

Abbreviations

AMRHAI	Antimicrobial Resistance and Healthcare Associated Infections
BKV	BK virus
CLSI	Clinical Laboratory and Standards Institute
EUCAST	European Committee on Antimicrobial Susceptibility Testing
GM	CSF-granulocyte-macrophage colony-stimulating factor
Hb	Haemoglobin
H&E	Haematoxylin and eosin
IDCOP	Infectious Diseases Community of Practice
IHC	Immunohistochemistry
IVlg	Intravenous immunoglobulin
MIC	Minimum inhibitory concentration
OUH	Oxford University Hospitals
PAP	Pulmonary alveolar proteinosis
PJP	Pneumocystis jirovecii pneumonia
sCr	Serum creatinine
SOT	Solid organ transplant
TMP	SMX-trimethoprim-sulfamethoxazole

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Author contributions

ThW wrote the first draft of the manuscript. MB, TW, and XHSC were the lead nephrology and infectious diseases consultants. All authors read and approved the final manuscript.

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Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Written informed consent was obtained from the patient himself.

Consent for publication

Written consent was obtained from our patient to be included in a case report.

Competing interests

The authors declare no competing interests.

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