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Nocardiosis and elevated beta-D-glucan in solid organ transplant recipients

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ABSTRACT

Beta-D-glucan (BDG) testing can expedite the diagnosis of invasive fungal infections in immunocompromised hosts. Elevated BDG levels have been reported in both *in-vitro* studies assessing cross-reactivity with *Nocardia* spp. and published cases of patients with nocardiosis, but there is little data on this association in solid organ transplantation (SOT) recipients. To explore this association, we conducted a case series of SOT recipients with culture-proven nocardiosis and BDG testing who received their care at our institution between 2016 and 2021. We found thirteen cases of nocardiosis in SOT recipients, of which three cases met our case definition of an elevated BDG. Their clinical courses are detailed in the present report. We found that BDG may be elevated in SOT with nocardiosis with no identified cause of false positive BDG, though a causal association cannot be determined. Future prospective studies that better evaluate the association between nocardiosis and BDG are warranted, as are studies that better characterize the possible variability in reactivity amongst *Nocardia* spp.

Introduction

Nocardia spp. are well-recognized pathogens that can lead to a range of infectious syndromes in immunocompetent [1] or immunocompromised [2] hosts, including solid organ transplant (SOT) recipients [3]. The diagnosis of nocardial infections rests on a combination of host, clinical, and radiographic factors along with the isolation of *Nocardia* spp. in culture from a suspected site of involvement [3]. A definitive diagnosis of nocardiosis often requires tissue sampling, a process that can prove challenging.

Invasive fungal infections (IFI) present an analogous dilemma. Antigen assays have resultantly been developed to assist in the diagnosis of several IFI including cryptococcosis [4] and endemic mycoses [5]. In particular, the beta-D-glucan (BDG) assay detects a component of the cell wall of most fungi [6] and can be useful in a variety of clinical contexts [7–9]. In a large meta-analysis, the sensitivity and specificity of BDG for IFI were 76.8% and 85.3%, respectively [9].

Since the clinical presentations of IFI and nocardiosis are often overlapping, patients diagnosed with *Nocardia* infection may undergo testing with BDG for work-up of IFI. Interestingly, elevated BDG levels (>60 pg/mL) have been reported in both *in-vitro* studies assessing cross-reactivity with *Nocardia* spp. [10] and published cases of patients with nocardiosis (Table 1) [10–12]. To our knowledge, BDG testing has not been previously described in the setting of nocardiosis in SOT recipients. To further explore this association, we conducted a case series of SOT recipients with culture-proven nocardiosis and BDG testing who received their care at our institution between 2016 and 2021.

Case reports

We retrospectively reviewed the electronic medical records of SOT recipients with positive *Nocardia* cultures treated at Yale New Haven Hospital between October 2016 and March 2021. Cases were identified by querying our institution's laboratory information system. For purposes of our study, we defined cases as (1) SOT recipients who had (2) a positive culture for *Nocardia* spp, (3) a clinical syndrome compatible with nocardiosis, and (4) underwent testing with the Fungitell® BDG assay (Associates of Cape Cod, MA, USA) as part of their workup. An elevated BDG was defined as > 60 pg/mL [13]. The Yale University Institutional Review Board approved this study (HIC#2000023859).

We identified thirteen cases of microbiologically-proven nocardiosis in SOT recipients (Fig. 1). Ten of thirteen cases underwent

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Table 1
Summary of reported cases with elevated beta-D-glucan in association with nocardiosis.

Age/sex	Relevant comorbidities	Immunosuppression	Antimicrobial prophylaxis	Diagnosis	Species	Diagnostic methods	Negative fungal diagnostics	Peak BDG level	Potential BDG confounders	Outcome	Reference
65/F	Autoimmune hemolytic anemia	Not reported	None	CNS	*Nocardia abscessus*	Histopathology, 16S ribosomal DNA sequencing	Serum cryptococcal antigen, serum galactomannan level, fungal CSF culture	>523 pg/mL (CSF); serum BDG negative	None	Not reported	[10]
86/F	Colorectal cancer, diabetes mellitus	6-month dexamethasone (4 mg/day) course	None	Pulmonary	*Nocardia nova*	Cytology, 16S rRNA sequencing	*Aspergillus* antigen, *Aspergillus* precipitating antibody, fungal sputum culture	≥300 pg/mL	None	Clinical improvement then death from underlying malignancy	[11]
73/M	Cryptogenic organizing pneumonia	10-month steroid and immunosuppressive regimen	None	Disseminated (pulmonary and CNS)	*Nocardia farcinica*	Histopathology, 16S rRNA sequencing	Serum cryptococcal antigen	94.7 pg/mL	Intravenous ampicillin-sulbactam use, serum galactomannan level elevated	Clinical improvement then death from aspiration pneumonia	[12]

Abbreviations: BDG, beta-D-glucan; CNS, central nervous system; CSF, cerebrospinal fluid; DNA, deoxyribonucleic acid; rRNA, ribosomal ribonucleic acid.

BDG testing during their clinical courses, and BDG results were available for nine cases. Three of nine cases had elevated BDG (>60 pg/mL) (Table 2), and their clinical courses are detailed below.

Case 1. – *Nocardia nova/africana*

A 51-year-old man with a history of focal segmental glomerulosclerosis underwent kidney transplantation with alemtuzumab induction. His post-transplant course was complicated by acute cellular rejection (three weeks post-transplantation) treated with anti-thymocyte globulin and methylprednisolone. His immunosuppressive regimen consisted of tacrolimus, mycophenolate mofetil (MMF), and prednisone (5 mg/day). Post-transplant prophylaxis included atovaquone and acyclovir.

Two months post-transplantation, he presented with acute-onset pleuritic chest pain, productive cough, and fever at an outside hospital. A chest radiograph demonstrated right middle lobe consolidation consistent with pneumonia. He was given ceftriaxone and azithromycin. Due to fever (38.9 °C) on hospital day (HD) 2, antimicrobial therapy was broadened to cefepime, doxycycline, micafungin, and vancomycin. On HD3, serum BDG drawn on day of admission resulted as >500 pg/mL (reference range <60 pg/mL). On HD4, a computed tomography (CT) scan of the chest revealed a large cavitary pulmonary lesion, several pulmonary nodules, and bilateral ground-glass opacities. On HD7, bronchoscopy was performed, and bronchoalveolar lavage (BAL) cultures grew *Nocardia nova/africana* prompting a switch in antimicrobial therapy to trimethoprim-sulfamethoxazole (TMP-SMX) and meropenem. Subsequently, magnetic resonance imaging (MRI) of the brain was obtained and ruled out central nervous system involvement. BAL studies including fungal culture, bacterial and mycobacterial culture, *Pneumocystis jirovecii* polymerase chain reaction (PCR), and galactomannan testing were negative. In addition, serum cryptococcal antigen and urine *Histoplasma* antigen were negative. After review of the cumulative data, the elevated BDG was attributed to nocardiosis.

Approximately one-month post-hospital discharge, his regimen was switched to TMP-SMX and ceftriaxone when susceptibility results were made available. He completed 10-weeks of combination therapy before transitioning to TMP-SMX monotherapy. One month after transitioning, his symptoms improved, and imaging with a CT scan of the chest demonstrated a stable cavitary lesion in the left upper lobe with improvement in the left lower lobe nodule.

Case 2. - *Nocardia abscessus*

A 62-year-old woman with a history of pancreas and kidney transplant due to type 1 diabetes mellitus (eleven years prior) presented with lower extremity edema, fatigue, and dysphagia. At time of presentation, she was on MMF, prednisone (5 mg/day), and tacrolimus. Her post-transplant course had been complicated by renal graft failure requiring hemodialysis.

A CT scan of the chest was ordered to work-up the dysphagia and revealed mediastinal lymphadenopathy. Due to concern for a bacterial infection, piperacillin-tazobactam was empirically started. Blood cultures, sputum culture, *Coccidioides* serology, serum galactomannan, *Histoplasma* urine antigen, and BDG were negative. On HD3 she developed worsening respiratory status and left thigh pain. Creatine kinase was found to be elevated (4552 U/L). An MRI of the left femur revealed left thigh myositis, and a left thigh muscle biopsy revealed necrotizing fibers and non-specific acute inflammation with negative stains and cultures for microorganisms. Due to progressive respiratory decline, bronchoscopy was performed, and BAL cultures grew *Nocardia abscessus*. Additionally, intravenous immunoglobulin (IVIG) was administered due to concern for a paraneoplastic or progressive neurological process.

One-week post-IVIG, serum BDG measured >500 pg/mL. Antimicrobial therapy was empirically switched to TMP-SMX and meropenem. Further imaging revealed a right upper lobe pulmonary

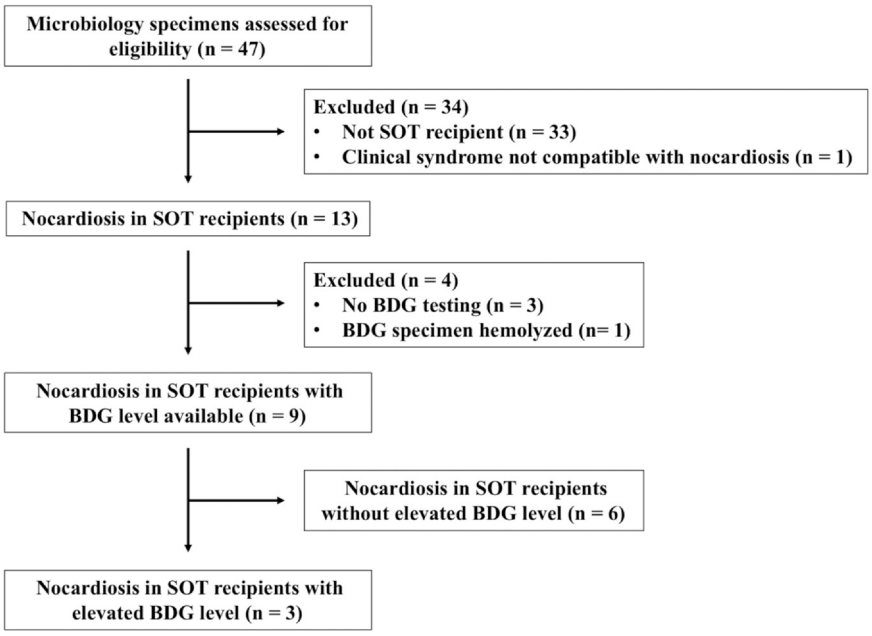

Fig. 1. Selection of nocardiosis cases in solid organ transplant recipients with beta-D-glucan testing. Abbreviations: BDG, beta-D-glucan; SOT, solid organ transplant.

mass likely due to nocardiosis. Five weeks after the last dose of IVIG, serum BDG remained elevated at 331 pg/mL. Two months into her hospital stay, she died from cardiogenic shock after undergoing mitral valve surgical repair.

Case 3. – *Nocardia anaemiae/Nocardia pseudovaccinii*

A 64-year-old man underwent liver transplantation for alcoholic cirrhosis, without the need for induction immunosuppression, and was on maintenance MMF, prednisone (5 mg), and tacrolimus. Notably, his post-transplant course was complicated by acute cellular rejection 1 month after transplant which was treated with methylprednisolone. Two years post-transplantation, he presented with failure to thrive, diarrhea, and a perineal rash involving the penis and gluteal folds.

The rash was due to *Herpes simplex* virus 2 (HSV-2) as determined by HSV-2 PCR of a skin swab from the affected area. He underwent sigmoidoscopy as part of his work-up for diarrhea. Cytomegalovirus (CMV) immunostaining of biopsied rectal tissue was positive and confirmed the diagnosis of CMV proctitis. Valganciclovir therapy was initiated. On HD8, he developed subcutaneous abscesses in the left calf and left elbow. Wound cultures obtained by incision and drainage of multiple abscesses grew *Nocardia* spp. identified as either *Nocardia anaemiae* or *Nocardia pseudovaccinii* by 16S rRNA sequencing. An MRI of the brain was obtained due to concern for disseminated nocardiosis and revealed multiple non-specific hypodensities concerning for abscesses. A transthoracic echocardiogram was negative for vegetations. He was placed on a treatment dose of TMP-SMX and meropenem. He later developed worsening liver function which peaked on HD25 with elevated aspartate aminotransferase 907 U/L (reference range 11–33 U/L), alanine aminotransferase 996 U/L (reference range 6–34 U/L), and alkaline phosphatase 1766 U/L (reference range 9–122 U/L). Liver biopsy was performed and revealed cholestatic hepatitis and bile duct injury consistent with drug-induced livery injury. TMP-SMX was considered a possible cause and switched to linezolid. He developed cytopenias on HD37 attributed to linezolid, so it was substituted with moxifloxacin and minocycline.

On HD51, he had a witnessed tonic-clonic seizure, and a repeat MRI revealed parenchymal enhancement suggestive of worsening nocardiosis or a new infectious process. A lumbar puncture was performed, and cerebrospinal fluid (CSF) studies revealed 0 nucleated cells/uL (reference range <6 cells/uL), glucose 81 mg/dL (reference range 40–70 mg/dL), and protein 69.2 mg/dL (reference range 15–45 mg/dL). CSF bacterial cultures were negative. A serum BDG was obtained to assess for a fungal infection due to unclear etiology of the MRI findings and returned elevated at >500 pg/mL. The patient was empirically started on anidulafungin; however, blood cultures, galactomannan, and CSF fungal and mycobacterial cultures were negative. On HD70, anidulafungin was discontinued, and the elevated BDG was attributed to nocardiosis. He was discharged with a plan to complete 6 months of therapy for nocardiosis. Unfortunately, he died 3 months later due to toxic metabolic encephalopathy complicated by aspiration pneumonia and acute renal failure.

Discussion

The utility of BDG as a diagnostic tool for IFI was first explored among patients with hematological malignancies [8,9], and the literature surrounding interpretation and applicability of BDG assays has since expanded to other populations, including SOT recipients [14]. In SOT recipients with a compatible clinical syndrome, an elevated BDG can be highly suggestive of an IFI; however, elevated BDG has also been reported in association with non-fungal organisms, particularly *Nocardia* species both in *in-vitro* studies and in case reports [10–12]. Notably, *Nocardia* can produce disease that is clinically indistinguishable from a fungal infection in an immunocompromised population. Despite this clinical and microbiological overlap, the diagnostic utility of the BDG assay for the diagnosis of nocardiosis has not been explored in a clinical setting. Indeed, there are no prior published reports describing elevated BDG in SOT recipients infected with *Nocardia*, and the American Society of Transplantation guidelines on nocardial infections did not include the BDG assay [3]. This case series builds on prior data in the non-transplant population suggesting that nocardiosis may be associated with elevated BDG and expands this observation to the setting of SOT.

In addition to cross reactivity with certain bacteria, there are other established reasons for falsely elevated BDG levels. These include hemodialysis with cellulose membranes [15], IVIG [16],

Table 2
Yale New Haven Hospital nocardiosis cases in solid organ transplant recipients who underwent beta-D-glucan testing.

Age/sex	Relevant comorbidities	Immunosuppression	SOT induction therapy	Antimicrobial prophylaxis	Time since SOT	Diagnosis	Species	Diagnostic methods	Negative fungal diagnostics	Peak BDG level	Potential BDG confounders	Outcome
Cases with elevated BDG												
51/M	Kidney transplant	Tacrolimus, mycophenolate mofetil, prednisone (5 mg/daily)	Alemtuzumab	Atovaquone, acyclovir	2 months	Pulmonary	*Nocardia nova/ africana*	BAL culture	Fungal BAL culture, *Pneumocystis jirovecii* BAL PCR, galactomannan BAL testing, serum cryptococcal antigen, urine *histoplasma* antigen	>500 pg/mL	None	Clinical improvement
64/M	Liver transplant	Tacrolimus, mycophenolate mofetil, prednisone (5 mg/daily)	N/A	None	2 years	Disseminated (skin abscess and CNS)	*Nocardia anaemiae/N. pseudovaccinii*	Abscess I&D and nocardia culture	Fungal wound culture, fungal blood culture, fungal CSF culture, urine *histoplasma* antigen, serum cryptococcal antigen, serum *coccidioides* antigen, toxoplasmosis PCR	>500 pg/mL	None	Deceased due to shock and decompensated cirrhosis
62/F[a]	Kidney-pancreas transplant	Tacrolimus, mycophenolate mofetil, prednisone (5 mg/daily)	N/A	None	11 years	Disseminated (pulmonary and cardiac)	*Nocardia abscessus*	BAL culture	Fungal BAL culture, fungal blood culture, urine *histoplasma* antigen, serum cryptococcal antigen, serum *coccidioides* antigen	>500 pg/mL	IVIG (1 week prior to initial BDG; BDG 5 weeks after last IVIG dose was 331 pg/mL)	Deceased due to cardiogenic shock after mitral valve repair
Cases with normal BDG												
52/M[a]	Kidney transplant	Mycophenolate mofetil, prednisone (5 mg/daily), belatacept monthly, eculizumab biweekly	Thymoglobulin	Atovaquone, valganciclovir	10 weeks	Disseminated (CNS, pulmonary, and skin)	*Nocardia farcinica*	Blood and deep wound cultures	Serum cryptococcal antigen	46 pg/mL	None	Clinical improvement
57/F	Heart transplant	Tacrolimus, mycophenolate mofetil, prednisone (5 mg/daily)	Basiliximab	Atovaquone, acyclovir	2 months	Skin abscess	*Nocardia farcinica*	Abscess I&D and nocardia culture	N/A	<31 pg/mL	None	Clinical Improvement
53/F	Kidney transplant	Tacrolimus, mycophenolate mofetil, prednisone (3 mg/daily)	N/A	None	17 years	Disseminated (CNS, pulmonary and skin)	*Nocardia* spp.	Abscess I&D and nocardia culture	N/A	<31 pg/mL	None	Clinical Improvement
66/M	Kidney transplant	Tacrolimus, mycophenolate mofetil, prednisone (5 mg/daily)	Alemtuzumab	None	3 years	CNS	*Nocardia farcinica*	Brain abscess drainage with tissue culture	N/A	<31 pg/mL	None	Clinical stability at follow up (2 months after discharge)

(*continued on next page*)

Table 2 (*continued*)

Age/sex	Relevant comorbidities	Immunosuppression	SOT induction therapy	Antimicrobial prophylaxis	Time since SOT	Diagnosis	Species	Diagnostic methods	Negative fungal diagnostics	Peak BDG level	Potential BDG confounders	Outcome
50/M	Kidney transplant	Belatacept, mycophenolate mofetil, prednisone (5 mg/daily)	Alemtuzumab	Valganciclovir, atovaquone	8 months	Disseminated (CNS, pulmonary)	*Nocardia nova* complex	Brain abscess drainage with tissue culture	N/A	<31 pg/mL	None	Clinical and radiologic improvement
65/M	Kidney transplant	Tacrolimus, mycophenolate mofetil, prednisone (5 mg/daily)	Alemtuzumab	None	1 year	Pulmonary	*Nocardia nova* complex	BAL culture	N/A	<31 pg/mL	None	Clinical and radiologic improvement

Abbreviations: BAL, bronchoalveolar lavage; BDG, beta-D-glucan; CNS, central nervous system; CSF, cerebrospinal fluid; I&D, incision and drainage; IVIG, intravenous immunoglobulin; N/A, not applicable; PCR, polymerase chain reaction; SOT, solid organ transplantation.

[a] Although these patients were on hemodialysis at the time of admission, our institution uses non-cellulose membranes. As a result, this was not deemed to confound the BDG level.

various antimicrobial agents [17], and surgical gauze containing glucan [18]. Although two cases in our series were on hemodialysis at the time of admission, our institution uses non-cellulose dialysis membranes which are not known to interfere with BDG levels [19]. Therefore, hemodialysis was not deemed to be a confounder of BDG testing. Notably, Case 2 did receive IVIG one week prior to measurement of serum BDG, but the BDG level remained significantly elevated (331 pg/mL) for >5 weeks after IVIG. In an analysis of 21 pediatric patients receiving IVIG, BDG normalized in 64% and 100% of patients at one and three weeks, respectively [20]. Given the sustained positivity in our case, it is unclear whether or not IVIG played a significant role in confounding the results.

It is interesting to note that all three cases of *Nocardia farcinica* (maximum BDG 46 pg/mL; Table 2) at our institution did not satisfy the case definition of BDG elevation (>60 pg/mL). This is consistent with an *in-vitro* study performed by Sawai et al. [12] that evaluated *N. farcinica* isolated in a brain specimen and reported mild elevation of BDG levels to about 20 pg/mL compared to control (pure blood agar). Other published studies suggest that levels of BDG may vary across infection with different *Nocardia* spp. [10–12] Whether species heterogeneity is related to variations in cell-wall content or to other pathogen or host factors is unclear and deserves further investigation.

Our case series is limited by its modest size, retrospective design and descriptive format, which precludes any causal inference. Additionally, the small number of nocardiosis cases in SOT recipients with BDG results available limits our ability to characterize BDG variation across *Nocardia* spp. The intent of the report is not to establish a causative relationship between BDG elevation and nocardiosis. Rather, this report serves to highlight an association that is poorly known in the clinical setting of transplantation.

In light of the abovementioned results, SOT recipients with elevated BDG, no identified cause of false positive BDG, and clinical concern for IFI with a negative comprehensive workup (including microbiology, histopathology, serology and antigen testing) may merit a work-up for infection with *Nocardia* spp. This series has important implications for the diagnostic utility of BDG for the diagnosis of nocardiosis in SOT. Future prospective studies that better evaluate the association between nocardiosis and BDG, are warranted, as are studies that better characterize the possible variability in reactivity amongst *Nocardia* spp.

Ethical approval

The Yale University Institutional Review Board approved this study (HIC#2000023859).

Consent

Need for informed consent was waived by our institution's IRB.

CRediT authorship contribution statement

Matthew Ringer: Writing – original draft, Conceptualization, Methodology. **Christopher Radcliffe:** Writing – original draft, Conceptualization, Methodology. **Christopher A. Kerantzas:** Data curation, Writing – review & editing. **Maricar Malinis:** Conceptualization, Methodology, Supervision, Writing – review & editing. **Marwan M. Azar:** Conceptualization, Methodology, Supervision, Writing – review & editing.

Declarations of Competing Interest

None.

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