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Review Article

Disseminated Nocardiosis in Renal Transplant Recipients: A Review of Published Cases

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Abstract

Background: Nocardia species cause opportunistic infection in renal transplant recipients, in whom dissemination to the central nervous system may be present without any neurological complaint.

Objective: To examine whether every recipient with nocardiosis needs brain imaging and whether reported sulfonamide resistance reflects biology or susceptibility testing.

Methods: Narrative review of published case-control studies, registry analyses, single-centre series and case reports.

Findings: In a European cohort of 117 transplant recipients, 30 had central nervous system nocardiosis and 13 of those 30 (43.3%) had no neurological symptoms; cerebral involvement extended median therapy to 499 days against 296 days among survivors overall. Reference-method testing found trimethoprim-sulfamethoxazole resistance in 2% of 1299 isolates, against 42% among referred isolates.

Conclusions: The published record supports brain imaging at diagnosis in every recipient; it does not resolve whether prophylaxis prevents nocardiosis, nor does prophylaxis exclude the diagnosis.

Keywords: Nocardiosis; Kidney transplantation; Central nervous system infection; Trimethoprim-sulfamethoxazole; Antimicrobial susceptibility testing

Introduction

Nocardia species are aerobic, branching, partially acid-fast actinomycetes that behave as opportunists in patients with impaired cell-mediated immunity. Solid organ transplant recipients are among the groups most consistently affected, and kidney recipients, because they form the largest transplant population, contribute a large absolute number of cases even though their per-patient risk is lower than that of lung or heart recipients [1]. Few centres accumulate more than a handful of cases, so the evidence base is assembled from multicentre case-control studies, registry analyses, single-centre series and individual reports [2].

This review draws only on published records. No patient was cared for by the authors, and every clinical particular below comes from a named published source.

Two questions dominate practical management, and both are answerable from the published literature. The first is whether the brain should be imaged in every renal transplant recipient with nocardiosis, including those with no neurological complaint. The second is whether the widely quoted figures for sulfonamide resistance in Nocardia describe biology or describe the assay. We argue that the answer to the first question is yes, and that the high resistance figures are most plausibly an artefact of susceptibility testing. A third question, whether trimethoprim-sulfamethoxazole (TMP-SMX) prophylaxis prevents nocardiosis, is genuinely unresolved, and we set out both sides rather than adjudicate.

Incidence and Timing After Renal Transplantation

In a matched case-control study covering 5126 transplants performed at a single United States centre between 1995 and 2005, 35 recipients (0.6%) developed nocardiosis; by organ the proportions were 18/521 (3.5%) for lung, 10/392 (2.5%) for heart, 2/155 (1.3%) for intestine, 3/1717 (0.2%) for kidney and 2/1840 (0.1%) for liver [1]. A more recent single-centre cohort of 6179 transplants performed in

Miami between January 2012 and June 2022 identified 13 cases (0.2%), of which 7/3845 (0.18%) were kidney recipients [3]. At population scale, 657 of 203,233 United States renal transplant recipients recorded in the United States Renal Data System between 2001 and 2011 carried a diagnostic code for Nocardia infection (0.32%), pneumonia being the commonest presentation at 15.2% and brain abscess the second commonest at 8.4% [4]. An Australian single-centre series identified 4 of 543 renal transplant recipients (0.74%) with culture-proven disease between 2008 and 2014 [5]. These are cumulative proportions over variable follow-up rather than person-time incidence rates.

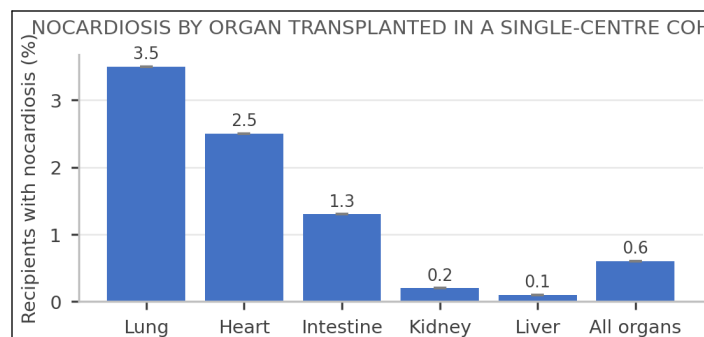


Figure 1: Proportion of recipients developing nocardiosis by organ transplanted among 5126 transplants performed at a single United States centre between 1995 and 2005, N = 5126 [1].

Timing is late and widely dispersed. Among 117 solid organ transplant recipients from 36 European centres, the median interval from transplantation to nocardiosis was 17.5 months, with a range of 2 to 244 months [6]. In 123 cases from three Mayo Clinic sites the median was 399 days, interquartile range 153.5 to 1471 days [7], and in the Miami cohort of 13 it was 8.8 months, range 3.7 to 98 months [3]. Renal-specific series give a median of 21 months, interquartile range 9.75 to 45 months, in 16 Indian recipients [8], and a median of 182 days, interquartile range 70.5 to 730 days, to pulmonary disease across 60 published kidney transplant cases [9]. The four Australian cases presented between 2 and 13 months after transplantation [5]. Nocardiosis is not confined to the early post-transplant window, and a late presentation is

no argument against the diagnosis.

Disease Extent and the Case for Imaging Every Brain

Localised pulmonary disease is the commonest presentation. Isolated pulmonary involvement accounted for 27/35 (77%) of cases in the Pittsburgh cohort, with dissemination in 7/35 (20%) [1]; for 60/82 (75.0%) of transplant recipients among 268 patients with invasive nocardiosis across three United States regions [10]; and for 16/26 (61.5%) of a renal allograft series from a single Indian centre [11]. Dissemination was documented at diagnosis in 33/123 (26.8%) of the Mayo cases, with localised pulmonary disease in 77/123 (62.6%) [7]; kidney recipients made up 57.6% of the disseminated cases, and all disseminated patients had pulmonary involvement [12]. Dissemination was more frequent in transplant recipients than in others, 18.3% against 9.7%, as was central nervous system involvement, 10/82 (12.2%) against 3.2% of 186 non-transplant patients [10].

The decisive observation comes from the European cohort. Of 117 solid organ transplant recipients, 30 had central nervous system nocardiosis, and 13 of those 30 (43.3%) had no neurological symptoms whatever [6]. Their cerebral disease was found only because imaging was performed. Nearly half of the central nervous system nocardiosis in that cohort would have been missed by a symptom-triggered imaging policy.

What follows from finding it is not, on present evidence, survival. In the Mayo cohort of 125 patients, disseminated infection was not independently associated with 12-month mortality [12]. What changes is the treatment plan. The 13 patients with central nervous system infection who completed therapy in that cohort were treated for a median of 499 days, interquartile range 364 to 528 days, against a median of 296 days, interquartile range 191.25 to 389.25 days, among survivors overall [12]. Occult cerebral disease also carries weight for the graft, since presentation with brain abscess was associated with graft failure in the registry analysis of 657 renal recipients, hazard ratio 1.85 [4]. Brain imaging at diagnosis in every renal transplant recipient with nocardiosis is therefore justified by its effect on duration of therapy, choice of regimen and prognostic counselling.

Species Identification and Laboratory Diagnosis

Nocardia grows as Gram-positive branching bacilli that are partially positive on modified acid-fast staining, but that stain applied directly to clinical samples is unreliable and should be used only to confirm the acid-fastness of organisms already seen on Gram stain [13]. 16S ribosomal RNA sequencing is the reference standard and returns a result in one to two days against weeks for phenotypic methods [14]. CLSI MM18-A requires at least 99.6% 16S similarity with more than 0.4% separation from the next species, and because interspecies similarity is high, multilocus sequence analysis using gyrB, secA1, hsp65 and rpoB is increasingly used, with MALDI-TOF mass spectrometry recommended as the first-line method and

Table 1: Published renal transplant cases and series of nocardiosis, with sites of disease, prophylaxis status at onset and outcome as reported [28] [9] [5] [8] [11].				
First author, year	Age, sex; months post-transplant	Species	Sites involved	On TMP-SMX; outcome
El Chediak 2022, patient 1	not stated, male; 36	not stated	Lung and brain	Stopped at 2 months; lesions improved
El Chediak 2022, patient 2	70s, female; about 120	not stated	Lung and brain	Stopped at 6 months; not stated
Dalil 2025, index case	not stated, male; 7	not stated	Lung only, brain CT normal	Yes, 80/400 mg daily; cured at 1 year
Shrestha 2016, 4 cases	not stated; 2 to 13	not stated	2 lung, 2 cerebral abscess	Yes, 160/800 mg twice weekly; all improved
Bhandari 2023, 16 cases	median 44, 14 male 2 female; 21	N. farcinica 4 of 6 typed	Lung 75%, brain 12.5%	None at onset; 5 of 16 died
Rajesh 2024, 26 cases	mean 45.4, male to female 5:1; not stated	14 of 26 not identified	Lung only 16 of 26	not stated; 1 death at 2 months

sequencing reserved for complexes and unusual isolates [15]. Measured against full-length 16S sequencing, MALDI-TOF with a supplemented library correctly assigned 146/150 (97.3%) of Japanese clinical isolates to species or complex level [16].

The cost of not having these tools is visible in the published series. Among 26 renal allograft recipients at one Indian centre, no species was identified in 14 [11]; at another Indian centre, species-level results were available only for the 6 patients diagnosed after MALDI-TOF was introduced [8]. Species distribution differs between transplant and non-transplant populations: among 82 transplant recipients, *N. farcinica* accounted for 17 (20.7%) and *N. cyriacigeorgica* for 16 (19.5%), and of the 16 patients with central nervous system disease in that series *N. farcinica* accounted for 6 (42.8%) [10]. In the earlier Pittsburgh cohort, *N. nova* was the commonest isolate at 17/35 [1].

Reported Sulfonamide Resistance as a Testing Artefact

The most frequently quoted resistance data come from 765 isolates referred to the United States Centers for Disease Control and Prevention between 1995 and 2004, of which 61% were resistant to sulfamethoxazole and 42% to TMP-SMX [17]. That figure has not been reproduced by reference-method testing. Broth microdilution performed under CLSI M24-A2 on 552 recent clinical isolates in six United States laboratories found approximately 2% sulfonamide resistance, and the authors attributed the discrepancy to the reading of the 80% growth-inhibition endpoint that CLSI specifies for sulfonamides but not for other agents [18]. A reference laboratory series of 1299 isolates with susceptibility results found TMP-SMX resistance in 2% ($n = 21$), with higher proportions only in *N. pseudobrasiliensis* at 31% ($n = 4$) and the *N. transvalensis* complex at 19% ($n = 16$) [19].

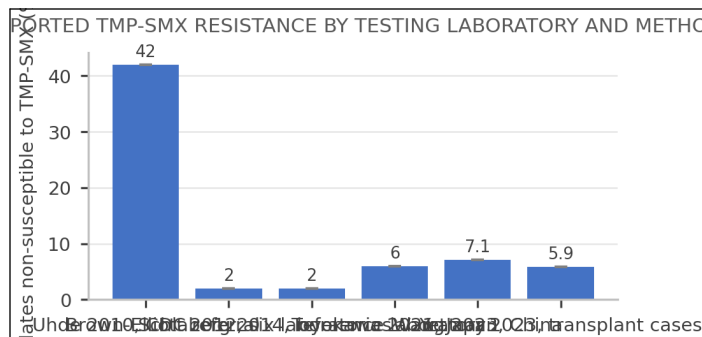


Figure 2: Reported non-susceptibility of *Nocardia* isolates to trimethoprim-sulfamethoxazole by testing laboratory and method, from 765 referred isolates [17], 552 isolates tested in six laboratories [18], 1299 reference laboratory isolates [19], 146 Japanese isolates [16], 198 Chinese isolates [21] and 119 solid organ transplant cases with testing available [7], $N = 3079$ isolates in total.

The control experiment is more damaging still. When six laboratories tested identical strains with identical panel lots and identical media, TMP-SMX readings for *N. wallacei*, a species expected to be susceptible, were reported as susceptible by 72.1% and resistant by 27.9%; agreement at the modal value plus or minus one dilution was 80.2%, and did not exceed 90% after removing any single laboratory [20]. Genotype points the same way: none of the phenotypically TMP-SMX-resistant isolates among 146 Japanese clinical isolates carried *sul1*, *sul2* or *dfrA* [16]. The Quebec series reporting 43% TMP-SMX resistance is not an independent replication, having been tested in the same laboratory as the referral collection [18].

Contemporary reference-method series converge: 94% of 146 Japanese isolates susceptible [16], 92.9% (184/198) of Chinese isolates tested by broth microdilution [21], 96.3% of isolates from 82 transplant recipients [10], and 7/119 (5.9%) resistant among solid organ transplant cases with testing available [7]. This is the weight of evidence rather than a proof, but reported sulfonamide resistance much above 5 to 10% should be treated as unconfirmed until reference-method testing supports it. The report of 4 of 6 *N. farcinica* isolates as TMP-SMX resistant in one Indian series [8] should be read with that caution, since all 26 *N. farcinica* isolates in the Mayo cohort were susceptible [7].

Prophylaxis, Breakthrough and the Limits of Prevention

Twenty-four of 35 solid organ transplant recipients with nocardiosis (69%) were receiving TMP-SMX for Pneumocystis prophylaxis when they became infected [1]. Nine of 13 in the Miami cohort (69%) were likewise taking prophylaxis at diagnosis, and all 7 isolates with susceptibility data were TMP-SMX susceptible [3]. All 4 Australian renal recipients were taking 160/800 mg twice weekly at onset, leading their authors to conclude that intermittent twice-weekly dosing appears insufficient to prevent Nocardia infection in renal transplant recipients [5]. The index case of a pooled analysis of published kidney transplant cases was taking 80/400 mg daily [9]. In the European multivariable model, low-dose cotrimoxazole prophylaxis was not found to prevent nocardiosis [6].

Set against that, current TMP-SMX prophylaxis was protective in a nested case-control study of 123 cases and 245 controls, adjusted odds ratio 0.34, 95% confidence interval 0.13 to 0.84 [7], and an individual patient data meta-analysis drawing on 5 comparative studies gave an adjusted odds ratio of 0.3, 95% confidence interval 0.18 to 0.52, with moderate certainty and no detectable dose effect [22]. Both estimates are open to confounding by indication. The same question has been examined in lung transplant recipients [23].

The two bodies of evidence are reconciled by the character of breakthrough disease. The pooled proportion of breakthrough isolates susceptible to TMP-SMX was 98%, 95% confidence interval 92 to 100 [22], and only 2 of 32 case patients taking prophylaxis had a resistant isolate in the Mayo series [7]. Prophylaxis at Pneumocystis doses reduces the risk of nocardiosis without abolishing it, and a patient taking TMP-SMX must not be considered to have nocardiosis excluded.

Treatment Duration and Outcome

Table 2: Disease extent and outcome across published cohorts of nocardiosis after transplantation [26] [12] [10] [3] [8] [9].			
Cohort	N	Disease extent	Key outcome
Lebeaux 2017, Europe	117	not stated	1-year mortality 16.2% (19/117) vs 1.3% (3/233) in controls
Yetmar 2023, Mayo	125	Disseminated 33 (26.4%)	12-month mortality 24.2% (8/33) vs 14.1% (13/92)
Gupta 2023, transplant	82	CNS 12.2% (10/82)	1-year survival 66/82 (80.5%)
Bini Viotti 2024, Miami	13	not stated	1-year mortality 46% (6/13)
Bhandari 2023, renal, India	16	Lung 75%, brain 12.5%	Mortality 5/16 (31.25%)
Dalil 2025, pooled published cases	60	Lung only 37, lung and brain 13	Cure 45/55 (81.8%)

Guidelines for solid organ transplant recipients recommend an initial parenteral phase followed by prolonged oral therapy for severe or disseminated disease [24]. Published practice reflects that. Among 54 pooled published kidney transplant cases, TMP-SMX was used in 32 (59.3%), carbapenems in 19 (35.2%), cephalosporins in 14 (25.9%) and linezolid in 11 (20.4%), with combination therapy in 38 (70.4%) [9]. In the Mayo cohort, 93.9% of patients with disseminated infection received initial combination therapy and 30/33 (90.9%) received a sulfonamide [12]. Monotherapy is feasible in selected patients: of 31 solid organ transplant recipients treated with TMP-SMX alone, 20/31 (65%) of them kidney recipients, 24 (77%) completed at least 30 days, while 10/31 (32%) discontinued prematurely, 3 for haematological toxicity and 3 for rising creatinine [25]. Cure rates by regimen in the pooled published series, 14/15 (93.3%) for monotherapy against 31/38 (81.6%) for combination therapy, are confounded by indication, because combination therapy was given to the sicker and more extensively infected patients [9].

Mortality is substantial. One-year all-cause mortality was 16.2% (19/117) among European transplant recipients with nocardiosis against 1.3% (3/233) in matched control recipients [26]. Twelve-month mortality was 24.2% (8/33)

with disseminated infection and 14.1% (13/92) without [12]. One-year survival was 66/82 (80.5%) in transplant recipients with invasive nocardiosis against 155/186 (90.6%) in non-transplant patients [10]. Renal series report mortality of 5/16 (31.25%) in India, concentrated among patients with brain involvement [8], and cure in 45/55 (81.8%) of pooled published kidney transplant cases with recorded outcome [9]. Earlier pooled analyses of published kidney transplant cases reached similar qualitative conclusions [27]. Among individually reported cases, outcomes were favourable where diagnosis was followed by prolonged susceptibility-guided therapy and reduction of maintenance immunosuppression [28].

Conclusion

The published record supports two firm recommendations and one honest ambiguity. Every renal transplant recipient with nocardiosis should have brain imaging at diagnosis, because 13 of the 30 patients with central nervous system disease in the largest European cohort had no neurological symptoms [6], and because cerebral involvement extended median therapy to 499 days against 296 days among survivors overall [12]. Reported sulfonamide resistance much above 5 to 10% should be regarded as unconfirmed: the 42% figure derived from referred isolates [17] is not reproduced by reference-method series [18] [19], is undermined by six-laboratory reproducibility data [20], and finds no support in the genotype of phenotypically resistant isolates [16]. TMP-SMX at Pneumocystis doses reduces but does not exclude nocardiosis [22], and neither imaging nor empirical therapy should be withheld because a patient is already taking it.

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