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Case report

Pulmonary nocardiosis in kidney transplant recipients: A case report and analysis of 60 published cases

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ABSTRACT

Background: Pulmonary nocardiosis (PN) is a rare life-threatening opportunistic infection, particularly involving immunocompromised patients, including kidney transplant recipients (KTRs). This study aimed to present a case of PN in a KTR and review published cases to describe the demographic, clinical, and treatment characteristics of PN in this population.

Methods and results: Here, we report a case of PN in a 58-year-old diabetic patient with a history of kidney transplantation. Upon admission, metronidazole and antifungal therapy was initiated empirically, considering a chest CT suggestive of fungal infection and pneumonia. In culture evaluations, *Nocardia* was isolated from bronchoalveolar lavage cultures. After PN diagnosis and tailoring antibiotic regimen to Trimethoprim-sulfamethoxazole (TMP-SMX), the patient showed significant improvement. This study also reviewed the English-language literature published from 1881 to 2024 about PN in KTRs and collected various characteristics of 60 similar patients. Moreover, antibiotic and the type of combination therapy used for treatment and outcomes were discussed. The median time of onset of PN after kidney transplantation was 180 days (75.5–730). Lung was the only affected organ by *Nocardia* in 37 patients, while in 18 patients, simultaneous involvement of the lungs and the brain was reported. Fever (74.3 %) was the most reported symptom, followed by cough (50.8 %), headache (41.8 %), and dyspnea (30.9 %). TMP-SMX was the most frequently prescribed antibiotic, having been provided to 32 patients (56.1 %). Other antibiotics included carbapenems, cephalosporins, amoxicillin/clavulanic acid, etc. Furthermore, 38 patients (70.4 %) received combination therapy, whereas 16 patients (29.6 %) received a single antibiotic agent. Lastly, the outcomes of 53 patients were reported. While the majority (81.8 %) were successfully treated, 10 patients (18.2 %) expired due to comorbidities.

Conclusion: Physicians should consider the diagnosis of PN in the differential diagnosis of KTR presenting with pneumonia, especially within the first six months post-transplant, when the risk of nocardiosis is elevated due to intensive immunosuppression.

Introduction

Nocardiosis is a rare infectious disease caused by the aerobic, gram-positive, catalase-positive, partially acid-fast, non-motile bacterium from the genus *Nocardia* spp. [1]. *Nocardia*, found mainly in soil and standing water, manifests as an opportunistic infection, especially in immunocompromised patients [2]. The incidence of *Nocardia* infection has been reported to be 0.4–3.6 % in solid organ transplant recipients [3, 4]. Kidney transplant recipients (KTRs) are more susceptible to various infection due to receiving immunosuppressive therapy, leading to severe complications [5]. However, the rate of nocardiosis in KTRs is lower compared to other solid organ transplant recipients [6].

Pulmonary infection is the most common manifestation of nocardiosis followed by brain abscesses in KTRs [7,8]. Pulmonary nocardiosis (PN) may present as a common type of localized disease or be the primary site of systemic infection spreading to other organs mostly the brain and skin. Inhalation of bacteria-containing aerosol is considered the most common route of lung infection. Further, the bacterium can

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inoculum directly within the skin and the soft tissues (for instance, the healthcare-associated infection due to invasive procedures) but cannot spread straight between humans [9].

The mortality of IFDs due to non-disseminated nocardiosis has been reported to be 15–20 %, while for disseminated nocardiosis it is significantly higher, ranging between 30 % and 60 % [7,10]. Previous retrospective studies have shown that the mortality rate could be reduced considerably through early diagnosis and timely treatment of nocardiosis in SOTRs [10]. Considering that it takes several days for the organism to grow in culture, clinical suspicion based on clinical manifestations and radiological findings could be lifesaving [11]. Here, we report a case of PN in a diabetic patient with a history of kidney transplantation. Further, we reviewed the English-language literature about PN in SOTRs, reporting the data on demographic, clinical characteristics, treatment, and outcome of included cases, and analyzed them using descriptive statistics to provide better knowledge of PN management in SOTRs.

Case presentation

A 63-year-old Iranian man with history of renal transplantation presented to Shariati hospital, Tehran, Iran, with bilateral pain of the lower thorax, mild fever, productive cough, and dyspnea, for 10 days. He had no symptoms of orthopnea, paroxysmal nocturnal dyspnea (PND), hemoptysis, and diaphoresis. His past medical history was remarkable for diabetes mellitus (DM), ischemic heart disease (IHD), hypertension (HTN), atrial fibrillation (AF), and hepatitis B. He had end-stage renal disease (ESRD) due to diabetic kidney disease (DKD).

Then, after receiving dialysis for months, he received a deceased donor kidney transplant seven months before admission. At the time of transplantation, the patient received anti-thymocyte globulin (ATG) as induction immunosuppressive therapy. The patient was on tacrolimus (3 mg twice daily), mycophenolate mofetil (1 g twice daily), prednisolone (20 mg daily), trimethoprim-sulfamethoxazole (TMP-SMX) (80/400 mg daily), amlodipine (200 mg daily), metformin plus sitagliptin (25 mg daily), and insulin. Of particular note is that this patient was already on TMP-SMX prophylaxis (80/400 mg daily).

On examination, he was alert and oriented. The initial vital signs were: Body temperature: 37.4 °C, Heart rate: 76 bpm, Respiratory rate: 18 breaths/min, Blood pressure: 130/75, Oxygen saturation: 93 %. Lung auscultation revealed a Rhonchi in the lower part of both lungs.

The initial blood tests revealed a white blood cell (WBC) count of 9,500/mm3, hemoglobin (Hb) 11 g/dL, mean corpuscular volume (MCV) 86, platelet count of 230000/mm^3, blood urea nitrogen (BUN) 22 mg/dL, serum creatinine (Cr) 1.49 mg/dL, erythrocyte sedimentation rate (ESR) 75 mm/hr, C-reactive protein (CRP) 260 mg/L. The arterial blood gas (ABG) test reported pH 7.37, pCO2 34 mmHg, and bicarbonate (HCO3-) 20 mmol/L. The urine analysis showed 2 + protein and 2 + glucose. The serum galactomannan antigen test was checked twice to rule out the invasive pulmonary aspergillosis and both resulted negative. Several blood culture tests and two urine culture tests were all negative.

A spiral chest computed tomography (CT) scan has demonstrated a 45 mm alveolar opacity in left lower lobe (LLL) with a small satellite lesion, and a ground glass lesion with a severe halo containing air bubble measuring 38 × 45 mm in right lower lobe (RLL) (Fig. 1). There was no evidence of pleural effusion. Transthoracic echocardiography (TTE) has shown no vegetation, pulmonary embolism (PE), clot, and ejection fraction (EF) was 50 %. Abdominal and pelvic ultrasound revealed the transplanted kidney with normal size and echogenicity and atrophic native kidneys. Other organs including the liver, gallbladder, spleen, pancreas, ureter, and bladder were normal. There were no findings compatible with infection in either abdomen or pelvis.

Since the patient was a case of kidney transplantation and had immunosuppression, antifungal, and antibiotic treatment was started empirically based on chest CT findings suspicious of bacterial or fungal pneumonia. The immunosuppressive regimen was modified with the discontinuation of the mycophenolate mofetil and the decrease of tacrolimus to 1 mg twice daily. The antimicrobial regimen was ciprofloxacin (500 mg/8 h), meropenem (1 g/8 h), vancomycin (750 mg/daily), and voriconazole (200 mg/12 h). Following, bronchoscopy showed mild to moderate secretion in RUL and LLL, and a specimen from bronchoalveolar lavage (BAL) fluid was sent to laboratory for microbial culture.

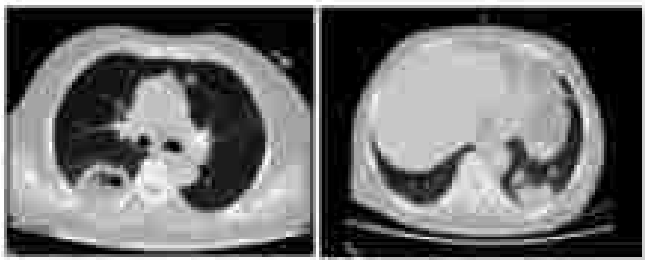

Fig. 1. Computed tomography (CT) scan of thorax has demonstrated a ground glass lesion with a severe halo containing air bubble measuring 38 × 45 mm in right lower lobe (RLL), and a 45 mm alveolar opacity in left lower lobe (LLL) with a small satellite lesion.

After receiving voriconazole for two weeks, the patient's tacrolimus serum level increased up to 30 ng/mL due to drug-drug interaction with voriconazole. As a result, tacrolimus was stopped for three days successfully. Then, the liposomal amphotericin B (150 mg/daily) was prescribed instead of voriconazole for one week. Further, the BAL fluid and sputum cultures yielded *Nocardia* spp. However, the species of *Nocardia* was not identified. Therefore, the patient's treatment was continued with intravenous injection of TMP-SMX (10 mg/kg/day of TMP) for a week and previous antibiotics were discontinued.

Seven days after diagnosing PN and administering disease antibiotic, the patient's symptoms of cough and dyspnea were markedly improved as well. Blood tests showed levels of CRP and ESR within the normal range and creatinine 1 mg/dL. Moreover, brain CT scan was normal and showed no dissemination. Following, the patient was discharged with instructions to continue therapy with oral TMP-SMX (8 mg/kg/day of TMP) along with tacrolimus (3 mg twice daily), mycophenolate mofetil (500 mg daily), and prednisolone (5 mg daily) for next 12 months. During one-year post-infection, the patient was evaluated every 2 months by the kidney center of Shariati Hospital. No complication or recurrence was observed. He survived and followed the treatment well to the end.

Analysis of published cases

The PubMed and Web of Science databases were searched using the following search strategy: (nocardiosis OR *Nocardia* infection*) AND (kidney OR renal) AND (transplant*). The duplicate literatures were initially deleted and results were limited to English-language case reports and case series articles published from 1980 to March 2025. Subsequently, 83 articles were screened based on title and abstract and 41 literatures containing 60 cases of RTR with PN were included [3–43]. In the next step, the included articles were thoroughly reviewed and the following data were extracted for reported cases: demographic and clinical characteristics, type of comorbidity, *Nocardia* species, clinical manifestations, lung imaging findings, treatments, and outcomes. Finally, all the data were analyzed based on descriptive statistics using SPSS software version 26 (SPSS Inc., Chicago, Illinois, USA).

Results

Table 1 demonstrates the demographic data and the clinical characteristics of RTRs with PN. The mean age of all patients was 49.33 ± 12.78 years (ranged 26–75 years). All the articles, except one [39],

Table 1
General, demographics and clinical characteristics of kidney transplant recipients with pulmonary nocardiosis.

	Number of patients	Cases	Percentage
Median age, years (Q1–Q3)	60	53 (41.5–65)	
Median time of onset of infection after TX, days (Q1–Q3)	60	182 (70.5–732)	
Gender	59		
Male		32	59.3%
Female		24	40.7%
Source of donor	59		
Deceased		37	62.7%
Living		22	38.3%
Rejection	37	9	24.3%
Cause of ESRD	59		
GN		13	48.3%
HTN		5	20.0%
PCKD		4	18.9%
DM		1	4.0%
Renal TB		1	4.0%
Pyelonephritis		1	4.0%
Immunosuppressant therapy	59		
Tacrolimus		22	38.4%
Glucocorticoids (mPSL)		19	48.7%
Corticosteroids		37	94.8%
Azathioprine		14	35.9%
Cyclosporine		13	33.3%
Medical history*	27		
HTN		18	55.5%
DM		2	7.4%
AF		2	7.4%
Valvular Heart Disease		2	7.4%
CKD		4	14.8%
Hepatitis and cirrhosis		2	7.4%
Hypothyroidism		2	7.4%
Hypogammaglobulinemia		1	3.7%
Asthma		2	7.4%
ESRD		2	7.4%
Fungal infection		2	7.4%

MTX: Hypertension, DM: Diabetes mellitus, GN: Glomerulonephritis, PCKD: Polycystic kidney disease, SLE: Systemic lupus erythematosus, TB: Tuberculosis, IHD: Ischemic heart disease, AF: Atrial fibrillation, PTE: pulmonary thromboembolism.

* a Patient may have more than one medical condition.

Table 2
Clinical symptoms, *Nocardia* species and lung imaging findings of patients.

	Number of patients	Percentage
Type of Nocardiosis	80	
Pulmonary alone	67	83.7%
Pulmonary + Cerebral	10	12.5%
Pulmonary + Cutaneous	2	11.7%
Pulmonary + Cerebral + Cutaneous	1	0.9%
Nocardia species	47	
N. asteroides	22	46.8 %
N. farcinica	22	38.8 %
N. brasiliensis	2	4.2 %
N. pseudobrasiliensis	2	4.2 %
N. nevadensis	2	4.2 %
N. wallacei/otitidiscaviarum	1	2.1 %
N. cyriacigeorgica	1	2.1 %
N. abscessus	1	2.1 %
N. beijingensis	1	2.1 %
N. pneumoniae	1	2.1 %
N. aurea	1	2.1 %
N. veterana	1	2.1 %
Clinical manifestations	85	
Fever	41	74.5 %
Cough	28	50.9 %
Dyspnea	17	30.9 %
Chest pain	17	30.9 %
Skin lesion	11	20.0 %
Impaired Consciousness	8	14.5 %
Neurological Symptoms	5	9.1 %
Weakness	5	9.1 %
Headache	4	7.2 %
Extremity pain	2	3.6 %
Shock	2	3.6 %
Lung imaging findings	58	
Consolidation	38	65.5%
Infiltration	23	39.6 %
Effusion	13	22.4 %
Cavitation	3	5.2 %
Lung Abscess	2	3.5 %

reported the gender of the patients. Of these, 32 were male (39.5 %) and 24 were female (40.7 %). The median time of *Nocardia* infection onset after kidney transplant was 182 days (70.5–732). 24 articles (containing 28 cases) stated the type of kidney donor as either deceased (60.7 %) or living (39.3 %) [12–14,16,20,24,26–28,30–33,35,37–41,43,44,47–49,51]. Some of the included papers reported the details of patients' transplants. The graft rejection was observed in nine patients (24.3 %) [12,13,33,39,50]. Among patients with graft rejection, the mean time from transplantation to onset of nocardiosis was 397.5 ± 305.02 days (ranged 32–1478 days), suggesting a tendency toward earlier infection in this subgroup. The level of Cr in kidney-transplanted patients was detailed in 13 papers (15 cases) (mean Cr level: 1.6 ± 0.4 mg/dL, 1.02–2.26).

The immunosuppressant therapy details were also discussed for 59 cases, where corticosteroids were most commonly used (94.9 %). Only a limited number of patients have data on induction immunosuppressive treatment. ATG and basiliximab were the most common drugs that reported. However, since induction regimen details were missing in the majority of the reviewed cases, a comprehensive analysis was not possible. Glomerulonephritis (GN) was the leading reported cause of ESRD among cases. The most common underlying disease reported in transplanted patients was hypertension (18 of 27 cases, 55.5 %).

Clinical manifestations, Nocardia species and lung imaging findings of patients are reported in Table 2. The most observed symptom was fever (74.5 %), followed by cough (50.9 %), headache and myalgia (41.8 %), and dyspnea (30.9 %). Neurologic manifestations such as seizure, unsteady gait, and focal numbness were also noticed in five cases (9.1 %) [13,21,24,28,50]. Moreover, eight patients (14.5 %) had impaired consciousness when diagnosed [13,20,22,25,28,29,50], and two cases (3.6 %) were in shock [21,25].

Regarding the involved organ with *Nocardia* infection, 37 patients had lung involvement alone while in 13 patients the lung and brain were involved. The identified *Nocardia* species were also discussed in most of the studies (47 cases). *N. asteroids* was the most commonly isolated species (46.8 %), followed by *N. farcinica* (n = 12, 25.5 %), *N. brasiliensis* (n = 2, 4.2 %), *N. pseudobrasiliensis* (4.2 %), and *N. nevadensis* (4.2 %). Clinical characteristics of the infected patients were specified in the majority of the articles (33 cases).

Lung imaging was discussed for 58 patients. Seen in 38 cases (80 %), pulmonary consolidation was the most commonly observed pattern. Infiltration (39.2 %) and effusion (22.4 %) were also frequently reported. In this article, 'infiltration' is being used to describe poorly defined or patchy, less dense and less well-defined opacities, compared with consolidation, representing homogenous shoulder filling.

The antibiotic agents administered to treat nocardiosis were discussed for 34 patients (Table 3). Being administered in 32 cases (94.2 %), TMP-SMX was the most commonly applied antibiotic. Other agents were carbapenems (Imipenem or Meropenem), cephalosporins (Ceftriaxone, Cefuroxime, Ceftazidime, etc.), Amoxicillin-Clavulanic acid, macrolides (Erythromycin, Azithromycin, etc.), aminoglycosides (Amikacin, Gentamycin, and Kanamycin) etc. About the treatment regimen, 25 patients (73.4 %) underwent combination antibiotic therapy (i.e., receiving two or more antibiotics at the same time), and 16 cases (26.5 %) received a single antibiotic (i.e., may be further changed to another agent but not receiving more than one antibiotic simultaneously). The duration of antibiotic therapy was reported for 43 cases

Table 2
Nocardiosis treatments and outcomes.

	Number of patients	Percentage
Antibiotics	34	
TMP-SMX	22	39.3%
Amoxicillin/Clavulanic acid	11	32.4%
Carbapenems	13	38.2%
Cephalosporin	14	41.2%
Minocycline	10	29.4%
Linezolid	11	32.4%
Aminoglycosides	5	14.7%
Macrolides	9	26.5%
Other Drugs*	10	29.4%
Combination Therapy**	38	70.4%
TMP-SMX + Linezolid	6	15.8%
TMP-SMX + Carbapenems	8	21.1%
Carbapenems + Linezolid	3	7.9%
Carbapenems + Cephalosporin	7	18.4%
TMP-SMX + Cephalosporin	9	23.7%
Outcomes	55	
Death	10	18.2%
Cure	45	81.8%

TMP-SMX, Trimethoprim-sulfamethoxazole.
* Other drugs include Penicillin, Sulfadiazine, Tetracycline, Amphotericin B, Fusidic acid and ...
** Patients might have used more than two agents as combination therapy.

(ranged 6–899 days) of which six died during the treatment. The mean duration of antibiotic therapy among expired patients and cured patients was 27.3 ± 29.7 days (ranged 6–84 days) and 236.0 ± 136.4 days (ranged 30–539 days), respectively.

In addition to management measures, the final outcome of 55 infected cases was also revealed. 45 cases (81.8 %) were cured, and 10 patients (18.2 %) expired due to nocardiosis. Of the 53 patients with both treatment regimens and outcome information available, 15 patients received monotherapy and 38 received combination therapy. Of the patients receiving monotherapy, 14 patients (93.3 %) were cured and one patient (6.7 %) died. The safe fatality among these patients had received TMP-SMX. In the combination therapy group, 31 patients (81.6 %) were cured and 7 patients (18.4 %) died.

Of the 10 patients who died, 7 had isolated pulmonary nocardiosis, while 3 had pulmonary and cerebral involvement, suggesting that disseminated infection may contribute to the increased mortality. Of the 8 patients for whom data on posttransplant injection were available, most had documented episodes of rejection. The mean time from transplantation to diagnosis of nocardiosis in the 8 patients was 810 days (range, 21–4745 days), suggesting that the diagnosis of post-transplant infection was later in this group.

Discussion

In our review, the mean duration between transplantation and PN onset was 36 months; however, the median time was shown as 6 months, indicating that PN most frequently presents early, but can occur even many years after transplant. For instance, Marak et al. documented cases of late-onset nocardiosis in several RTRs, even after extended follow-up periods [32]. Studies reported that the threat of nocardiosis rises in the initial year after kidney transplantation. The risk is greatest in the first six months post-transplantation [33]. This is usually attributed to the highest intensity of immunosuppressive agents to avoid transplant rejection [34]. Yu et al. [34] study showed that those who experienced prior graft rejection appeared to develop Nocardia infection faster (mean: 13.8 months). In our review, data about graft rejection was provided in 37 patients out of 60 cases, of which just 8 patients with PN experienced post-transplant rejection. Similarly, this study demonstrated that Nocardia infections may happen earlier in patients experiencing graft rejection (mean: 11.76 months). Although the duration between transplantation and the diagnosis of nocardiosis was seven months in our case, there was no evidence of graft rejection.

In a recent study from Pakistan, Nasim et al. examined 48 RTRs who developed nocardiosis. They found that, on average, the infection was diagnosed 2.43 years after the transplant, indicating that late-onset nocardiosis continues to pose a serious risk for this group. The research also highlighted two independent risk factors for developing the condition: the use of high-dose methylprednisolone and severe CMV infection [35].

The data regarding medical history was recorded for 37 cases, where hypertension was the most reported (35.5 %), followed by diabetes (14.5 %). These diseases were also noted in our case. Hypertension is associated with CKD, which may necessitate kidney transplantation. Moreover, Zuhair et al. also noted hypertension as the most frequent underlying disease in PN cases [36]. Other studies demonstrated that comorbidities such as diabetes, persistent pulmonary or hepatic diseases, alcoholism, and immune-compromising conditions like malignancies, hematopoietic cells and organ transplantation, as well as immunosuppressive medications, all increase the risk of developing PN [37–39]. The most common cause of ESRD among RTRs with PN was GN, with 46 % of cases. GN patients are treated with immunosuppressive medications, which increase the risk of opportunistic infections such as Nocardia.

Previous research has shown that the clinical presentations of nocardiosis may be similar between immunosuppressed patients and normal hosts [37,40,41]. Clinical manifestations of PN were described in 55 patients. The most reported symptoms were fever (74.5 %), cough (60.8 %), and dyspnea (30.9 %), respectively. All of these symptoms were also observed in our case. However, cases with a compromised immune system might be more susceptible to Nocardia dissemination, hospital admissions, and positive blood specimens. They are also at higher risk of death due to nocardiosis [37]. Neurologic symptoms and impaired consciousness were also reported in five (9.1 %) and eight (14.5 %) cases. These manifestations may infiltrate the CNS infection and necessitate early CNS imaging and the start of appropriate treatment. Interestingly, some studies reported nocardiosis cases with asymptomatic CNS infection [37,41]. Hence, imaging the CNS (e.g., MRI) can be considered an evaluation tool in patients with nocardiosis even in the absence of neurologic manifestations [37].

Our review showed that PN was confined to the lung in the majority of RTRs (37 of 60 cases). This may be attributed to the mechanism of infection, which is mainly inhalation. However, in 13 patients (21.6 %), the Nocardia infection spread to the brain, and they developed pulmonary and cerebral nocardiosis. When the infection is evident in two separate organs or in the presence of central nervous system (CNS) involvement, disseminated nocardiosis is identified [34]. Nasim et al. also found that 25 % of their patients developed disseminated nocardiosis, including involvement of the CNS, further highlighting the aggressive nature of the disease in immunocompromised individuals [35]. Dissemination is more likely when there is evident immunosuppression, such as consumption of high doses of steroids and organ transplantation [37]. Previous research demonstrated that up to 50 % of PN patients may subsequently have disseminated nocardiosis [35]. In our patient, the infection was limited to the lungs, and investigations revealed no evidence of other organ involvement. Among the 60 cases reviewed, the outcome of 55 patients was reported, among which the mortality rate was 18.2 %, which was slightly higher than Yu et al. [34] study. In the study by Nasim et al., the overall mortality rate was reported at 20 %, which is comparable to the mortality rates observed in our review [35]. As RTRs were receiving immunosuppressive therapies, the incidence of disseminated nocardiosis and death was expected to be higher compared to immunocompetent patients [34].

Regarding Nocardia species, specified for 47 patients, *N. asteroides* (46.8 %) was the most recognized species, mainly reported between 1981 and 2000 [13,18,20,22,34,38–42,42,43–47]. It should be noted that older articles may suffer from misconception of Nocardia species because historically several species have been misidentified as

N. asteroides. Furthermore, *N. farcinica* was identified in 12 patients (22.6 %). This is consistent with some previous studies [28,47]. However, Fujita et al. reported that *N. nova* was the most frequent species (49 %) responsible for PN occurring in organ transplant recipients [32]. On the contrary, Chuaychoot et al. demonstrated that the species *N. farcinica* (38 %), followed by *N. nova* (24 %), were the most commonly recognized [43]. Hence, the geographical predominance of species, involved organs, and the medical history of patients may play a role in the species implicated in nocardiosis [20,21].

The radiological presentations were reported for 56 cases, of which 28 (50 %) had pulmonary consolidations. Observed in 22 patients (39.3 %), infiltration was the second most common finding. Other radiological findings were effusion (23.2 %), cavitation (8.9 %), and lung abscess (3.6 %). On CT imaging of our patient, the most noticeable findings were consolidation and a ground glass lesion. Some previous studies described consolidation followed by cavitation as the most common radiological findings in PN [34,35]. Zabala et al. reported that infiltration followed by nodules and consolidation were the most observed radiological patterns [36]. Furthermore, infiltration and abscess were noted as the most frequent findings in the Ye et al. study [32]. In that regard, there is not a thorough consensus among previous investigations regarding the most common radiological pattern. Some studies also argued that there may be an association between some radiological findings in PN and immunosuppression. Steinbrink et al. [37] and Kim et al. [38] described cavitation, and Blackmon et al. [39] noted nodules as associated with immunosuppression in PN patients.

The first-choice treatment for nocardiosis is TMP-SMX, including in transplant recipients, which is commonly used in combination with carbapenems in complicated patients [27]. For severe infections, including pulmonary or disseminated nocardiosis, dosing is recommended as follows: 15 mg/kg/day of TMP, divided for either oral or intravenous dosing in 2–4 doses, depending upon severity of illness and host status [24]. For any-dose or milder cases, oral dosing at lower levels (5–10 mg/kg/day of TMP) can be employed. Transplant hosts tend to require adjustment in dosing in accordance with the patient's renal function, as TMP-SMX can rise serum levels of creatinine as well as potassium. Adjustment in dose interval or TMP-SMX discontinuation should in some cases be employed in those with compromised GFR. Prolonged treatment mandates monitoring of the glomerular function, hepatic levels, as well as monitoring of electrolytes. Prophylactic dosing (e.g., once-a-day dosing with 80/400 mg) decreases *Pneumocystis jirovecii* as well as some *Nocardia* spp. infections, yet breakthrough still occurs, most notably in those with intensive immunosuppression [3,40].

In this review, data regarding the treatment of 54 cases was provided, of which 39 (39.8 %) received TMP-SMX. Carbapenems were also used in 19 (35.2 %) patients. The third most frequently prescribed agents were cephalosporins (28.9 %). Moreover, combination therapy was used in most of the cases (72.4 %), and the most common compounds were TMP-SMX plus carbapenems (22.7 %) or TMP-SMX plus cephalosporins (22.7 %). Some studies exhibited that TMP-SMX is still effective in the treatment of nocardiosis and the resistance is not considerable [20,27,47,48]. However, some studies reported resistance to TMP-SMX among some *Nocardia* species, especially *N. farcinica* and *N. nova* [28,47]. Therefore, it may be important to isolate and identify the *Nocardia* species by investigating the patients' specimens and performing sensitivity tests to apply the most appropriate therapy [27].

Since *Nocardia* species have variable antimicrobial susceptibility patterns, susceptibility testing is needed in order to direct appropriate therapy [49,50]. *Nocardia* species have a broad range of intrinsic and acquired resistances that are also variable between species. For example, *N. asteroides* is usually susceptible to TMP-SMX, but *N. farcinica* and *N. otitidiscaviarum* may be resistant to first-line drugs such as TMP-SMX or certain β-lactams [47,48]. Delayed diagnosis and inappropriately directed therapy in immunocompromising conditions, including recipients of kidney transplants, are associated with severe clinical deterioration and increased mortality [4,5,51].

Fujita et al. argued that commencing the treatment based on the culture depends on the patients' immunity status. While delayed therapy may not compromise immunocompetent cases with a positive culture and temporary observation may be an approach in these patients, the high incidence of *Nocardia* infection in culture-positive immunosuppressed patients necessitates the prompt initiation of appropriate treatment [32]. However, previous studies were not consistent regarding the prescription of TMP-SMX as a prophylactic agent for nocardiosis in transplant patients. A lower dose of TMP-SMX was recommended as a successful prophylaxis in some studies [52]. In contrast, nocardiosis was reported in a considerable number of transplant patients despite using TMP-SMX for prevention [53]. Similarly, Marvin et al. reported a higher incidence of nocardiosis in KTRs who experienced refusal or interrupted TMP-SMX prophylaxis, particularly during the COVID-19 pandemic [54]. Additionally, Yoon et al. pointed out that while TMP-SMX prophylaxis is routinely administered, breakthrough nocardiosis can still occur, underscoring the importance of ongoing vigilance even in patients receiving prophylaxis [55].

In our series, the mean course of antibiotic treatment among patients dying was significantly shorter compared with the surviving patients (27.2 vs. 236.2 days). This most likely represents early death secondary to severe or disseminated infection or late diagnosis. Early recognition and prompt treatment of nocardiosis is necessary because nocardiosis progresses extremely rapidly in immunocompromised patients, particularly when CNS involvement is present.

Although we systematically searched the PubMed and WOS databases and reviewed the retrieved articles (based on PRISMA guidelines), our review faced several limitations including 1) a limited number of included patients, 2) publication bias, 3) missing cases that are not present in included databases, 4) not including non-English-language articles, and 5) missing data between cases. Further, this report only reviewed literature until March 2025 and other relevant publications about *Nocardia* infections may have been published and not included in this report. In a new idea, instead of just reviewing the results of the included articles, we tried to accumulate the data obtained from the cases and analyze them using descriptive statistics to provide better knowledge of the subject.

Conclusions

PN can develop at any time since kidney transplant, though there may be a higher incidence in early post-transplant. The transplant physicians should have a high index of suspicion for PN in any case of pneumonia occurring in KTRs, particularly in those with prolonged or augmented immunosuppression. Accurate early diagnosis along with appropriate antibiotic treatment can help in avoiding potentially lethal complications. TMP-SMX, given alone or in combination with carbapenems or cephalosporins, continues to be a mainstay of treatment. Additionally, prophylaxis with TMP-SMX seems to decrease nocardiosis in transplant recipients, though there can still be breakthrough infections.

CRediT authorship contribution statement

Davood Dalili: Writing – review & editing, Writing – original draft, Supervision, Investigation, Data curation, Conceptualization. Fatemeh Taghizabi: Writing – review & editing, Project administration, Investigation, Conceptualization. Farnaz Tavabali: Writing – review & editing, Investigation. Seyyed Mohammad Mostafa: Writing – review & editing, Writing – original draft, Formal analysis. Mahdi Isakhani: Writing – review & editing, Resources.

Ethics Statement

The Research Ethics Committee of Tehran University of Medical Sciences approved all procedures performed in the current case report.

with the approval number: IR.TUMS.SHARIATI.REC.1402.104.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability Statement

The data used to support the findings of this study are available from the corresponding author upon request.

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