Renal Replacement Therapy

CASE REPORT

Open Access

Invasive aspergillosis in the patient with focal segmental glomerulosclerosis initiating hemodialysis: a case report and mini-review

Noriaki Sato[1], Hideki Yokoi[1*], Mitsuhiro Ichioka[1,2], Akira Ishii[1], Takeshi Matsubara[1] and Motoko Yanagita[1]

Abstract

Background: Invasive aspergillosis (IA) is a severe form of fungal infection caused by the genus *Aspergillus* in immunocompromised hosts and has a high mortality rate. End-stage kidney disease (ESKD) is one of the risk factors for developing fungal infection; however, the detailed clinical and treatment course of ESKD patients with IA has been scarcely reported, especially for the patient initiating hemodialysis (HD). Here, we experienced a patient under immunosuppressive therapy for focal segmental glomerulosclerosis (FSGS) who suffered from IA involving lung and brain and resulted in initiating HD.

Case presentation: A 66-year-old male patient with a history of suspected non-tuberculosis mycobacterial lung disease was initially admitted to the hospital with minimal change disease and subsequently diagnosed as FSGS with worsening urinary protein levels. The combined treatment including immunosuppressive treatments of cyclosporin and glucocorticoids and low-density lipoprotein apheresis was initiated, and then, he experienced the symptoms of dry cough, somnolence, and disorientation, which were subsequently diagnosed as IA involving lung and brain. The patient required renal replacement therapy, and maintenance HD was continued. Despite the intensive treatment with multiple antifungals of liposomal amphotericin B, voriconazole, micafungin, and amphotericin B, the pneumonia of the patient did not improve, and he subsequently passed away.

Conclusions: We report the case of the IA under immunosuppressive treatment, who was subsequently initiated maintenance HD. The detailed clinical course of medications used to treat the patient is presented with the literature review of IA in ESKD and HD patients and those with past acid-fast bacterial infections. The careful determination of the intensity of immunosuppression and monitoring of the patient's symptoms and early definitive diagnosis is crucial in treating IA in immunocompromised hosts with ESKD or in HD under immunosuppressive treatment, as the mortality for these patients is suspected to be high despite the intensive treatment.

Keywords: Invasive aspergillosis, Hemodialysis, End-stage kidney disease, Antifungals

Background

Invasive aspergillosis (IA) is a severe form of fungal infection in immunocompromised hosts caused by the fungal genus *Aspergillus*, which affects multiple organs [1]. Patients with IA have a high mortality rate of one study reporting a twelve-week disease-specific survival rate of 59.8% [2]. The risk factor for developing IA includes

*Correspondence: yokoih@kuhp.kyoto-u.ac.jp

[1] Department of Nephrology, Graduate School of Medicine, Kyoto University, 54 Shogoin Kawahara-Cho, Sakyo-Ku, Kyoto 606-8507, Japan
Full list of author information is available at the end of the article

chronic obstructive pulmonary disease, admission to intensive care unit (ICU), the comorbidities like diabetes mellitus (DM), or medications including chemotherapy [3]. Glucocorticoid use and acute kidney injury, as well as renal replacement therapy (RRT) in ICU, were identified to be prognostic factors of IA [4, 5].

Although aspergillosis in peritoneal dialysis (PD) patients presenting with aspergillus peritonitis has been reported [6], the detailed clinical course of IA in the patient with end-stage kidney disease (ESKD) or hemodialysis (HD), especially those initiating HD, has been scarcely reported. We experienced a patient with a history of suspected non-tuberculosis mycobacterial (NTM) lung disease, who was diagnosed with focal segmental glomerulosclerosis (FSGS) and initiated immunosuppressive therapy and died from IA involving lung and brain despite the intensive treatment with multiple antifungals.

Thus, the detailed clinical course with administered antifungals is reported, along with the literature review focusing on summarizing the previous case reports of IA in ESKD or HD patients as there is currently no prognostic study focusing on these patients.

Case presentation

A 66-year-old male with suspected latent tuberculosis was admitted to the previous hospital with the complaint of worsening edema 3 years before corresponding admission. He has no smoking history. On arrival, the urine protein-to-creatinine ratio (uPC) was 14 g/gCre, and the serum albumin value was 1.3 g/dL. He was initially diagnosed with minimal change disease (MCD) by kidney biopsy and was initiated immunosuppressive therapy combining intravenous and oral glucocorticoids, cyclosporine, and low-density lipoprotein (LDL) apheresis.

He was transferred to our hospital for follow-up with oral prednisolone (PSL) of 45 mg/day and oral cyclosporine (CyA) of 100 mg/day. On admission, serum creatinine value (sCr) was 1.85 mg/dL, and uPC was 3.0 g/gCre. As he was positive for interferon-gamma release assay, he was screened for acid-fast bacillus (AFB) smear test and was found negative. He was suspected of NTM lung disease from the plain computed tomography (CT) findings of nodular shadows with cavity formation in left upper lung.

The kidney biopsy was performed again, and he was diagnosed again with MCD. The oral mizoribine (MIZ) was added to taper the PSL dosage. He was discharged and was followed by our hospital with a uPC of around 1 g/gCre and sCr of 1.6 to 1.9 mg/dL. The prednisolone was tapered to 10 mg/day. He was also followed up for the suspected NTM lung disease.

On the year of the corresponding admission, his peripheral edema was worsening. On laboratory test performed on admission, uPC was worsened to 5.5 g/gCre, serum albumin value was lowered to 2.9 g/dL, and sCr was 1.86 mg/dL. The condition was suspected to be the recurrence of MCD, and he was hospitalized with increasing the dosage of PSL (20 mg/day). The laboratory data upon hospitalization are shown in Table 1. The patient was administered intravenous immunoglobulin (IVIG, 15 g) when the immunoglobulin G (IgG) value was below 300 mg/dL throughout the hospital stay. Despite increased PSL of 50 mg/day, uPC remained high, and the second renal biopsy in our hospital was reperformed on day 32. The diagnosis of FSGS was made based on the increased sclerosis region in glomeruli and the lack of atherosclerotic lesions. Subsequently, LDL apheresis was performed 12 times with the insertion of the blood access from day 44. PSL was gradually tapered with the initiation of LDL apheresis. CyA dose was adjusted to maintain 600 to 1000 ng/mL of the serum concentration value after 2 h of administration. We have followed the treatment suggestion described in the Evidence-Based Clinical Practice Guideline for Nephrotic Syndrome 2017 for selection of treatment for MCD and FSGS.

After initiating the treatment, uPC value gradually improved; however, he began to realize the symptom of dry cough. On examination, the plain chest CT revealed lobular central granular and branching shadows in the right upper lobe. The findings of the suspected NTM infection previously identified were not observed at this

Table 1 The laboratory data on hospitalization

Test (unit)	Value
White blood cell (/μL)	14,700
Neutrophil count (/μL)	13,400
Hemoglobin (g/dL)	13.0
Creatinine (mg/dL)	1.77
Estimated glomerular filtration rate (mL/min/1.73m^2)	30.7
Total protein (mg/dL)	4.9
Albumin (mg/dL)	2.7
Blood urea nitrogen (mg/dL)	42
Total cholesterol (mg/dL)	297
Low-density lipoprotein cholesterol (mg/dL)	178
Sodium (mEq/L)	138
Potassium (mEq/L)	4.2
Chloride (mEq/L)	105
Calcium (mg/dL)	8.4
Phosphate (mg/dL)	2.9
Immunoglobulin G (mg/dL)	251
Urinary pH	6.0
Urinary protein ratio (g/gCre)	6.43
Urinary protein (dipstick)	3+
Urinary occult blood (dipstick)	Negative

time. On the follow-up CT performed on day 82, new infiltrating shadows were found on the right upper and middle lobe (Fig. 1A). At this time, it was revealed that he was positive for the serum aspergillus antibody and β-D glucan (37.7 pg/mL) and was planned for bronchoscopy for the definitive diagnosis, and he was started ceftriaxone (1 g/day). On follow-up CT performed on day 93, nodular shadow with cavity formation in the right upper lung and infiltrative shadow in the bilateral lower lung were observed (Fig. 1B). The bronchial wash culture obtained on the bronchoscopy revealed the growth of *Aspergillus* spp. During this period, the IgG level of the patient remained low not exceeding 400 mg/dL despite the treatment with IVIG, possibly owing to the high level of proteinuria.

The pulmonary aspergillosis along with bacterial infection was suspected, and tazobactam/piperacillin (TAZ/PIPC, 3.375 g every 6 h) and liposomal amphotericin B (L-AMB, 150 mg/day: 3 mg/kg) were started as a treatment for aspergillosis from day 94. The overall clinical chart depicting the laboratory data, clinical events, and administered antifungals is shown in Fig. 2. On day 95, a chest X-ray revealed worsening infiltration shadow, and TAZ/PIPC was replaced for meropenem (MEPM, 1 g every 12 h). He was then revealed positive for *Pseudomonas aeruginosa* on blood culture. The inflammation markers were improved, and MEPM was replaced for ceftazidime (2 g every 12 h) on day 99. The immunosuppression with MIZ was stopped on day 107, and CyA was stopped on day 115.

However, on around day 117, the symptoms of somnolence and disorientation were recognized. The magnetic resonance imaging (MRI) revealed ring-shaped, high-signal areas in diffusion-weighted imaging at both lobes of the cerebrum, which were ring-shaped on T2 (Fig. 3A). The patient was suspected of having a brain abscess. A cerebrospinal fluid test was also performed, and cerebrospinal β-D-glucan was high (73.1 pg/mL) despite the ongoing treatment with L-AMB. Thus, L-AMB was increased to 250 mg/day. Subsequently, L-AMB was replaced with oral voriconazole (VRCZ, loading 300 mg every 12 h, maintained at 200 mg every 12 h) controlling for the blood concentration and micafungin (MCFG, 300 mg/day). The antibiotics were aborted as his symptoms gradually settled.

From day 131, inflammation markers again increased, and MEPM (1 g every 12 h) was again started. The pulmonary CT showed fixed cavity and pleural effusion on day 137 (Fig. 1C). On day 149, his respiratory status worsened, and thus, he was admitted to the ICU and was intubated and initiated mechanical ventilation. Subsequently, metabolic acidosis was worsened and urine output was lowered, and continuous hemodiafiltration (CHDF) was initiated. The used dialyzer was sepX-iris 150 under the quantity of dialysate flow rate (Qd) of 500 ml/hour, the quantity of substitution flow rate (Qs) of 500 ml/hour, and quantity of blood flow rate (Qb) of 80 ml/min. Nafamostat was used for 30 mg/hour as the anticoagulant. The amount of body fluid removal rate was adjusted according to blood pressure. The dosage of antifungals was remained unchanged under the CHDF condition. (VRCZ was administered 4 mg/kg every 12 h with the adjustment by blood concentration, and MCFG was administered 300 mg/day.)

The oliguria and pleural effusion persist, and the maintenance HD was started from day 169 and he was discharged from ICU on day 176. The maintenance HD was performed 4 h under the conditions of APS-08SA as a dialyzer, Qb of 120 ml/min, Qd of 500 ml/min. Nafamostat was used as anticoagulant. VRCZ was administered under the same conditions as during CHDF, and MCFG was administered at 300 mg daily and post-dialysis on dialysis days.

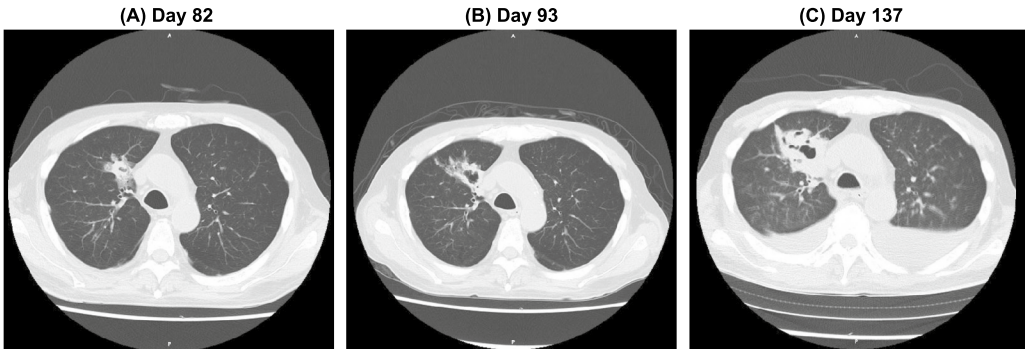

Fig. 1 The time course of lung CT images. The time course of lung CT images is shown. **A** Day 82 when the infiltration shadow is observed at the right upper lobe. **B** Day 93 when the cavity formation was observed. **C** Fixed cavity on day 137

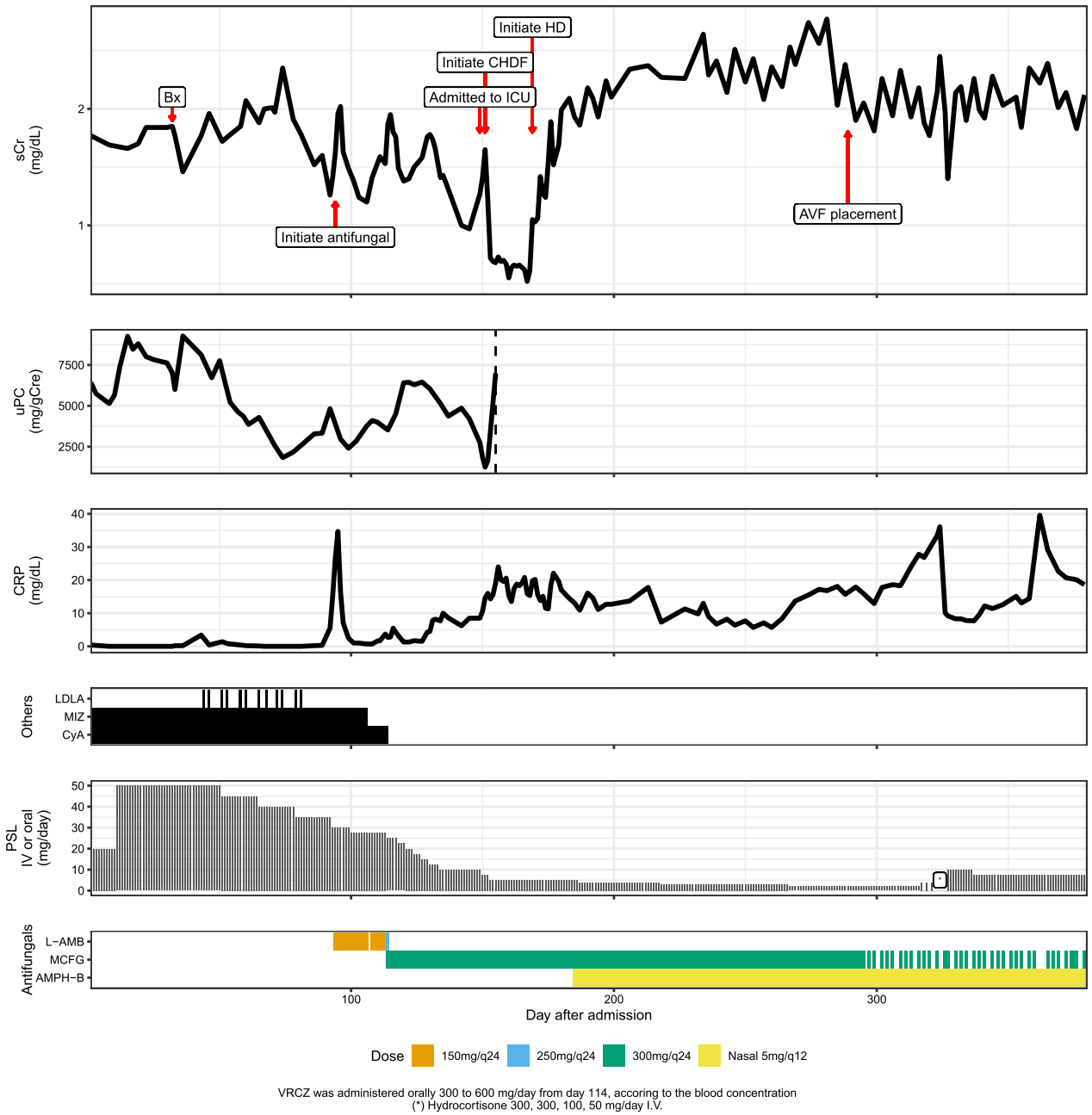

Fig. 2 The summary of the clinical course of the patient. The transition of serum creatinine, urinary protein–creatinine ratio, C-reactive protein, treatment, and the administered drugs are depicted. The events are summarized in the panel of serum creatinine. The x-axis shows the day after admission. AMPH-B, amphotericin B; AVF, arteriovenous fistula; Bx, biopsy; CHDF, continuous hemodiafiltration; CyA, cyclosporine; HD, hemodialysis; ICU, intensive care unit; L-AMB, liposomal amphotericin B; LDLA, LDL apheresis; MCFG, micafungin; MIZ, mizoribine; PSL, prednisolone; sCr, serum creatinine; uPC, urine protein-to-creatinine ratio; and VRCZ, voriconazole

The findings of plain lung CT remained unchanged, and amphotericin B (AMPH-B) from the nasal cavity was additionally started (5 mg/day). On day 220, brain abscess was gradually improved on brain CT (Fig. 3B); however, the findings of pneumonia remained unchanged. The arteriovenous fistula was placed on day 289, and he was discharged for the other hospital on day 380. He was reported to pass away one month later as the blood pressure could not be maintained despite the treatment with catecholamines.

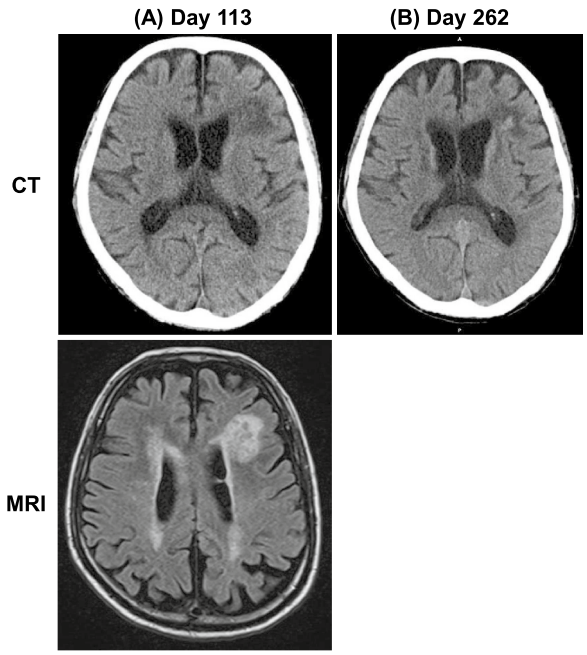

Fig. 3 The time course of brain CT and MRI images. The time course of brain CT and MRI images. **A** CT and MRI images at the timing of first detection of brain abscess at day 113. **B** The improvement in the abscess lesion in CT at day 262

Discussion

We report a case of IA with the involvement of lung and brain in the ESKD patients on immunosuppressive therapy resulting in initiating HD, and subsequent death despite the multiple treatments of antimicrobial and antifungal therapy in ICU. Previous studies indicate high mortality of those who developed IA with ESKD, despite the intensive treatment with multiple antifungals.

Patients with ESKD or those on HD are at increased risk for infections because of immune dysfunction [7, 8]. Although the induction of hemodialysis itself is thought not to have a major influence on developing IA considering the time course in this case, dialysis-related conditions such as malnutrition and iron overload have been reported to increase the risk of infection through impaired immunity, and it is important to manage these risks in long-term dialysis patients [9, 10].

Immunosuppressive treatment is a common risk factor for developing an infection. Among them, especially glucocorticoids, which are used to treat the present case, are reported to increase the risk of fungal infection [11, 12]. As the patient was thought to be steroid-resistant and dosage of PSL and duration of exposure was high with using CyA and MIZ for the reduction of uPC, these factors of disease severity and subsequent immunosuppressive intensity are thought to primarily contribute to developing IA. As such, extra caution for the early symptom of the fungal infection should be made for patients with ESKD under long immunosuppressive treatment to start early treatment. Additionally, factors such as low level of immunoglobulin G were considered to contribute to developing infection [13].

Although the causal relationship between suspected NTM lung disease and aspergillosis in terms of plain CT findings is unclear in the present case, one study found a higher prevalence of *Aspergillus* infection in NTM disease patients compared with the control in bronchiectasis patients [14]. Additionally, the ESKD patient presenting with IA suspected to occur in a past tuberculous cavity is reported [15]. We presumed that the ESKD patient with a history of tuberculosis or NTM diseases should be considered a high risk for developing aspergillosis, and the intensity of immunosuppression should be determined carefully.

As a treatment for IA, we gradually tapered the PSL, stopped the other immunosuppressive medications, and initiated a series of L-AMB, oral VRCZ, MCFG, and nasal AMPH-B to treat the patient which resulted in the improvement of brain findings but not in the pulmonary findings. Compared with previous reports, one-year survival from disease onset was considered to be relatively long in IA patients with ESKD.

In this regard, more rapid reduction of immunosuppression may have reduced the risk of IA in this case. In addition, the immediate administration of antifungal agents at the onset of symptoms, in this case, dry cough, may have been associated with a better prognosis.

Mini-review of invasive aspergillosis in ESKD and HD patients

Diagnosis

Aspergillosis develops in multiple organs, including the lung, brain, paranasal sinuses, kidney, and skin [16, 17]. The diagnosis of IA is described by The European Organization for Research and Treatment of Cancer and the Mycoses Study Group Education and Research Consortium [18], in which the criteria for the probable or proven IA are discussed. While the proven IA needs a histopathologic examination of biopsy samples, the criteria for probable IA include host factors like prolonged use of corticosteroids, clinical features like presence of cavity in the CT, and mycological evidence like the presence of galactomannan antigen for *Aspergillus*. The criteria for the probable IA are applicable for immunocompromised hosts only, while those for the proven IA are for any patient. Clinically, the most commonly occurring site of positive *Aspergillus* infection in the ICU is reported to be lung, and the proven IA is common in those with

a positive culture of *Aspergillus* in the abdominal, brain, and endovascular samples [5].

Past pulmonary infections and Aspergillus infection

The relationship between tuberculosis and NTM lung disease history and aspergillus infection has been well documented. The growth of *Aspergillus* occurs in residual cavity lesions caused by tuberculosis infection, and the relationship between post-tuberculosis lung disease and chronic pulmonary aspergillosis is confirmed in the survey analysis reported in the 1970s [19]. Patients with NTM lung disease are also shown to have a higher prevalence of *Aspergillus* infection [14], and they argued that the identification of *Aspergillus*-related lung disease in patients with NTM lung disease is crucial.

Symptoms

The symptoms vary based on the different types of aspergillosis. The symptoms of the most commonly occurring pulmonary infection are cough, rales, and pleuritic chest pain, which are reported to be noted early in the clinical course [20]. In critically ill patients, the clinical signs are reported to be worsening lung function, dyspnea, and refractory fever [5]. For aspergillosis with central nervous system involvement like in this case, the symptoms of changes in mental status, hemiparesis, and seizures are reported to be most common [21]. For invasive sinusitis, the early symptoms are reported to include dark-colored nasal septal or palatal ulcers, headache, nasal crusting, and epistaxis [22].

Treatment

The key treatments of IA include the early diagnosis and early initiation of antifungal therapy, the surgical resection for the patient with some clinical manifestations, and the reduction of immunosuppressive therapy [23]. For invasive pulmonary aspergillosis, the primary choice of antifungal medication is VRCZ, and the alternative choice is L-AMB. Additionally, echinocandins in combination may be considered [23]. It is recommended to treat the patients on dialysis with the oral VRCZ rather than intravenous administration for the consideration of the possible adverse events by its vehicle sulfobutylether-beta-cyclodextrin [24]. L-AMB is reported to be used for patients with HD without dosing or interval adjustments [25]. It is reported that no dosage adjustment is required for echinocandins in continuous RRT [26].

Prognosis

IA is associated with high mortality. One of the largest studies reported a twelve-week overall and disease-specific survival rate of 52.2% and 59.8%, respectively [2]. In critically ill patients, one study reported a twelve-week mortality rate of 72%. In kidney-transplant recipients, the twelve-week post-infection survival rate was reported to be 94%, and the risk of graft loss was higher after IA [27]. The specific clinical study investigating prognostic information of IA in ESKD or HD patients is currently not available in the literature, and we summarize the case reports of these patients available in the literature.

Case reports of IA in ESKD patients

Several previous studies reported IA in ESKD patients. We listed the reported cases in Table 2. A study reported a 15-year-old male receiving continuous ambulatory PD who developed intracranial hemorrhagic abscesses and subsequent death despite the treatment of L-AMB [28], and another study reported a 55-year-old female who developed rapid *Aspergillus* infection in the lung and resulted in subsequent death with the treatment of L-AMB after the placement of a PD catheter [29]. Additionally, a case series reported a 34-year-old male with ESKD, past pulmonary tuberculosis, and type 1 DM on HD who developed invasive pulmonary aspergillosis on the tuberculous cavity and laryngeal tissue mass, and subsequent death with the treatment of VRCZ, and a 54-year-old female with type 2 DM and ESKD on HD who developed invasive aspergillus rhino-sinusitis [15]. She was treated with antifungals and surgical drainage, and the outcome of the patient was not reported. A case of IA in ESKD on HD patient presenting with submandibular swelling was recently reported. The patient was treated with caspofungin and subsequent oral VRCZ and remained stable [30].

Conclusion

In conclusion, we present a detailed clinical course including used antifungals and the dosage of an ESKD patient with the suspected NTM lung disease who suffered from IA and resulted in initiating maintenance HD. We summarized the IA cases in ESKD patients with detailed information as there is no study investigating prognosis in ESKD population.

As immunosuppression like steroid therapy and impaired immunity caused by chronic kidney disease or ESKD can lead to both AFBs and *Aspergillus* infections [31, 32], the intensity of immunosuppressive therapy on ESKD patients, especially with past AFB infection, should be determined carefully. Further, the careful monitoring of the patient's symptoms, early definitive diagnosis of IA as well as rapid initiation of treatment, is supposed to be critical for ESKD patients on immunosuppressive treatment.

Table 2 The summary of cases of invasive aspergillosis in ESKD patients

Age	Gender	Kidney diseases	Pulmonary complications	Other comorbidities	Pathogen	Site/clinical manifestation	Treatment (start timing if described)	Dosage of antifungals	Outcome	Reference
66	Male	ESKD from FSGS	Past NTM disease	None	*Aspergillus* spp.	Lung, intracranial abscess	Described in the manuscript	Described in the manuscript	Discharge (day 380) and death one month later	This case
15	Male	CAPD, cause unknown (global sclerosis)	Not described	Not described	*Aspergillus* spp.	Intracranial hemorrhagic abscesses	L-AMB	Not described	Death (day 13)	[28]
55	Female	ESKD starting PD, cause reflux nephropathy	A smoker of 20 cigarettes per day	Not described	*Aspergillus fumigatus*	Lung	L-AMB (from day 3)	Not described	Death (day 3)	[29]
34	Male	HD	Past pulmonary tuberculosis	Type 1 DM	Not described	Lung (in a tuberculous cavity), laryngeal tissue mass	VRCZ	6 mg/kg every12 hours	Death (Two days later)	[15]
54	Female	HD	Not described	Type 2 DM	Not described	Rhino-sinusitis	The antifungals with surgical drainage	Not described	Unknown	[15]
49	Male	HD	Not described	long-standing hypertension	Not described	submandibular swelling and lung	Caspofungin, oral VRCZ	Not described	Stable	[30]

CAPD continuous ambulatory peritoneal dialysis; *DM* diabetes mellitus; *ESKD* end-stage kidney disease; *FSGS* focal segmental glomerulosclerosis; *HD* hemodialysis; *L-AMB* liposomal amphotericin B; *NTM* nontuberculous mycobacterial; *PD* peritoneal dialysis; and *VRCZ* voriconazole

Abbreviations
AMPH-B: Amphotericin B; CT: Computed tomography; CyA: Cyclosporine; DM: Diabetes mellitus; ESKD: End-stage kidney disease; FSGS: Focal segmental glomerulosclerosis; HD: Hemodialysis; IA: Invasive aspergillosis; ICU: Intensive care unit; L-AMB: Liposomal Amphotericin B; MCFG: Micafungin; MEPM: Meropenem; MIZ: Mizoribine; MRI: Magnetic resonance imaging; PD: Peritoneal dialysis; PSL: Prednisolone; TAZ/PIPC: Tazobactam/piperacillin; VRCZ: Voriconazole.

Acknowledgements
We gratefully acknowledge Y. Setsuda and M. Ozone in the Department of Nephrology, Graduate School of Medicine, Kyoto University, for secretarial assistance.

Author contributions
NS and HY wrote the manuscript. HY, MI, AI, TM, and MY treated the patient, discussed the content of the manuscript, and revised the manuscript. All the authors confirmed the content of the manuscript.

Funding
There was no funding relevant to the study.

Availability of data and materials
All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
The written informed consent was obtained from the patient.

Consent for publication
The written informed consent was obtained from the patient.

Competing interests
H. Yokoi is the associate editor of Renal Replacement Therapy. The other authors declare that they have no competing interests.

Author details
[1]Department of Nephrology, Graduate School of Medicine, Kyoto University, 54 Shogoin Kawahara-Cho, Sakyo-Ku, Kyoto 606-8507, Japan. [2]Department of Nephrology, Japanese Red Cross Wakayama Medical Center, Wakayama, Japan.

Received: 15 March 2022 Accepted: 18 December 2022
Published online: 26 December 2022

References

1. Hori A, Kami M, Kishi Y, Machida U, Matsumura T, Kashima T. Clinical significance of extra-pulmonary involvement of invasive aspergillosis: a retrospective autopsy-based study of 107 patients. J Hosp Infect. 2002;50:175–82.
2. Nivoix Y, Velten M, Letscher-Bru V, Moghaddam A, Natarajan-Amé S, Fohrer C, et al. Factors associated with overall and attributable mortality in invasive aspergillosis. Clin Infect Dis. 2008;47:1176–84.
3. Baddley JW. Clinical risk factors for invasive aspergillosis. Med Mycol. 2011;49(Suppl 1):S7-12.
4. Sun K-S, Tsai C-F, Chen SC-C, Huang W-C. Clinical outcome and prognostic factors associated with invasive pulmonary aspergillosis: an 11-year follow-up report from Taiwan. PLoS ONE. 2017;12:e0186422.
5. Taccone FS, Van den Abeele A-M, Bulpa P, Misset B, Meersseman W, Cardoso T, et al. Epidemiology of invasive aspergillosis in critically ill patients: clinical presentation, underlying conditions, and outcomes. Crit Care. 2015;19:7.
6. Dotis J, Kondou A, Koukloumperi E, Karava V, Papadopoulou A, Gkogka C, et al. Aspergillus peritonitis in peritoneal dialysis patients: a systematic review. J Mycol Med. 2020;30: 101037.
7. Kato S, Chmielewski M, Honda H, Pecoits-Filho R, Matsuo S, Yuzawa Y, et al. Aspects of immune dysfunction in end-stage renal disease. Clin J Am Soc Nephrol. 2008;3:1526–33.
8. Li PK-T, Chow KM. Infectious complications in dialysis–epidemiology and outcomes. Nat Rev Nephrol. 2011;8:77–88.
9. Mattern WD, Hak LJ, Lamanna RW, Teasley KM, Laffell MS. Malnutrition, altered immune function, and the risk of infection in maintenance hemodialysis patients. Am J Kidney Dis. 1982;1:206–18.
10. Ishida JH, Johansen KL. Iron and infection in hemodialysis patients. Semin Dial. 2014;27:26–36.
11. Lionakis MS, Kontoyiannis DP. Glucocorticoids and invasive fungal infections. Lancet. 2003;362:1828–38.
12. Fardet L, Petersen I, Nazareth I. Common infections in patients prescribed systemic glucocorticoids in primary care: a population-based cohort study. PLoS Med. 2016;13: e1002024.
13. Elluru SR, Kaveri SV, Bayry J. The protective role of immunoglobulins in fungal infections and inflammation. Semin Immunopathol. 2015;37:187–97.
14. Kunst H, Wickremasinghe M, Wells A, Wilson R. Nontuberculous mycobacterial disease and Aspergillus-related lung disease in bronchiectasis. Eur Respir J. 2006;28:352–7.
15. Jabrane M, Skandour D, Rochdi Y, Nouri H, Aderdour L, Raji A, et al. Severe forms of aspergillosis in patients on hemodialysis. Saudi J Kidney Dis Transpl. 2016;27:174–6.
16. Bernardeschi C, Foulet F, Ingen-Housz-Oro S, Ortonne N, Sitbon K, Quereux G, et al. Cutaneous invasive aspergillosis: retrospective multicenter study of the french invasive-aspergillosis registry and literature review. Medicine. 2015;94: e1018.
17. Haq JA, Khan MA, Afroze N, Haq T. Localized primary renal aspergillosis in a diabetic patient following lithotripsy–a case report. BMC Infect Dis. 2007;7:58.
18. Donnelly JP, Chen SC, Kauffman CA, Steinbach WJ, Baddley JW, Verweij PE, et al. Revision and update of the consensus definitions of invasive fungal disease from the european organization for research and treatment of cancer and the mycoses study group education and research consortium. Clin Infect Dis. 2020;71:1367–76.
19. British Thoracic and Tuberculosis Association. Aspergilloma and residual tuberculous cavities--the results of a resurvey. Tubercle. 1970;51:227–45.
20. Schwartz S, Thiel E. Clinical presentation of invasive aspergillosis. Mycoses. 1997;40(Suppl 2):21–4.
21. Ruhnke M, Kofla G, Otto K, Schwartz S. CNS aspergillosis: recognition, diagnosis and management. CNS Drugs. 2007;21:659–76.
22. Deshazo RD. Syndromes of invasive fungal sinusitis. Med Mycol. 2009;47(Suppl 1):S309–14.
23. Patterson TF, Thompson GR 3rd, Denning DW, Fishman JA, Hadley S, Herbrecht R, et al. Practice guidelines for the diagnosis and management of aspergillosis: 2016 update by the infectious diseases society of America. Clin Infect Dis. 2016;63:e1-60.
24. Hamada Y, Tokimatsu I, Mikamo H, Kimura M, Seki M, Takakura S, et al. Practice guidelines for therapeutic drug monitoring of voriconazole: a consensus review of the Japanese society of chemotherapy and the Japanese society of therapeutic drug monitoring. J Infect Chemother. 2013;19:381–92.
25. Obata Y, Takazono T, Tashiro M, Ota Y, Wakamura T, Takahashi A, et al. The clinical usage of liposomal amphotericin B in patients receiving renal replacement therapy in Japan: a nationwide observational study. Clin Exp Nephrol. 2021;25:279–87.
26. González de Molina F, Martínez-Alberici M, Ferrer R. Treatment with echinocandins during continuous renal replacement therapy. Crit Care. 2014;18:218.
27. Desbois A-C, Poiree S, Snanoudj R, Bougnoux M-E, Sberro-Soussan R, Lanternier F, et al. Prognosis of invasive Aspergillosis in kidney transplant recipients: a case-control study. Transplant Direct. 2016;2: e90.
28. Cicek N, Yildiz N, Kadayifci EK, Gokce I, Alpay H. Invasive aspergillosis in a patient with end stage renal disease. Med Mycol Case Rep. 2017;18:12–4.
29. Sahlén AO, Suvarna SK, Wilkie ME. A case of invasive pulmonary aspergillosis in renal failure. Nephrol Dial Transplant. 2004;19:2687.
30. Iqbal P, Dakhlia S, Hassen SS, Mahdi S. An unusual presentation of invasive aspergillosis with submandibular swelling in a 49-year-old man with end-stage renal disease: a case report. Respirol Case Rep. 2022;10:e0905.

31. Romanowski K, Clark EG, Levin A, Cook VJ, Johnston JC. Tuberculosis and chronic kidney disease: an emerging global syndemic. Kidney Int. 2016;90:34–40.
32. Cahuayme-Zuniga LJ, Brust KB. Mycobacterial infections in patients with chronic kidney disease and kidney transplantation. Adv Chronic Kidney Dis. 2019;26:35–40.

Publisher's Note