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CASE REPORT

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Antibiotic-driven pathogen replacement events in a kidney transplant recipient with ADPKD: a case report

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Abstract

Background Retaining the native bilateral kidneys after transplantation is a common treatment for patients with end-stage autosomal dominant polycystic kidney disease. However, this strategy poses the risks of potential complications from polycystic kidney infection. The efficiency of antibiotic therapy and the optimal time for native nephrectomy in managing these infections remain uncertain.

Case presentation We report a case of a kidney transplant recipient with retained bilateral polycystic kidneys who experienced recurrent cyst and bloodstream infections, accompanied by antibiotic-driven pathogen replacement. After multiple failed attempts at antibiotic therapy, the patient subsequently underwent unilateral polycystic kidney resection. Subsequently, a new infection episode occurred, leading to the other native nephrectomy. Cystic tissue and fluid samples were collected from both shallow and deep layers of the polycystic kidneys, along with peripheral blood and urine samples. These samples were analyzed using microbial culture, metagenome sequencing, and digital polymerase chain reaction to identify infectious pathogens. Pathogen replacement occurred across different infection episodes, with the dominant bacterial species being *Escherichia coli*, *Klebsiella aerogenes*, and *Enterococcus faecium*, in succession.

Conclusions This case highlights the replacement of dominant pathogens under antibiotic selection pressure in polycystic kidney infections, primarily involving gram-negative bacilli. When initial and subsequent antibiotic therapy fail, re-evaluation of the cyst infection definition is necessary, and preemptive native nephrectomy should be considered.

Keywords Cyst infection, Pathogen replacement, Antibiotic therapy, Kidney transplantation, Autosomal dominant polycystic kidney disease

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Introduction

Autosomal dominant polycystic kidney disease (ADPKD) accounts for up to 10% of patients undergoing kidney transplantation (KT) [1, 2]. Most transplant centers proceed with KT without native nephrectomy due to the typically asymptomatic nature of cysts and the potential complications associated with native nephrectomy [3]. Although the volume of native kidneys is expected to decrease by 30-40% within 1 year following KT, various renal and extrarenal complications have been reported in KT recipients with ADPKD [4]. The most common reasons for impaired survival of patients and grafts are sepsis complications directly related to polycystic kidneys [5, 6]. Cyst infection (CyI) in ADPKD are often recurrent and refractory due to limited antibiotic penetration into cysts and fluid [7]. Repeated antibiotic therapy may lead to antibiotic-driven pathogenl replacement and the emergence of antibiotic-resistant strains. The optimal timing for post-KT native nephrectomy when antibiotic therapy fails remains a subject of debate [8, 9].

Herein, we present a case that illustrates the time course of antibiotic-driven predominant pathogen replacement and emphasize the importance of post-KT native nephrectomy evaluation in KT recipients with ADPKD, particularly in the context of recurrent CyI.

Case presentation

A 48-year-old male with end-stage ADPKD underwent KT in 2016, receiving a right-sided kidney from a donor, after cardiac death, without prior native nephrectomy. His immunosuppressive regimen was "tacrolimus + mycophenolate mofetil + prednisolone.". Ultrasound examination in 2018 showed that the volumes of native polycystic kidneys were 11.6 cm × 4.3 cm × 4.8 cm (left kidney) and 11.9 cm × 4.9 cm × 5.1 cm (right kidney). His postoperative serum creatinine (Scr) level stabilized at 90–105 μmol/L.

In April 2023, the patient began having frequent hospital admissions owing to episodes of fever (peak temperature of 38.5°C, duration of 48 h) associated with bacteremia, along with chest pain. Laboratory test results were as follows: Scr 106 μmol/L, C-reactive protein (CRP) 206.9 mg/L, procalcitonin (PoCT) 1.2 ng/L, 1 + protein in urine, and no hematuria. Chest radiography and computed tomography were performed to rule out respiratory infections, and no significant abnormalities were detected. The computed tomography report revealed chronic inflammation in the lower lobes of both lungs and the left upper lobe's lingual segment, and bilateral pleural thickening. A small nodule in the posterior segment of the left upper lobe was also noted, which was considered an inflammatory nodule (Lung-RADS category 2). These findings were not significantly different compared to previous annual follow-up scans, suggesting no acute respiratory infection. Ultrasound examination showed that the volume of the left polycystic kidney increased to 14.2 cm × 7.7 cm × 7.9 cm, while the right one could not be measured. No abdominal or lumbar pain was observed. The results from midstream urine, peripheral blood microbial cultures, and metagenomic next-generation sequencing (mNGS) indicated an *Escherichia coli* (*E. coli*) infection, suggesting hematogenous dissemination caused by a urinary tract infection. There was insufficient evidence to diagnose a cyst infection (CyI). The co-occurrence of urinary tract and bloodstream *E. coli* infections, along with his compromised immune status, prompted the prescription of meropenem as a broad-spectrum empirical therapy. Thus, initial antibiotic therapy was meropenem (1 g, VD, every 12 h) for a duration of 10 days and then amoxicillin (0.375 g, oral, TID) for 4 days. Considering that recurrent infections may be driven by excessive immune suppression, mycophenolate mofetil was switched to mizoribine. The patient was discharged after relief from infection symptoms.

In May 2023, the patient presented with recurrent fever (peak temperature of 38.5 °C, duration of 48 h), and mild discomfort below the rib cage that improved with rest. Subsequently, he was readmitted with sepsis caused by *E. coli*, with a Sequential Organ Failure Assessment score of 4. Laboratory test results were as follows: Scr 158 μmol/L, CRP 313.8 mg/L, PoCT 31.8 ng/L, and red blood cell count in urine (U_RBC) 16.6/μL. Abdominal computed tomography did not indicate any changes in the cyst compared to before. Meropenem as the empirical therapy for 4 days exhibited poor efficacy. Further testing identified a resistant strain of *E. coli*, prompting a modification of the antibiotic therapy: an addition of moxifloxacin (0.4 g, VD, QD) for 9 days. The infection symptoms were eventually relieved.

In November 2023, the patient was once again admitted to the hospital due to recurrent fever (peak temperature of 38.5°C, duration of 72 h), accompanied by right lower chest pain, nausea, painful urination, and shortness of breath, with no abdominal or lumbar pain. Laboratory test results were as follows: Scr 87 μmol/L, CRP 286.7 mg/L, PoCT 13.6 ng/L, 2 + protein in urine, white blood cell count in urine (U_WBC) 71.5/μL, and U_RBC 9728.6/μL, indicating suspected cyst hemorrhage. Chest radiography and computed tomography scans did not indicate a respiratory infection. Peripheral blood microbial cultures indicated infection with gram-negative bacilli. Considering the patient's history of resistant *E. coli* infection, empirical antibiotic therapy with meropenem (1 g, IV, every 12 h) and immunoglobulin therapy for balancing the risk of infection and rejection were initiated; however, this proved to be ineffective. Furthermore, peripheral blood mNGS identified *Klebsiella aerogenes*

(*K. aerogenes*) as the dominant pathogen with 11,319 sequences, leading to a diagnosis of *K. aerogenes* sepsis. Urine mNGS detected BK polyomavirus (30 sequences) and human herpesvirus 6B (17 sequences). The patient also developed diarrhea, and fecal examination revealed the presence of fungal spores. Therefore, the antibiotic therapy was modified to ceftazidime/avibactam (2.5 g IV, every 8 h), vancomycin (0.5 g, oral, TID), and oseltamivir added as antiviral therapy (Fig. 1A). Additionally, mizoribine was discontinued, and tacrolimus was reduced to lower immunosuppressive intensity. Despite these adjustments, the patient continued to experience recurrent fever. The antibiotic therapy was further adjusted to meropenem combined with sulfamethoxazole/trimethoprim (0.48 g, oral, BID), resulting in partial resolution of infection symptoms, although the fever persisted intermittently. Following specialist consultation, the infection was determined to be caused by multidrug-resistant *K. aerogenes*. The treatment was escalated to a combination of aztreonam (2.5 g, IV, every 8 h) and ceftazidime/avibactam (2.5 g IV, every 8 h). After three days of combination therapy, the patient's temperature normalized. Antibiotic therapy was maintained for 14 days, during which the CRP decreased from 286.67 mg/L to 30.85 mg/L. Repeat mNGS revealed 42 *K. aerogenes* sequences. Taking into account the patient's significant polycystic kidney disease and the recurrent bloodstream infections that likely originated from intrarenal CyI and cyst hemorrhage, a planned nephrectomy was proposed. The surgery aimed to prevent further extrarenal complications, persistent infection, and potential pathogen evolution under antibiotic pressure, which could lead to the emergence of superbugs.

In December 2023, the patient was readmitted to the hospital due to fever (peak temperature of 39.0°C, duration of 48 h) accompanied by cough and mild right lower chest pain. Laboratory test results were as follows: Scr 121 μmol/L, CRP 230.48 mg/L, and PoCT 1.3 ng/L. The other laboratory results are shown in Fig. 1B. The transplanted kidney was enlarged, and the increased creatinine level raised the suspicion of acute kidney rejection. Methylprednisolone (40 mg, IV, QD) was administered for anti-rejection therapy for three days, and Scr levels decreased to 92 μmol/L. Peripheral blood mNGS identified *K. aerogenes* with 4,825 sequences. The patient was treated with ceftazidime/avibactam (2.5 g, IV, every 8 h) for the infection, but the response was poor, with persistent recurrent fever. The antimicrobial regimen was adjusted to meropenem plus moxifloxacin, leading to the resolution of fever and cough symptoms. Anti-infection therapy was continued for 14 days. To address the persistent impact of a potential polycystic kidney infection as the primary source, a da Vinci robot-assisted laparoscopic right nephrectomy of the polycystic kidney was performed in January 2024 after the infection had stabilized (Fig. 1C). Both shallow and deep cyst tissue of polycystic kidney, as well as cyst fluid, were sent for mNGS and digital polymerase chain reaction (PCR). Sequencing results indicated that the *K. aerogenes* found in the cyst tissue and fluid were consistent with those detected in the peripheral blood, further confirming the association between cystic kidney and bloodstream infections (Table 1). Postoperatively, the patient recovered well, and laboratory tests showed a consistent decline in inflammation-related markers.

In April 2024, three months after the right nephrectomy, the patient was readmitted with fever, cough, chest tightness, and shortness of breath for three days, with symptoms resembling those of the previous episodes. Laboratory test results were as follows: Scr 109 μmol/L, CRP 194.5 mg/L, PoCT 2.0 ng/L, 1 + protein in urine, and U_RBC 22.2/μL. The computed tomography examination results are basically the same as before. Peripheral blood cultures indicated infection by *Staphylococcus aureus* (*S. aureus*) infection, and urine mNGS identified *Enterococcus faecium* (*E. faecium*) as the dominant pathogen, with 1,910 sequences, signifying a shift in the patient's infection profile. Meropenem (1 g, IV, every 12 h) was administered to prevent the recurrence of persistent bloodstream infection. After nine days of treatment, left nephrectomy was performed for the remaining polycystic kidney (Fig. 1D). Both shallow and deep cyst tissue of polycystic kidney, as well as cyst fluid, were sent for mNGS and digital PCR (Table 1). Sequencing results indicated the same *K. aerogenes* in the shallow cyst tissueand fluid. The patient had an uneventful postoperative recovery, with laboratory tests showing a consistent decline in inflammatary markers and was subsequently discharged.

Discussion and conclusions

Although CyI remains one of the most prevalent complications in ADPKD, its diagnosis and management are challenging. In KT recipients, the incidence of CyI is 1.6 per 100 person-years and is associated with an elevated risk of graft loss [10, 11]. For patients with retained bilateral polycystic kidneys, the management of postoperative immunosuppression and infection prevention is particularly complex, and no consensus exists on the standard interventions. Effective antibiotic strategies to mitigate pathogen replacement and evolution, as well as determining the optimal time for native nephrectomy, necessitates further clinical research and evidence to establish comprehensive guidelines.

Diffusion of antibiotics into renal cysts depends more on transepithelial transport than on glomerular filtration [12]. Nevertheless, the properties of cyst walls limit or delay the diffusion of most antibacterial drugs, resulting

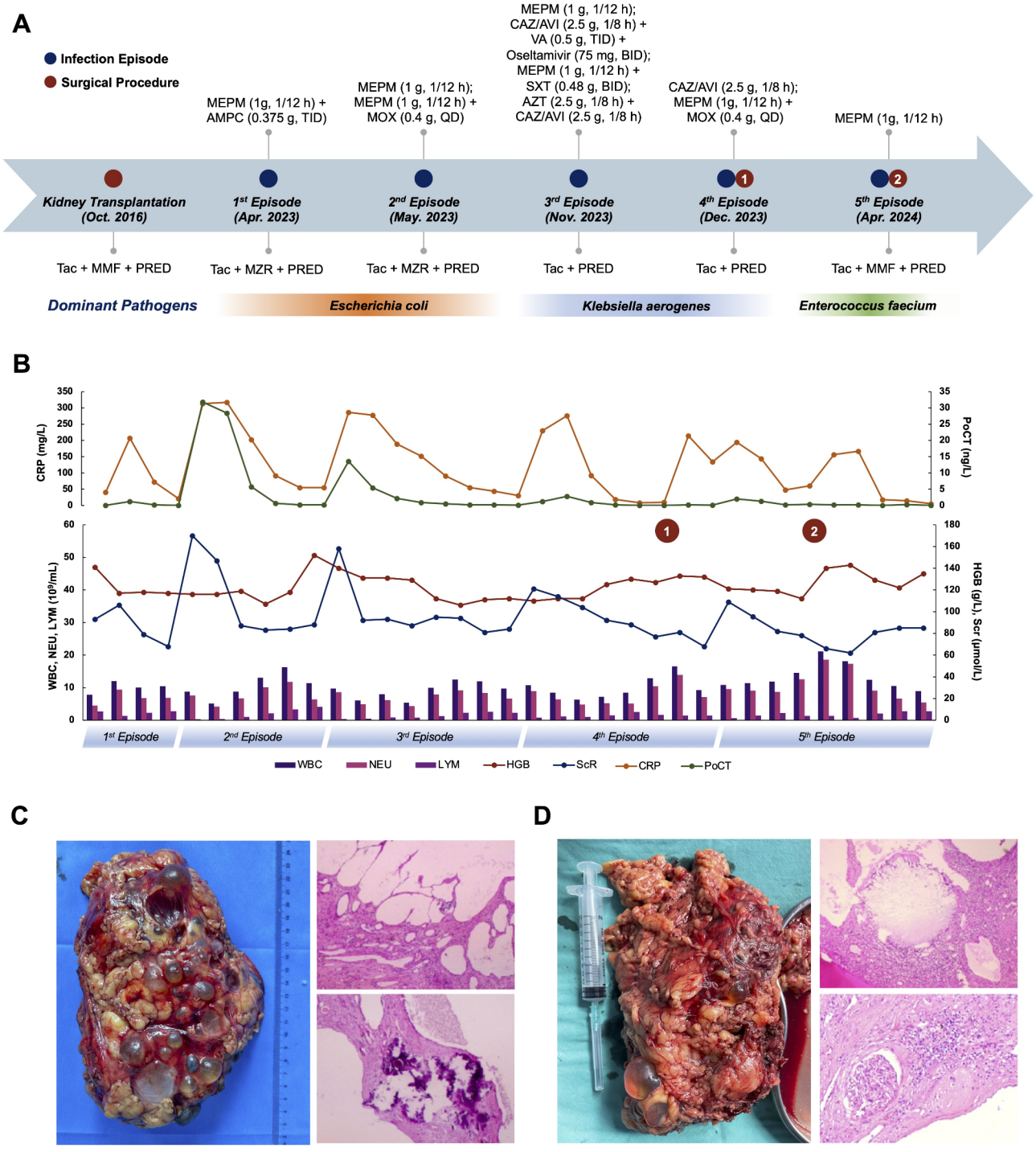

Fig. 1 Clinical information of the patient from recurrent infection to bilateral native nephrectomy. (**A**) The antibiotic therapy, immunosuppressive regimen, and predominant pathogen replacement in peripheral blood during the five infection episodes. (**B**) The clinical course across the five episodes of infection-related hospitalizations. (**C**, **D**) Gross photograph and pathology of the right (**C**) and left (**D**) native nephrectomy specimen, indicating focal calcification and chronic inflammation in polycystic kidney. MEPM, meropenem; AMPC, amoxicillin; Mox, moxifloxacin; CAZ/AVI, ceftazidime/avibactam; VA, vancomycin; SXT, sulfamethoxazole/trimethoprim; AZT, aztreonam; Tac, tacrolimus; MMF, mycophenolate mofetil; PRED, prednisolone; MZR, mizoribine; CRP, C-reactive protein; PoCT, procalcitonin; WBC, white blood cell; NEU, neutrophils; LYM, lymphocyte; HGB, hemoglobin; Scr, serum creatinine

Table 1 mNGS and dPCR results from clinical specimens in two episodes of native nephrectomy

Specimens	mNGS	dPCR	
	Pathogen (sequences)	Klebsiella aerogenes	Aspergillus fumigatus
Right nephrectomy			
Urine	*Aspergillus fumigatus* (1)	positive	negative
Peripheral blood	negative	negative	negative
Shallow cyst fluid	*Torque teno virus* (1296) *Human gamma herpesvirus 4* (36)	positive	positive
Shallow cyst tissue	*Aspergillus fumigatus* (1)	positive	positive
Deep cyst fluid	*Torque teno virus* (206)	negative	negative
Deep cyst tissue	*Human beta herpesvirus 6B* (6)	negative	negative
Left nephrectomy			
Urine	*Enterococcus faecium* (1910) *Corynebacterium pseudogenitalium* (15) *Corynebacterium aurimucosum* (14) *Aspergillus fumigatus* (1) *Human polyomavirus 1* (3342)	negative	positive
Peripheral blood	*Torque teno virus* (9)	negative	negative
Shallow cyst fluid	negative	positive	positive
Shallow cyst tissue	*Torque teno virus* (23)	positive	positive
Deep cyst fluid	*Human beta herpesvirus 5* (1395) *Torque teno virus* (6)	negative	negative
Deep cyst tissue	negative	negative	negative

* mNGS, metagenomic next-generation sequencing; dPCR, digital polymerase chain reaction

in a high rate of treatment failures and recurrences [8]. Therefore, antibiotic therapy for CyI often requires higher doses and longer durations than those used for routine infections [6, 13]. A 28-day course may be necessary to achieve satisfactory outcomes [8]. This prolonged, ineffective antibiotic treatment approach provided ample time for pathogens to evolve or replace each other, further increasing the difficulty, recurrence, and incidence of new episodes of CyI. Meanwhile, under sustained high-intensity antibiotic pressure, the host's microbiome undergoes alterations, resulting in pathogen replacement in the extrarenal system. In our case, the predominant pathogen in bloodstream infections shifted from *E. coli* to *K. aerogenes*, and subsequently to S. aureus, while in urinary tract infections, *E. coli* was replaced by *K. aerogenes* and later by *E. faecium*. The patient consequently experienced frequent antibiotic exposure, immunosuppressive regimen adjustments, and multiple hospitalizations, ultimately leading to bilateral nephrectomy.

Moreover, when the immunosuppressive regimen was adjusted early to effectively manage recurrent infections, an increase in the transplanted kidney size and Scr levels was observed in this case. These changes were promptly addressed with steroid pulse therapy, which successfully reversed the progression of potential rejection episodes. Therefore, caution is warranted when reducing the intensity of immunosuppression to control infection, as this may increase the risk of graft rejection.

Another challenge with CyI studies lies in definite diagnosis without invasive procedure. CyIs are classified into three definitions based on the method of diagnosis: definite (cyst puncture), probable (imaging variables), and possible (clinical variables) [8, 14]. Currently, cyst puncture is rarely performed, and the diagnostic accuracy of conventional computed tomography imaging is limited [15]. Clinical evidence requires ruling out extrarenal factors and cyst hemorrhage as causes of abdominal or lumbar pain [16]. In cases like the one described, where none of these criteria can be fully met, the diagnosis of CyI can be delayed. Recent studies have shown that positron emission tomography (PET) imaging with intravenous 18 F-fluorodeoxyglucose (FDG), combined with computed tomography imaging, is superior to conventional radiological techniques for identifying and localizing renal CyI [17]. Therefore, when there is a high clinical suspicion of CyI, FDG-PET/CT should be preferred over computed tomography for evaluating polycystic kidneys.

Although percutaneous intervention, such as cyst drainage, is a potential option for localized infections, it was not pursued in this case due to systemic involvement, including bacteremia and urinary tract infection [6]. Percutaneous drainage alone does not address pathogen replacement, a significant concern in immunocompromised transplant recipients. Given the failure of antibiotic therapy and the risk of sepsis or graft damage, we opted for native nephrectomy to effectively remove the infection source and prevent further complications. Thus, while percutaneous procedures may be useful in some cases, surgical intervention remains essential for complex or systemic infections.

The need, indications, timing, and approach for native nephrectomy in patients with ADPKD remain controversial. Recent studies have found that patients with ADPKD are at a higher risk of urinary tract infections and urosepsis compared to non-ADPKD recipients, with this increased risk being associated with recipient age and steroid treatment [18]. Although no differences in graft survival have been observed between the two groups, personalized steroid-free immunosuppression and unilateral or bilateral nephrectomy may help reduce the risk of recurrent and severe infections in these patients [18]. Moreover, evidence suggests no significant difference in outcomes whether native nephrectomy is performed before or after KT [2, 19, 20]. Native nephrectomy after transplantation is generally indicated for infectious complications, elevated intra-abdominal pressure, or suspected malignancy, with cyst infection being the primary indication [21]. CyI is typically attributed to hematogenous spread or retrograde urinary tract infection [6]; however, this relationship can be bidirectional. Hematogenous or retrograde infection can colonize cysts, leading to CyI, cyst rupture, and exacerbation of bloodstream infections. Thus, when hematuria occurs, it is crucial to differentiate cyst hemorrhage from other causes and monitor for bacterial presence to prevent sepsis.

In conclusion, recurrent and refractory CyI imposes significant antibiotic selection pressure in patients with ADPKD. The inability of antibiotic therapy to fully eradicate infection in polycystic kidneys leads to ongoing pathogen replacement. Therefore, in KT recipients with ADPKD, especially those with complex infection histories, the risks of retaining native kidneys should be carefully evaluated. Close monitoring of pathogen dynamics post-transplant is essential, and early consideration of native nephrectomy should be made when warranted. Additionally, antibiotic selection should be carefully guided by pathogen sensitivity to avoid unnecessary broad-spectrum use, which can drive resistance in immunocompromised KT recipients. Advanced technologies such as mNGS and digital PCR enhance precise identification and tracking of infection sources, providing valuable insights for clinical management.

Abbreviations

ADPKD	Autosomal dominant polycystic kidney disease
KT	Kidney transplantation
Scr	Serum creatinine
CRP	C-reactive protein
PoCT	Procalcitonin
mNGS	Metagenomic next-generation sequencing
CyI	Cyst infection
U_RBC	Red blood cell count in urine
U_WBC	White blood cell count in urine

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Author contributions
RFX, YM and ZYY designed and directed the study. ZYY wrote the manuscript. JX and BY contributed new reagents and analytic tools. ZYY, WLZ and JLH collected the data. YCW and RFX revised the manuscript. All authorship read and approved the final version.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The study was approved by the ethical committee of Nanfang Hospital, Southern Medical University (NFEC-2019-190). Written informed consent to participate was provided by the patient involved in our study.

Consent for publication
Written informed consent for publication of the clinical details and clinical images was obtained from the patient involved in our study.

Competing interests
The authors declare no competing interests.

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