

Epidemiology, Antimicrobial Resistance, and Risk Factors for Early Gram-Negative Bacterial Infections after Deceased-Donor Kidney Transplantation: A 10-Year Single-Center Analysis

Qian-yong ZHAO

Huazhong University of Science and Technology

Bo ZHANG

Huazhong University of Science and Technology

Bo YANG

Huazhong University of Science and Technology

Yuan-yuan ZHAO

Huazhong University of Science and Technology

Zhi-shui CHEN

Huazhong University of Science and Technology

Lai WEI

Huazhong University of Science and Technology

Dong CHEN

dchen0369@163.com

Huazhong University of Science and Technology

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Abstract

Objective: To analyze epidemiology, multidrug resistance (MDR), and risk factors for Gram-negative bacterial (GNB) infections within 30 days post-kidney transplantation.

Methods: Retrospective analysis of 2,875 DCD kidney recipients (2015–2024); 102 with early GNB infection were included. Pathogen distribution, antimicrobial susceptibility, and risk factors for MDR were assessed.

Results: A total of 129 GNB isolates were obtained, with an infection incidence of 3.5% (102/2875). *Klebsiella pneumoniae* (39.5%) and *Acinetobacter baumannii* (20.9%) were the predominant pathogens. The surgical site was the most common infection location (62.8%). MDR isolates accounted for 48.8% (63/129), exhibiting a resistance rate of 92.1% to carbapenems while maintaining relatively high susceptibility to tigecycline (resistance rate 9.5%). Multivariate analysis identified a lymphocyte count $\leq 0.3 \times 10^9/L$ (OR=3.328, P=0.019) and a serum albumin level ≤ 35 g/L (OR=3.385, P=0.029) as independent risk factors for MDR infection. The detection rate of difficult-to-treat resistant pathogens has shown a declining trend since 2019.

Conclusion: Early post-transplant GNB infections at our center are primarily caused by *Klebsiella pneumoniae* and *Acinetobacter baumannii*, with the surgical site being the most frequent location. Profound postoperative lymphopenia and hypoalbuminemia are independent early warning indicators for MDR infection. Monitoring these parameters aids in the early identification of high-risk patients and guides empirical anti-infective therapy. The recent decline in the detection of difficult-to-treat resistant pathogens suggests that current comprehensive prevention and control measures have demonstrated a degree of effectiveness.

1 Introduction

Infection remains a major complication and a leading cause of mortality following kidney transplantation^{1,2}. This is primarily due to the use of immunosuppressive agents post-transplantation, which reduces the body's immune responses, weakens defense mechanisms, and increases susceptibility. Furthermore, urological surgical procedures, anatomical alterations, and indwelling urinary catheterization further elevate the risk of infection³. In the early post-transplant period, the most common sources of infection include perioperative donor-derived infections, nosocomial (hospital-acquired) infections, and the reactivation of latent infections⁴. However, significant variations exist in epidemiological characteristics and antimicrobial resistance profiles across different geographic regions and transplant centers^{5,6}. Therefore, to formulate effective empirical treatment protocols and optimize perioperative management strategies, a comprehensive understanding of the epidemiological features and risk factors for early postoperative Gram-negative bacterial (GNB) infections specific to our center is crucial. This study aims to retrospectively analyze the clinical data of kidney transplant recipients who developed GNB infections within 30 days after surgery at our center between 2015 and 2024. The findings are expected to provide a critical evidence base for refining our perioperative anti-infective strategies and improving patient outcomes.

2 Materials and methods

2.1 Study population and data collection

The data for this study were derived from an initial cohort comprising all recipients (n = 2875) who underwent kidney transplantation from deceased donors after citizen's death (DCD) at our hospital between January 2015 and December 2024. Through a retrospective review of the clinical records from this cohort, patients who developed a GNB infection within 30 days after surgery were identified, forming the final analysis cohort (n = 102).

Clinical data and patient information were extracted from electronic medical records and outpatient clinic notes. This included basic demographic characteristics, transplant date, operative duration, renal cold ischemia time, induction immunosuppression regimen, microbiological test results, antibiotic treatment regimens, and the occurrence of postoperative complications. These complications comprised septic shock, acute rejection, infectious renal artery rupture, renal artery thrombosis and delayed graft function (DGF). Laboratory data (collected within 24 hours of culture sampling) included white blood cell count, hemoglobin level, platelet count, lymphocyte count, serum albumin level, and serum creatinine level.

The primary endpoint of this study was the incidence of GNB infection. Secondary endpoints included: risk factors associated with multidrug-resistant GNB (MDR-GNB) infection, the microbiological characteristics of the causative pathogens, and the incidence of infection-related complications. This study was approved by the Ethics Committee of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology (Approval No.: TJ-IRB202308113). The requirement for written informed consent was waived due to the retrospective nature of the study. We confirm that all research methods were conducted in accordance with the principles of the Declaration of Helsinki.

2.2 Inclusion and exclusion criteria

The inclusion criteria were as follows: 1) kidney transplant recipients who received a DCD graft; 2) development of a GNB infection within 30 days after surgery; 3) transplantation procedure performed between January 1, 2015, and December 31, 2024.

The exclusion criteria were as follows: 1) specimens suspected of being contaminated; 2) incomplete clinical data; 3) death within 48 hours postoperatively due to non-infectious causes; 4) transfer to another hospital or lost to follow-up within 30 days after surgery; 5) recipients of multi-organ transplantation.

2.3 Definiton

In this study, a GNB infection was defined as a positive culture result obtained from blood, sputum (or other respiratory secretions), lavage fluid, deep wound sites, catheter drainage fluid, or other relevant specimens, accompanied by corresponding clinical symptoms/signs or elevated infection-related laboratory

parameters (e.g., procalcitonin > 0.5 ng/mL), and with exclusion of specimen contamination and simple bacterial colonization⁷. Multidrug resistance was defined as resistance to at least one antimicrobial agent from three or more classes. Difficult-to-treat resistance (DTR) was defined as non-susceptibility (resistant or intermediately susceptible) to all tested carbapenems, β -lactams (including cephalosporins and penicillins with β -lactamase inhibitors, where applicable), and fluoroquinolones⁸. Septic shock was defined as systolic blood pressure < 90 mmHg requiring vasopressor support⁹. DGF was defined as requirement of at least one dialysis treatment within 1 week post-transplantation¹⁰.

2.4 Microbiologic studies

Following kidney transplantation, routine cultures of perigraft drain fluid and sputum were performed daily for 3 to 5 days, with the duration extended as clinically indicated. Other culture investigations, such as those of surgical incision secretions, bronchoalveolar lavage fluid, blood, and urine, were conducted based on clinical judgment. All microbiological data were collected based on the test results reported by our hospital's bacteriology laboratory. The laboratory employed the Kirby-Bauer disk diffusion method for routine antimicrobial susceptibility testing and reporting.

2.5 Statistical analysis

Statistical analysis was performed using SPSS software (version 27.0). Normally distributed continuous variables are presented as mean $\pm$ standard deviation and were compared using the t-test. Non-normally distributed continuous variables are presented as median (interquartile range) and were compared using the Mann-Whitney U test. Categorical variables are presented as number (percentage) and were compared using the chi-square test or Fisher's exact test, as appropriate. Binary logistic regression analysis was employed to identify risk factors associated with MDR-GNB infection, and odds ratios (OR) with their 95% confidence intervals (CI) were calculated. All statistical tests were two-sided, and a p-value of < 0.05 was considered statistically significant.

3 Result

3.1 Distribution of Pathogens and Infection Sites

Among the 2875 recipients who underwent DCD kidney transplantation between January 2015 and December 2024, a total of 102 (3.5%) patients developed a GNB infection within 30 days postoperatively and were included in the final analysis. A total of 129 GNB isolates were obtained. *Klebsiella pneumoniae* was the most frequently isolated pathogen (51 isolates, 39.5%), followed by *Acinetobacter baumannii* (27 isolates, 20.9%), *Escherichia coli* (15 isolates, 11.6%), and *Pseudomonas aeruginosa* (13 isolates, 10.1%). Other species accounted for 23 isolates (17.8%). Regarding the site of infection, surgical site infections yielded the highest number of isolates (81 isolates, 62.8%), followed by urinary tract infections (22 isolates, 17.1%), respiratory tract infections (10 isolates, 7.8%), bloodstream infections (9 isolates, 7.0%), and surgical incision infections (7 isolates, 5.4%) (Table 1).

3.2 Antimicrobial Susceptibility of Multidrug-Resistant Isolates

Among the 102 patients with GNB infection, a total of 63 MDR isolates (48.8%) were identified. Antimicrobial susceptibility testing (Table 2) revealed high resistance rates to commonly used antibiotics. Resistance to piperacillin, ampicillin/sulbactam, ceftazidime, cefepime, aztreonam, ciprofloxacin, and levofloxacin all exceeded 95%. Resistance rates to imipenem and meropenem were both 92.1%. Relatively lower resistance rates were observed for tigecycline (9.5%, 6/63), minocycline (36.5%, 23/63), and trimethoprim-sulfamethoxazole (63.5%, 40/63) (Table 2). Among the MDR isolates, 35 (27.1%) were difficult-to-treat resistant *Klebsiella pneumoniae* (DTRKP) and 16 (25.4%) were difficult-to-treat resistant *Acinetobacter baumannii* (DTRAB). The number of these two difficult-to-treat resistant pathogens detected showed an overall downward trend after 2019 (Fig. 1).

3.3 Risk Factors for Multidrug-Resistant Infection

Univariate analysis indicated that the induction immunosuppression regimen ($P = 0.043$), a lymphocyte count $\leq 0.3 \times 10^9/L$ ($P = 0.007$), and a serum albumin level ≤ 35 g/L ($P = 0.016$) were associated with MDR infection. Notably, the proportion of patients who received anti-thymocyte globulin (ATG) induction was significantly higher in the MDR infection group (18/49, 36.7%) than in the non-MDR infection group (10/53, 18.9%), suggesting that ATG induction may be associated with a higher risk of MDR infection. Multivariate analysis further identified a lymphocyte count $\leq 0.3 \times 10^9/L$ (adjusted HR: 3.328, 95% CI: 1.213–8.996, $P = 0.019$) and a serum albumin level ≤ 35 g/L (adjusted HR: 3.385, 95% CI: 1.135–10.100, $P = 0.029$) as independent risk factors for postoperative MDR-GNB infection. Other factors, including age, gender, BMI, and cold ischemia time, showed no statistically significant difference between the two groups (Table 3).

3.4 Treatment and Outcomes of Recipients with MDR-GNB Infection

Among the 49 kidney transplant recipients who developed MDR-GNB infections, the antibiotic treatment regimens and outcomes are summarized in Table 4. In terms of treatment strategies, a total of 41 recipients (83.7%) received a combination regimen of tigecycline plus imipenem or meropenem, while the remaining 8 recipients (16.3%) were treated with ceftazidime-avibactam (CAZ-AVI). Regarding outcomes and complications, in the tigecycline plus carbapenem group, there was 1 case of acute rejection, 1 case of septic shock, 3 cases of infectious renal artery rupture, 4 cases of renal artery thrombosis, 3 cases of local graft site infection, and 2 cases of DGF. This group ultimately resulted in 5 graft nephrectomies (1 due to infectious renal artery rupture, 3 due to renal artery thrombosis, and 1 due to local graft infection), and 2 deaths within 30 days post-surgery (1 death on postoperative day 24 due to cerebral hemorrhage, and 1 death on postoperative day 15 due to septic shock). In the CAZ-AVI treatment group, there was 1 case of acute rejection, and 2 recipients developed local graft infection complicated by septic shock; both of these recipients ultimately underwent graft nephrectomy.

4 Discussion

Early postoperative GNB infections in kidney transplant recipients often lead to severe adverse outcomes, particularly in the context of the increasing prevalence of MDR strains, which poses substantial challenges to empirical therapy. With the widespread adoption of DCD, the proportion of DCD donors among all graft sources has markedly increased¹¹. DCD donors typically experience prolonged intensive care, broad-spectrum antibiotic exposure, and multiple invasive procedures, significantly elevating the risk of colonization or occult carriage of MDR pathogens. These resistant bacteria can be transmitted to recipients via the transplanted organ or perfusate, thereby severely impacting recipient prognosis¹². Therefore, a systematic analysis of the epidemiological characteristics and resistance risk factors for early postoperative GNB infections specifically in the DCD kidney transplant population is of great importance for optimizing perioperative infection prevention and control strategies.

This study retrospectively analyzed the clinical data of 2,875 DCD kidney transplant recipients between January 2015 and December 2024. A total of 102 recipients (3.5%) developed GNB infections within 30 days after transplantation. In this study, 129 GNB strains were isolated, with *Klebsiella pneumoniae* (39.5%) and *Acinetobacter baumannii* (20.9%) being the predominant pathogens, a distribution consistent with most previous reports^{13,14}. Both pathogens are common hospital-acquired strains that readily colonize and spread in healthcare settings and possess potent acquired resistance capabilities. Kidney transplant recipients, being in an immunosuppressive state, have impaired clearance of these organisms, rendering them a major threat for post-transplant infections. Moreover, surgical site drainage fluid (62.8%) in the early post-transplant period was the most frequent source of pathogen isolation, far exceeding other specimen types such as urine (17.1%) and respiratory tract (7.8%). DCD donors, owing to their critical underlying conditions, often experience prolonged intensive care, with widespread exposure to broad-spectrum antibiotics, multiple invasive procedures, and a high risk of colonization by healthcare-associated resistant bacteria. In addition, the high-dose vasopressors used to maintain circulation can lead to intestinal ischemia and impaired mucosal barrier function, promoting bacterial translocation and occult bacteremia¹⁵. These pathogens, present in the donor's blood or tissues, may contaminate the graft during organ procurement and preservation, and subsequently form infectious foci at the graft site in the immunosuppressed recipient upon restoration of blood flow after transplantation¹⁶. Therefore, donor-derived occult infection may be a critical factor contributing to the predominance of MDR-GNB infections at the surgical site in the early post-transplant period.

The statistical results of this study indicate that the current status of GNB resistance after kidney transplantation at our center remains concerning. The isolated MDR-GNB strains accounted for 48.8% (63/129) of all GNB isolates and exhibited extremely high resistance rates to commonly used clinical β -lactams (including carbapenems), quinolones, and most aminoglycosides, posing substantial challenges to empirical therapy. Kadri et al⁸ proposed the classification of difficult-to-treat resistance for isolates exhibiting intermediate or resistant susceptibility to all antibiotics in the β -lactam (including carbapenems) and fluoroquinolone classes, which are highly effective, low-toxicity first-line antibiotics for treating GNB infections; therefore, the DTR classification is considered more clinically relevant than other resistance classifications¹⁷. In this study, 35 DTRKP strains were identified, accounting for 27.1% (35/129) of all isolates, and 16 DTRAB strains were identified, accounting for 25.4% (16/63) of MDR strains. Annual surveillance of DTR demonstrated a declining trend in the detection of both DTRKP and DTRAB after 2019. This trend may be attributable to the systematic prevention and control strategies implemented at our center during this period, including: optimization of donor-derived infection screening and maintenance protocols to reduce transmission of resistant bacteria via grafts; reinforcement of antimicrobial stewardship by restricting the use of carbapenems and tigecycline, with bacterial cultures obtained prior to antibiotic administration; strict implementation of contact isolation and environmental disinfection to block nosocomial transmission routes; and the introduction of novel antimicrobial agents (e.g., CAZ-AVI), which improved treatment outcomes and reduced bacterial colonization and transmission¹⁸.

In addition, we evaluated the impact of demographic, clinical, and microbiological variables on risk factors for MDR-GNB infection. Univariate analysis revealed that perioperative induction immunosuppressive regimen, lymphocyte count $\leq 0.3 \times 10^9/L$, and albumin ≤ 35 g/L were significantly associated with MDR-GNB infection. Binary logistic regression analysis showed that lymphocyte count $\leq 0.3 \times 10^9/L$ and albumin ≤ 35 g/L were independent risk factors for MDR infection. Lymphopenia directly reflects an excessively immunosuppressed state in patients, leading to impaired clearance of pathogenic microorganisms. Serum albumin primarily serves as a surrogate biomarker for the diagnosis of malnutrition¹⁹; patients with end-stage renal disease face a higher risk of malnutrition due to multiple contributing factors, including dietary restrictions, anorexia, endocrine disorders, persistent inflammation, acidosis, energy loss during dialysis, and increased resting energy expenditure²⁰. Santos et al. analyzed the relationship between nutritional status and post-transplant infection incidence in a cohort of 451 patients and found that patients at high risk of malnutrition had a higher infection rate (35.1%). Early postoperative monitoring of these two simple indicators may help identify patients at high risk for MDR infection. In addition, ATG was significantly associated with MDR infection in univariate analysis. ATG is a potent lymphocyte-depleting agent commonly used in kidney transplant recipients who are highly sensitized or undergoing retransplantation to reduce the incidence of acute rejection²¹. However, excessive or inappropriate immunosuppression can significantly increase infection risk. Studies have demonstrated that ATG induction is associated with an increased risk of bacterial infections after solid organ transplantation, and enhanced monitoring of recipient immune function should be implemented in clinical practice²².

Early postoperative MDR-GNB infection in kidney transplant recipients often leads to poor prognosis and poses a serious challenge to clinical anti-infective therapy. The results of this study showed that among the 49 recipients with MDR-GNB infection, despite treatment with tigecycline combined with carbapenems or CAZ-AVI alone, a notable proportion still developed severe complications and graft loss. In this cohort, 41 recipients with MDR-GNB infection received tigecycline combined with imipenem or meropenem. Infection-related complications in this group included 1 case of septic shock, 3 cases of infectious renal artery rupture, and 3 cases of local graft site infection; some of these patients eventually underwent graft nephrectomy, and the patient with septic shock died on postoperative day 15 despite aggressive management. In addition, 8 recipients received CAZ-AVI, among whom 2 developed septic shock and ultimately required graft nephrectomy. Although CAZ-AVI is a key agent for treating resistant Enterobacteriaceae, such as KPC-producing strains, these novel reserve antibiotics are often reserved for salvage therapy in critically ill patients^{23,24}. However, relevant studies have indicated²⁵ that in immunosuppressed populations, once infection has progressed to severe stages such as septic shock, even with CAZ-AVI, clinical mortality and the risk of adverse outcomes remain high. Moreover, treatment failure may also be attributed to the inability of CAZ-AVI to cover metallo- β -lactamase-producing strains

or the emergence of induced resistance. This suggests that monotherapy still has limitations in the face of complex and severe infections. In addition, surgical intervention for complications remains the ultimate approach to halt infection deterioration. In this cohort, 3 recipients across the two treatment groups underwent graft nephrectomy due to uncontrollable infection. When local infection has caused irreversible vascular destruction or refractory shock, the role of medical therapy becomes very limited; timely graft nephrectomy to completely eradicate the infectious focus is an important surgical decision to prioritize recipient safety.

This study has several limitations. First, as a single-center retrospective study, selection bias and information bias are inevitable. Second, although the overall sample size is substantial, the number of cases in the MDR infection subgroup is limited, which restricts the statistical power for deeper subgroup analyses, such as direct comparisons between different treatment regimens. Third, we did not perform molecular epidemiological analyses, precluding the ability to elucidate the relationships of resistance gene transmission and clonal strain spread. Finally, treatment regimens were not randomly assigned and were influenced by disease severity and physician preference, preventing causal conclusions regarding treatment efficacy.

5 Conclusion

In summary, early post-transplant Gram-negative bacterial infections at our center are predominantly caused by *Klebsiella pneumoniae* and *Acinetobacter baumannii* and are primarily concentrated at the surgical site. Profound lymphopenia and hypoalbuminemia serve as independent early warning indicators for multidrug-resistant infections. Notably, the detection rate of difficult-to-treat resistant pathogens has shown a declining trend in recent years. This may be associated with comprehensive improvements implemented at our hospital in donor management, antimicrobial stewardship, and infection control measures, suggesting that the current prevention and control strategies hold significant clinical value.

Abbreviations

AK, amikacin; ATG, anti-thymocyte globulin; ATM, aztreonam; BMI, body mass index; CAZ, ceftazidime; CAZ-AVI, ceftazidime-avibactam; CI, confidence interval; CIP, ciprofloxacin; CN, gentamicin; DCD, deceased donors after citizen's death; DGF, delayed graft function; DTR, difficult-to-treat resistance; DTRAB, difficult-to-treat resistant *Acinetobacter baumannii*; DTRKP, difficult-to-treat resistant *Klebsiella pneumoniae*; FEP, cefepime; GNB, Gram-negative bacterial; Hb, hemoglobin; IPM, imipenem; KPC, *Klebsiella pneumoniae* carbapenemase; KT, kidney transplantation; LEV, levofloxacin; MDR, multidrug-resistant; MDR-GNB, multidrug-resistant Gram-negative bacterial; MEM, meropenem; MH, minocycline; OR, odds ratio; PRL, piperacillin; SAM, ampicillin/sulbactam; SCF, cefotaxime/sulbactam; SD, standard deviation; SMX, sulfamethoxazole; SPSS, Statistical Package for the Social Sciences; TGC, tigecycline; TOB, tobramycin; TZP, piperacillin/tazobactam; WBC, white blood cell.

Declarations

Ethical Approval and Consent to participate

This study was approved by the Ethics Committee of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology (Approval No.: TJ-IRB202308113). The requirement for written informed consent was waived due to the retrospective nature of the study.

Human Ethics

We confirm that all research methods were conducted in accordance with the principles of the Declaration of Helsinki.

Consent for Publication

Not applicable.

Clinical Trial Number:

Not applicable.

Data availability statement

The datasets generated and analyzed during the current study are not publicly available due to the privacy protection of the participants and ethical restrictions, but are available from the corresponding author upon reasonable request.

Conflicts of interest

There are no conflicts of interest.

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Author contributions

All the authors participated in the study design, data interpretation, and critical review of the manuscript. Q Z, B Z, and Y Z collected and assembled the clinical data. Y Z and B Y performed the statistical analyses. Q Z drafted the initial manuscript. L W, Z S, and D C conceived the study, provided overall scientific guidance, and supervised the project. All authors have approved the final version of the manuscript.

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Tables

Table 1Distribution of 129 Gram-negative bacterial isolates.

	Surgical site infection	Surgical incision	Blood stream	Respiratory tract	Urinary tract infection	Totaln%
<i>Acinetobacter baumannii</i>	21	1	0	3	2	2720.9%
<i>Klebsiella pneumoniae</i>	28	4	6	5	8	5139.5%
<i>Pseudomonas alcaligenes</i>	1	0	0	0	0	10.8%
<i>Enterobacter aerogenes</i>	2	0	0	1	1	43.1%
<i>Pseudomonas aeruginosa</i>	9	0	1	1	2	1310.1%
<i>Escherichia coli</i>	8	1	2	0	4	1511.6%
<i>Citrobacter freundii</i>	0	0	0	0	2	21.6%
<i>Morganella morganii</i>	1	0	0	0	0	10.8%
<i>Pseudomonas putida</i>	1	0	0	0	1	21.6%
<i>Enterobacter cloacae</i>	1	0	0	0	0	10.8%
<i>Acinetobacter lwoffii</i>	2	0	0	0	0	21.6%
<i>Moraxella</i> spp.	1	0	0	0	0	10.8%
<i>Stenotrophomonas maltophilia</i>	1	1	0	0	2	43.1%
<i>Burkholderia cepacia</i> complex	1	0	0	0	0	10.8%
<i>Acinetobacter johnsonii</i>	2	0	0	0	0	21.6%
<i>Serratia marcescens</i>	1	0	0	0	0	10.8%
<i>Proteus mirabilis</i>	1	0	0	0	0	10.8%
Totaln%	8162.8%	75.4%	97.0%	107.8%	2217.1%	129

Table 2. Antimicrobial susceptibility of 63 multidrug-resistant isolates.

	<i>Acinetobacter baumannii</i> 16/25.4%	<i>Klebsiella pneumoniae</i> 35/27.1%	<i>Enterobacter aerogenes</i> 1/1.6%	<i>Pseudomonas putida</i> 2/3.2%	<i>Escherichia coli</i> 5/7.9%	<i>Pseudomonas aeruginosa</i> 2/3.2%	<i>Serratia marcescens</i> 1/1.6%	<i>Proteus mirabilis</i> 1/1.6%
Piperacillin(PRL)	16	35	1	2	5	0	1	1
Ampicillin/sulbactam(SAM)	16	35	1	2	5	0	1	1
Cefotaxime/sulbactam(SCF)	16	35	1	2	2	0	1	0
Piperacillin/tazobactam(TZP)	16	35	1	2	3	0	1	0
Ceftazidime(CAZ)	16	35	1	2	5	0	1	0
Cefepime(FEP)	16	35	1	2	5	0	1	1
Aztreonam(ATM)	16	35	1	1	5	1	1	0
Imipenem(IPM)	16	35	1	2	2	1	1	0
Meropenem(MEM)	16	35	1	2	2	1	1	0
Amikacin(AK)	14	33	1	1	2	0	1	0
Tobramycin(TOB)	15	33	1	2	5	0	0	0
Gentamicin(CN)	16	33	1	2	5	0	1	0
Ciprofloxacin(CIP)	16	35	1	2	5	0	1	0
Levofloxacin(LEV)	16	35	1	2	5	0	1	0
Sulfamethoxazole(SMX)	15	15	1	2	5	1	0	1
Minocycline(MH)	7	11	0	1	2	1	0	1
Tigecycline(TGC)	2	1	0	1	0	0	1	1

Table 3. Univariate and multivariate analysis of multidrug-resistant Gram-negative bacterial (MDRGNB) infection in kidney transplant recipients.

Characteristics	MDR n=49	No MDR n=53	Univariate analysis	Multivariate Analysis	
			P	OR 95%CI	P
Age(y), Mean ± SD	37.9±14.6	41.2±12.4	0.167		
Gender (male) n%	34/69.4	31/58.5	0.253		
BMI (kg/m ²), Mean ± SD	21.3±3.1	22.7±3.3	0.582		
Year of KT n%			0.125		
2015-2018	39/80.0	35/66.0			
2019-2024	10/20.0	18/34.0			
Cold ischemia time	360/270.0-520.0	390/300.0-600.0	0.485		
Induction therapy n%			0.043	0.617 4.397	0.320
Basiliximab	31/63.3	43/81.1			
Anti-thymocyte globulin	18/36.7	10/18.9			
Laboratory variables n%					
WBC count ≥ 10 × 10 ⁹ /L	24/49.0	27/50.9	0.843		
Hb level ≤ 90g/L	13/26.5	23/43.4	0.075		
Lymphocyte count ≤ 0.3 × 10 ⁹ /L	19/38.8	8/15.1	0.007	1.213 8.996	0.019
Albumin level ≤ 35 g/L	15/30.6	6/11.3	0.016	1.135 10.100	0.029
Creatinine(μmol/L)	252/145.0-438.0	362/151.0-616.0	0.276		

Table 4 Antibiotic Regimens and Outcomes in 49 Recipients with MDR-GNB Infection								
	Acute rejection	Septic shock	Infectious renal artery rupture	Local graft infection	Renal artery thrombosis	DGF	Graft loss	30-day mortality
Tigecycline + imipenem / meropenem 41/49	1	1	3	3	4	2	5	2
CAZ-AVI 8/49	1	2	0	2	0	0	2	0

Figures

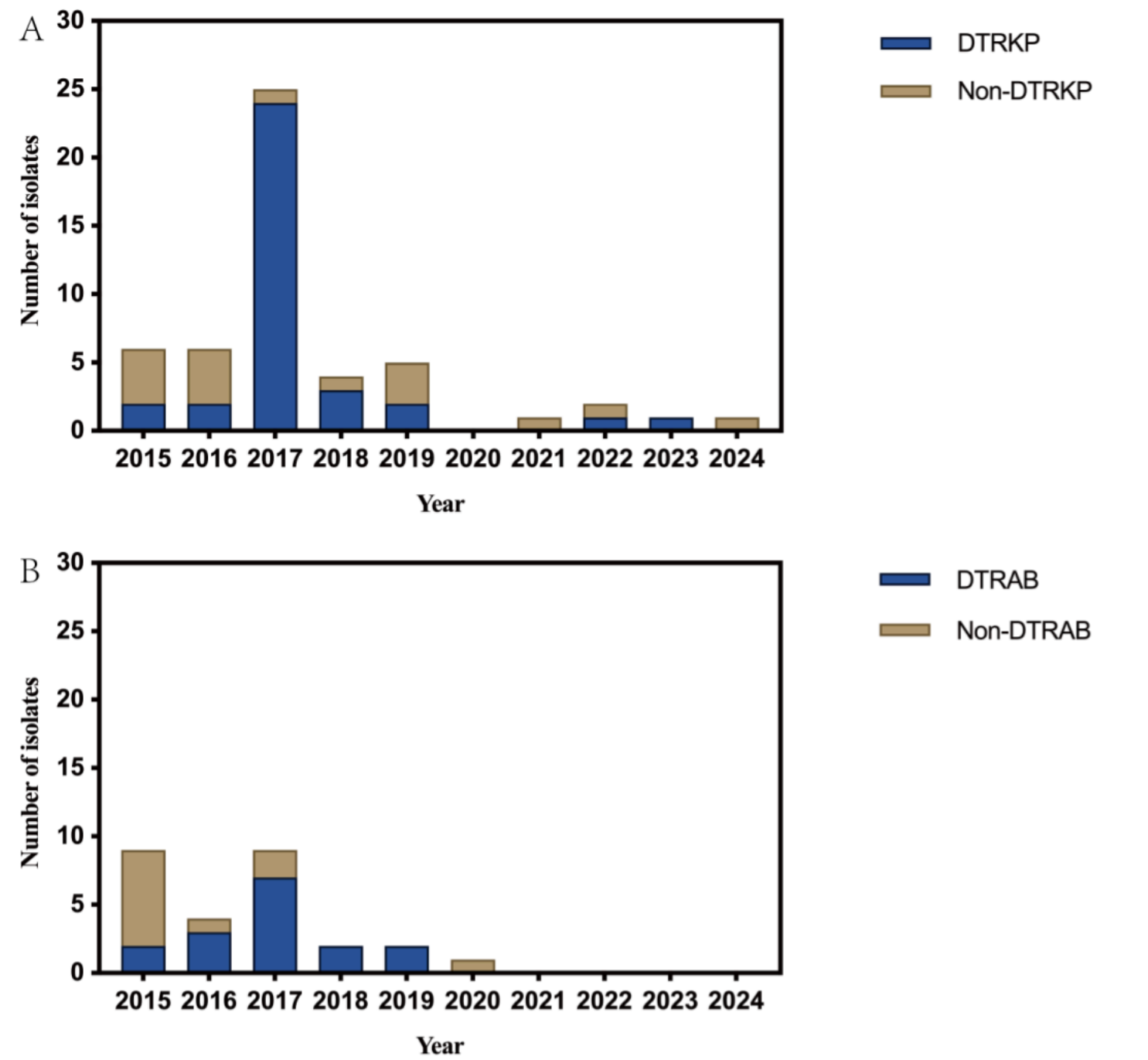


Figure 1

Detection of DTRKP and DTRAB from 2015 to 2024 (A: DTRKP; B: DTRAB).