

RESEARCH

Open Access



Effects of preservative fluid associated possible donor-derived carbapenem-resistant *Klebsiella Pneumoniae* infection on kidney transplantation recipients

Fei Zhang^{1,2,3†}, Jinbiao Zhong^{1,2,3†}, Handong Ding^{1,2,3†} and Guiyi Liao^{1,2,3*}

Abstract

Background: Infections remain a major cause of morbidity and mortality in kidney transplant (KT) recipients. This study aimed to investigate the preservation fluid (PF) samples from deceased donors and report the impacts of possible donor-derived carbapenem-resistant *Klebsiella pneumoniae* (pdd-CRKP) infections on KT recipients.

Methods: A retrospective study was performed that included all recipients who received kidney transplantation from deceased donors in our hospital between December 2018 and December 2020. A total of 212 patients received kidney transplantation from deceased donors, a total of 206 PF samples were collected, and 20 recipients had a CRKP-positive culture. Both donors and recipients with CRKP-positive PF cultures were divided into two groups, and continuous variables between the two groups were compared using independent-sample t tests and Mann-Whitney tests. Categorical variables were compared using the chi-square test or Fisher's exact test. The significance level of *p* values was set at 0.05.

Results: A total of 337 recipients underwent kidney transplantation, including 212 recipients of organs from deceased donors and 110 corresponding deceased donors. A total of 206 PF samples were collected, and 20 recipients had CRKP-positive PF cultures. The donors' length of ICU stay was a potential risk factor for CRKP positivity in the PF culture ($P < 0.05$). Fifteen recipients were infected with pdd-CRKP, and the incidence of pdd-CRKP infection was 7.3% (15/206). The use of antibiotics, including ceftazidime-avibactam (CAZ-AVI), was a potential protective factor against death and graft loss in recipients with a CRKP-positive PF culture ($P < 0.05$).

Conclusions: This study shows that the incidence of pdd-CRKP is high in our centre, recipients with pdd-CRKP infection can still achieve a good prognosis with the use of antimicrobial agents including CAZ-AVI.

Keywords: Donor-derived infection, Carbapenem-resistant *Klebsiella pneumoniae*, Preservation fluid, Possible donor-derived carbapenem-resistant *Klebsiella pneumoniae*, Kidney transplantation

Introduction

Kidney transplantation is the most effective therapy for end-stage kidney diseases [1]. However, approximately 60% of the deaths and graft losses in the first year after transplantation are caused by infection and perioperative complications [2, 3]. Infection has become a major cause of morbidity and mortality after solid organ

*Correspondence: liaoguiyi@ahmu.edu.cn

†Fei Zhang, Jinbiao Zhong and Handong Ding contributed equally to this work.

¹ Department of Urology, the First Affiliated Hospital of Anhui Medical University, Hefei, Anhui Province, China

Full list of author information is available at the end of the article



transplantation (SOT). Bacterial infection is the most common type of infection after transplantation, with most infections occurring in the first three months after transplantation [3, 4]. An accumulation of data over the past few years confirm an increasing trend of gram-negative bacterial infections and the emergence of multidrug-resistant (MDR) bacterial pathogens [5]. An Italian study on SOT recipients showed that 16% of organ recipients were infected by gram-negative microorganisms, most of whom were infected with carbapenem-resistant *Klebsiella pneumoniae* (CRKP) [6].

In recent years, with the increase in the number of kidney transplant (KT) recipients, the gap between the number of available kidneys and the number of potential recipients has widened. The increased demand has led to increased utilisation of organs from marginal donors, including infected marginal donors [7]. Donor-derived infections (DDIs) have also become one of the major problems after SOT. Some studies reported that recipients with this type of infection could achieve favourable outcomes with appropriate antimicrobial therapy [8–11]; however, some authors suggested DDIs are associated with severe morbidity and mortality [12, 13], depending on the type of pathogenic microorganism [14]. CRKP has been reported as one of the most common pathogens in DDIs after kidney transplantation from deceased donors [15] and is characterised by high mortality, a high transmission rate and MDR [16]. Data showed that the morbidity of CRKP infection after SOT ranged from 2.5 to 35%, showing a high case fatality rate of 62.5 to 82% [17, 18].

The transmission of microorganisms through preservation fluid (PF) is a potential method for early DDI in KT recipients after KT, and routine microbial analysis of PF can help identify patients with a high risk of early post-transplantation infection [19]. Most studies have shown that coagulase-negative Staphylococcus and Enterococci are the most common contaminants in PF, most of which are low-virulence bacteria [20–22]. To date, the impact of CRKP contamination in PF on kidney recipients is not clear, and the best strategies for preventing and treating subsequent infections in kidney recipients remain unclear. Therefore, this study aimed to investigate the incidence of CRKP contamination in deceased donor PF and the impacts of possible donor-derived carbapenem-resistant *Klebsiella pneumoniae* (pdd-CRKP) infection after KT on recipients; their prevention and treatment options are also discussed.

Materials and methods

Study design and population

This study was a single-centre retrospective study. The case data of all KT recipients and the corresponding donors from December 2018 and December 2020 were

reviewed via electronic medical records in the Kidney Transplantation Center of the First Affiliated Hospital of Anhui Medical University. Six recipients who did not have PF cultures were excluded from the study. None of these kidneys were obtained from prisoners. In this study, all kidneys were donated voluntarily after the death of the donors, and were obtained by an organ procurement organisation established by the hospital. The study was approved by our Institutional Ethics Review Committee and conducted in accordance with the Declaration of Helsinki guidelines.

Some variables were collected from the recipients' medical records, including age, sex, body mass index, aetiology of kidney failure, delayed graft function, type of induction therapies, type of dialysis, duration of dialysis, site of infections, use of antibacterial drugs, crude mortalities and graft losses within six months, complications after kidney transplantation, perioperative infection events and their pathogens, and antibiotic sensitivities. In addition, donor data were collected, including age, sex, cause of death, classification of utilised organs, time spent in the intensive care unit (ICU) before donation, and warm ischaemia time.

Definitions

The classification of utilised organs was as follows: China Category I (C-I), donation after brain death (DBD); China Category II (C-II), donation after cardiac death (DCD); and China Category III (C-III), donation after brain death plus cardiac death (DBCD) [23]. DDI refers to an infection occurring when the pathogen existing in the donor's body caused the recipient to be infected with the same pathogen after organ donation through the organ transplantation process [24]. Accurate diagnosis of DDI requires highly sensitive technologies, such as whole genome sequencing and the confirmed genetic relationships between pathogens. A possible DDI was defined as an infection occurring when the pathogen in the course of the recipient infection was identical to the microorganism cultured in the PF and had the same drug sensitivity profile [21]. The criteria used in our study to define and classify infections were proposed by the Centers for Disease Control and Prevention [25]. Delayed graft function was defined as a decrease in daily serum creatinine less than 10% from the previous day for 3 consecutive days in the first postoperative week or serum creatinine failing to decrease to 400 $\mu\text{mol/L}$ in the first postoperative week [21].

Preservation fluid and method for detecting microorganisms

The graft PF consisted of a hypertonic citrate purine solution (S400, Shanghai, China) and was used for graft

perfusion during organ retrieval and for preservation during graft transportation. Prior to kidney transplantation, two samples of PF (15 ml) were collected from the bag containing the kidney, and each sample was added to a sterile blood culture bottle and fungal culture flasks for microbial culture. Bacterial species were cultured and identified using the VITEK-2 system (Biomérieux, Marcy-l'Étoile, France), and the minimum inhibitory concentration was interpreted according to the breakpoint set by the Clinical and Laboratory Standards Institute [26].

Immunosuppressive therapy and infection prevention programs

All enrolled recipients received triple immunosuppression (tacrolimus or cyclosporin A, prednisone, and mycophenolate mofetil), and some patients were induced with anti-thymocyte immunoglobulin. The recipients were given meropenem 1 g intravenous infusion to prevent infection during the operation, and cefoperazone sulbactam sodium was given for infection prevention after the operation for at least 7 days. At the same time, antifungal prophylaxis was administered for 2 weeks, and the postoperative antimicrobial treatment was adjusted according to the microbial resistance spectrum identified in the recipient specimens and PF.

Statistical analysis

Statistical analysis was performed using SPSS software [Version 25.0; SPSS Inc., Chicago, IL, USA]. Continuous variables with a normal distribution are described as the means and standard deviations; otherwise, they are represented as the medians and interquartile ranges (IQRs). The Kolmogorov-Smirnov test was used to assess variable distributions. Independent-sample *t* tests were used to compare quantitative variables with a normal distribution between groups. When quantitative variables did not follow a normal distribution, the Mann-Whitney test for nonparametric variables was used for comparisons between groups. Categorical variables are presented as frequencies and percentages. The chi-square test or Fisher's exact test was used for the comparison of categorical variables between groups, as appropriate. For all tests, $P < 0.05$ was defined as statistically significant.

Results

A total of 337 recipient underwent kidney transplantation between December 2018 and December 2020, including 212 recipients of organs from deceased donors and 110 corresponding deceased donors. A total of 206 PF samples were collected, and 20 recipients had

CRKP-positive PF cultures. The incidence of death or graft loss in recipients with CRKP-positive PF cultures was 6/20(30.0%), and the incidence of death or graft loss in recipients with CRKP-negative PF cultures was 15/186(8.1%).

Characteristics of deceased donors

During the study, twenty CRKP-positive recipient kidneys derived from 11 deceased donors were cultured in PF. The characteristics of the donors are shown in Table 1. There was a significant difference in the length of ICU stay between CRKP-positive kidneys cultured in PF and CRKP-negative kidneys cultured in PF ($P < 0.05$), but there were no significant differences in sex, age, cause of death, China classification of donation or warm ischaemia time. In both groups of donors, brain trauma was the most common cause of death, followed by cerebrovascular accidents. The vast majority of donors were classified as China Category III.

Comparison of recipients with graft loss or death and recipients without graft loss or death when PF culture was CRKP-positive

A total of 20 recipients were included in the study, with an average age of 44.3 years, 15 of whom were male. The incidence of pdd-CRKP infection was 7.3% (15/206). The most common aetiology of kidney failure was glomerulonephritis, and 15 recipients developed pdd-CRKP infection. The drug sensitivity profiles of CRKP cultured in the recipients were the same as those in the PF, and the infections occurred shortly after the operation, with an average time of 7.7 days. The difference between the two groups in the use of antibiotics, including ceftazidime-avibactam (CAZ-AVI), was statistically significant ($P < 0.05$), suggesting that the use of antibiotics, including CAZ-AVI, was a potential protective factor against graft loss or death in recipients. Due to the limited sample size, univariate and multivariate analyses could not be performed. The characteristics of the recipients are shown in Table 2.

Impacts of pdd-CRKP infection on recipients and outcomes

The kidneys of 10 recipients with pdd-CRKP infection were derived from six donors, and six liver transplantation recipients from these six donors also had isolated CRKP with the same drug susceptibility profile. The other 5 recipient kidneys with pdd-CRKP infection were derived from three other deceased donors. Two of the donors did not perform liver donation, and the other donors' liver recipients did not cultivate CRKP. The isolation of the same drug-sensitivity profile of CRKP from six liver recipients provided

Table 1 Baseline characteristics of donors with or without a CRKP-positive preservation fluid culture

Characteristics	Donors with CRKP-positive PF N = 11	Donors without CRKP-positive PF N = 93	P Value
Sex, male n (%)	7(63.6)	51(54.8)	0.751
Age (years)	50.82 ± 9.19	48.57 ± 9.93	0.476
Cause of death n (%)			
Brain trauma	6(54.5)	51(54.8)	0.985
Cerebrovascular accidents	3(27.3)	19(20.4)	0.696
Brain tumour	1(9.1)	11(11.8)	0.788
Others	1(9.1)	12(12.9)	0.718
China classification of donation n (%)			
I	1(9.1)	17(18.3)	0.685
II	1(9.1)	21(22.6)	0.450
III	9(81.8)	55(59.1)	0.197
ICU stay in days, median (IQR)	30(21-39)	11(9-14)	P<0.001
Warm ischaemia time (min), median (IQR)	11(10-13)	11(9-14)	0.928

Table 2 Baseline characteristics of recipients with a CRKP-positive preservation fluid culture

Characteristics	Recipients with graft loss or death N = 6	Recipients without graft loss or death N = 14	P Value
Sex, male n (%)	5(83.3)	10(71.4)	0.573
Age (years)	36.8 ± 8.9	36.0 ± 11.8	0.879
BMI (kg/m ²)	22.5 ± 3.8	22.4 ± 3.6	0.948
Aetiology of kidney failure, n (%)			
HTA	1(16.7)	1(7.1)	0.521
DM	1(16.7)	1(7.1)	0.521
Glomerulonephritis	4(66.7)	11(78.6)	0.613
Others	0(0.0)	1(7.1)	0.502
Type of dialysis n (%)			
HD	4(66.7)	12(85.7)	0.549
PD	2(33.3)	2(14.3)	0.549
Duration of dialysis (years) median (IQR)			
Duration of HD	1.5(0.0-5.5)	2.0(0.4-5.0)	0.588
Duration of PD	0.0(0.0-1.8)	0.0(0.0-0.0)	0.345
DGF n (%)	1(16.7)	5(35.7)	0.613
ATG induction n (%)	2(33.3)	5(35.7)	0.919
Pdd-CRKP infection (%)	6(100.0)	9(64.3)	0.091
Antibiotic regimen containing ceftazidime avibatan (%)	1(16.7)	11(78.6)	0.018

Abbreviations: BMI body mass index, HTA hypertension, DM diabetes mellitus, HD haemodialysis, PD peritoneal dialysis, DGF delayed graft function, ATG anti-thymocyte globulin

stronger evidence of CRKP transmission from donors to recipients. However, none of these CRKP-infected recipients could be classified as having a proven DDI, and all 15 CRKP-infected recipients were classified as having a possible DDI. The infection characteristics of the patients with pdd-CRKP infection are shown in

Table 3. The most common type of infection was surgical site infection, followed by bloodstream infection. Among the 15 recipients, 3 underwent graft nephrectomy due to CRKP infection. In addition, three recipients died, one due to rupture of the graft artery and two due to septic shock.

Table 3 Frequency and incidence of infection in recipients with a CRKP-positive preservation fluid culture

Infectious events	Number of cases (n)	Incidence (n = 20)
Surgical site infection	11	55.0%
Infectious allograft kidney artery disruption	1	5.0%
Urinary tract infection	3	15.0%
pneumonia	2	10.0%
Bloodstream infection	10	50.0%

Antibiotics protocol for CRKP-positive culture in PF

At the time of transplantation, PF culture results had not yet been reported, so the standard antimicrobial drug prevention regimen was adopted in our centre. When the PF culture results were reported, the antimicrobial drug regimen was adjusted according to the drug sensitivity results. The antibiotics selected in the 20 recipients with positive CRKP in recipient PF cultures are shown in Table 4. Eight of the 20 recipients received tigecycline combined with meropenem or imipenem for prophylactic anti-infection, 4 received CAZ-AVI alone, 5 received CAZ-AVI combined with meropenem, and 3 were first given tigecycline combined with imipenem and then CAZ-AVI for salvage treatment. Three deaths and two graft losses occurred during the prophylactic anti-infective regimen of tigecycline combined with meropenem or imipenem. The other surviving recipients underwent graft nephrectomy with tigecycline combined with imipenem after failing to respond to anti-infective therapy, followed by salvage therapy with CAZ-AVI. Two of the four recipients of ceftazidime alone on a prophylactic anti-infective regimen developed surgical incision infection but recovered successfully. Among the 5 recipients who used CAZ-AVI combined with meropenem as a preventive anti-infective regimen, although 2 cases of incision infection and one case of bacteraemia occurred, they all recovered smoothly after anti-infective treatment. The spectrum of susceptibility of

Table 5 Antimicrobial susceptibility of isolates from recipients with pdd-CRKP infections

Antibiotic	Susceptible (%)
Ceftazidime	0.0
Levofloxacin	13.3
Gentamycin	20.0
Imipenem	0.0
Meropenem	0.0
Amikacin	40.0
Polymyxin	86.7
Tigecycline	93.3
Ceftazidime-Avibatam	100.0

CRKP strains isolated from recipients with pdd-CRKP infection is shown in Table 5.

Discussion

Although numerous reports have shown that approximately 3% of SOT recipients are affected by fatal DDI, the use of organs from marginal donors is increasing due to the current organ shortage [11, 27]. The incidence of DDI is reduced by donor screening and the use of prophylactic antimicrobials in recipients, but transmission can still occur and may lead to increased morbidity and mortality [28]. The PF is a possible pathway for DDI. Most of the microorganisms in PF are coagulase-negative *Staphylococci* and *Enterococci* [20, 21], and most of them are contaminated saprophytic flora. Because of the widespread use of prophylactic antibiotics in transplant recipients, these low-virulence organisms are difficult to transmit through PF, resulting in significantly reduced pathogenicity [29]. However, MDR *Enterobacteriaceae* are more virulent and more likely to transmit through PF [30, 31]. Nevertheless, there are few data on the donor-derived transmission of MDR gram-negative bacilli through PF, especially CRKP. To our knowledge, this report represents the largest study of kidney transplant recipient DDIs caused by CRKP to date.

Table 4 Prophylactic antimicrobial therapy regimens in recipients with a CRKP-positive preservation fluid culture

Prophylactic antimicrobial therapy regimens(n)	Death(n)	Graft loss(n)	Patient and graft survival(n)
Tigecycline+ imipenem / meropenem (8/20)	3	2	3
CAZ-AVI + meropenem (5/20)	0	0	5
CAZ-AVI as initial therapy (4/20)	0	0	4
CAZ-AVI as salvage therapy (3/20)	0	1	2

Abbreviations: CAZ-AVI Ceftazidime-Avibatam

In our study, a 7.3% (15/206) incidence of pdd-CRKP infection was associated with PF, and the risk of total infection events due to PF contamination ranged from 0.83 to 14.3% in previous reports [32, 33]. However, only CRKP in PF was investigated, and DDIs caused by other microorganisms in PF were not investigated in this study. The rate of positive CRKP culture in PF in our centre is high, which may be related to the high incidence of CRKP in our centre. In China, CHINET data showed that the resistance rates of *Klebsiella pneumoniae* to imipenem and meropenem rose steadily from 9.2 and 9.2% in 2010 to 23.3 and 24.2% in 2020, respectively. The detection rate of CRKP in Anhui Province, where our centre is located, increased steadily from 8.9% in 2010 to 25% in 2020. On the other hand, in our cohort, the ICU stay of donors in the CRKP-positive PF culture group was longer than that in the CRKP-negative PF culture group, and the difference was statistically significant. Prolonged (>7 days) ICU stays, the use of vasopressors, and the need for cardiopulmonary resuscitation were reported to be independent risk factors for predicting potential infections in donors [34]. Studies have found that a hospital stay of as little as 2 days is sufficient to acquire MDR hospital pathogens that can be transmitted by transplantation [35]. In our study, the age of donors with CRKP-positive PF cultures was generally higher, with an average age of 50.8 years. A prospective study by Oriol et al. found that higher donor age was an independent risk factor for positive culture of high-risk microorganisms in PF [36]. Microbial contamination of the ICU environment and intestinal damage during the organ retrieval process may also contaminate the PF and lead to a higher positive rate of CRKP cultures.

Whether microbial contamination of PF in SOT is related to DDI is controversial. Bertrand et al. reported a negligible incidence of clinical complications due to PF contamination [20]. However, some other studies have suggested that it is a common cause of early post-transplant infection [29, 37]. The occurrence of DDI also depends on the type of contaminating microorganism. MDR *Klebsiella pneumoniae*, MDR *Acinetobacter baumannii* and other bacteria are more likely to cause DDIs associated with PF than other sensitive *Cocci* species [21]. Cai et al. reviewed the literature on donor-derived CRKP infection from 2011 to 2018, including a total of 18 donor-derived CRKP infection recipients. Postoperatively, 1 patient died early (9 days), 5 patients died late (≥ 28 days), and 1 patient lost the graft kidney due to rupture of the graft artery. The overall mortality was 33.3% [13], which was higher than the overall mortality of pdd-CRKP infection in our centre (20%).

Most of the DDIs associated with PF were surgical site infections, including local infection of the graft site,

infectious renal graft artery rupture and wound infection [21], which was consistent with the results of our centre. The most common type of infection in our centre was surgical site infection, and 11 of the 20 recipients had surgical site infection, followed by bloodstream infection. CRKP bloodstream infections have been reported to be the most fatal type of infection, with a mortality of up to 20–50% [38, 39]. A total of 10 CRKP-positive recipients developed bloodstream infection in our centre. Among these 10 CRKP-positive bloodstream infection recipients, there were 2 deaths and 3 graft losses. One of the recipients died of graft artery rupture, a fatal complication of donor-derived CRKP infection, which has been reported in both liver and kidney recipients with donor-derived CRKP infection [13, 40].

Due to the lack of evidence, there is no broad consensus regarding the treatment recipients with of CRKP-positive cultures in PF. Some authors have suggested that when MDR is detected in donors and PF, targeted antibacterial treatment should be performed after transplantation, which could prevent the transmission of MDR gram-negative bacteria. DDIs have been reported despite antibiotic prophylaxis [41, 42]. The authors also suggested that extended antibiotic use for 10 days after surgery might help reduce the risk of transmission and associated mortality [19]. Our study confirmed the clinical efficacy of CAZ-AVI in the prevention and treatment of CRKP infection. None of the 9 recipients who were initially given prophylactic anti-infective therapy with CAZ-AVI alone or CAZ-AVI combined with meropenem died or had graft loss. It has been reported that CAZ-AVI treatment is the only independent predictor of survival in CRKP-infected patients [43], and recent clinical trials have also demonstrated that CAZ-AVI-based therapy has successfully treated CRKP infection in immunocompromised populations [44]. In addition to drug control of infection, effective source control, such as allogeneic nephrectomy and complete debridement, can increase the success rate of cured infections [45–47]. Allograft nephrectomy not only provides complete debridement but also reduces the bacterial load. In our study, all three surviving recipients with severe CRKP infection received allograft nephrectomy, complete debridement and drainage, and all achieved good outcomes.

Test protocols for the rapid detection of pathogens carried by donors await further study. The time required for routine microbial culture in PF from specimen collection to final results (including drug susceptibility results) may range from 48 h to 120 h, depending on the testing time and the need for additional resistance testing. This delay affects the time to start appropriate antimicrobial therapy. Although the polymerase chain reaction method can quickly obtain test results,

it is expensive, has limited specificity and cannot provide information about the sensitivity of the drug. Delay in communication is another important factor in the transmission of infection [48]. Effective and timely communication between the organ procurement organisation (OPO) and the transplant centre is essential to control the transmission of pathogens and should be conducted within 24 h. Therefore, routine sample culture (blood, urine, and tracheal secretions) of potential donors in hospitals in areas where CRKP pathogens are endemic is recommended for the rapid identification of pathogens [9]. Rapid communication between the organ transplant centre and the OPO is also necessary, and it can play an important role in accelerating the use of targeted antibiotic treatment in patients who receive organs from infected donors.

The study has several limitations. First, the lack of routine microbiological screening of donors made it difficult to determine the source of CRKP and the relationship between donor infection and donor-derived CRKP infection after transplantation. Second, there were no data on the use of antibiotics given to donors. Third, regarding the transmission of DDI, genotyping technology provides necessary objective evidence for this event. Unfortunately, this technology is not widely used in our hospital. Therefore, our detection algorithm may overestimate transmission events. Finally, our sample size was small, mainly because of the low incidence of donor-derived CRKP infection, and our results may not be applicable to other transplant centres.

Conclusion

In summary, this study showed that the incidence of pdd-CRKP is high in our centre. Recipients with pdd-CRKP infection can still achieve a good prognosis with the use of antimicrobial agents including CAZ-AVI and donor screening for CRKP should be included in measures to contain the incidence of DDI.

Abbreviations

CRKP: Carbapenem-resistant *Klebsiella pneumoniae*; DDI: Donor-derived infections; PF: Preservation fluid; ICU: Intensive care unit; KT: Kidney transplant; MDR: Multidrug resistance; pdd-CRKP: possible donor-derived carbapenem-resistant *Klebsiella pneumoniae*; SOT: Solid organ transplantation.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12882-022-02733-7>.

Additional file 1.

Additional file 2.

Acknowledgements

The authors are grateful to Yuyang Zhang for the assistance provided.

Authors' contributions

Fei Zhang, Jinbiao Zhong, Handong Ding: study design, statistical analysis, data interpretation, manuscript preparation, literature search. Guiyi Liao: study design, provision of materials and resources, data interpretation. All authors read and approved the final manuscript.

Funding

This project received funding from the Natural Science Foundation of Anhui Province (Grant No. 1508085SMH226).

Availability of data and materials

All data generated or analysed during this study are included in this published article [and its supplementary information files].

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki guidelines. In the retrospective cohort, the requirement of informed consent from study participants was waived because of the retrospective and anonymized nature of this study and was approved by the Ethics Committee of The First Affiliated Hospital of Anhui Medical University (Approval Number Quick-PJ 2021-12-15).

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Author details

¹Department of Urology, the First Affiliated Hospital of Anhui Medical University, Hefei, Anhui Province, China. ²Institute of Urology, the First Affiliated Hospital of Anhui Medical University, Hefei, Anhui Province, China. ³Anhui Province Key Laboratory of Genitourinary Diseases, Anhui Medical University, Hefei, Anhui Province, China.

Received: 6 November 2021 Accepted: 9 March 2022

Published online: 14 March 2022

References

- Hernandez D, Muriel A, Abraira V. Current state of clinical end-points assessment in transplant: key points. *Transplant Rev (Orlando)*. 2016;30(2):92–9. <https://doi.org/10.1016/j.trre.2016.02.003>.
- Siritip N, Nongnuch A, Dajsakdipon T, Thongprayoon C, Cheungprasitporn W, Bruminhent J. Epidemiology, risk factors, and outcome of bloodstream infection within the first year after kidney transplantation. *Am J Med Sci*. 2021;361(3):352–7. <https://doi.org/10.1016/j.amjms.2020.10.011>.
- KOC, Kennedy C, Skally M, et al. Surveillance of common infections in the early period after renal transplantation in a national center: 2014–2017. *Transpl Infect Dis*. 2020;22(3):e13261. <https://doi.org/10.1111/tid.13261>.
- Fishman JA. Infections in immunocompromised hosts and organ transplant recipients: essentials. *Liver Transpl*. 2011;17(Suppl 3):S34–7. <https://doi.org/10.1002/lt.22378>.
- Barchiesi F, Montalti R, Castelli P, et al. Carbapenem-Resistant *Klebsiella pneumoniae* influences the outcome of early infections in liver transplant recipients. *BMC Infect Dis*. 2016;16(1):538. <https://doi.org/10.1186/s12879-016-1876-5>.
- Lanini S, Costa AN, Puro V, et al. Incidence of carbapenem-resistant gram negatives in Italian transplant recipients: a nationwide surveillance study. *PLoS One*. 2015;10(4):e0123706. <https://doi.org/10.1371/journal.pone.0123706>.
- McKeown DW, Bonser RS, Kellum JA. Management of the heartbeating brain-dead organ donor. *Br J Anaesth*. 2012;108(Suppl 1):i96–107. <https://doi.org/10.1093/bja/aer351>.

8. Ison MG, Grossi P, Practice ASTIDCo. Donor-derived infections in solid organ transplantation. *Am J Transplant*. 2013;13(Suppl 4):22–30. <https://doi.org/10.1111/ajt.12095>.
9. Mularoni A, Bertani A, Vizzini G, et al. Outcome of transplantation using organs from donors infected or colonized with Carbapenem-resistant gram-negative Bacteria. *Am J Transplant*. 2015;15(10):2674–82. <https://doi.org/10.1111/ajt.13317>.
10. Mills JP, Wilck MB, Weikert BC, et al. Successful treatment of a disseminated infection with extensively drug-resistant *Klebsiella pneumoniae* in a liver transplant recipient with a fosfomycin-based multidrug regimen. *Transpl Infect Dis*. 2016;18(5):777–81. <https://doi.org/10.1111/tid.12578>.
11. Altman DR, Sebra R, Hand J, et al. Transmission of methicillin-resistant *Staphylococcus aureus* via deceased donor liver transplantation confirmed by whole genome sequencing. *Am J Transplant*. 2014;14(11):2640–4. <https://doi.org/10.1111/ajt.12897>.
12. Kieslichova E, Protus M, Nemcova D, Uchytlova E. Single multidrug resistant enterobacteriaceae donor-derived infection in four solid organ transplant recipients: a case report. *BMC Surg*. 2019;19(1):111. <https://doi.org/10.1186/s12893-019-0574-9>.
13. Cai YS, Xiao H, Zhang S, et al. Carbapenem-resistant *Klebsiella pneumoniae* infection causing rupture of graft artery in solid organ recipients: case report and review of literature. *Medicine (Baltimore)*. 2019;98(45):e17878. <https://doi.org/10.1097/MD.00000000000017878>.
14. Janny S, Bert F, Dondero F, et al. Microbiological findings of culture-positive preservation fluid in liver transplantation. *Transpl Infect Dis*. 2011;13(1):9–14. <https://doi.org/10.1111/j.1399-3062.2010.00558.x>.
15. Wang Y, Lei H, Zhang Y, et al. Epidemiology of carbapenem-resistant *Klebsiella pneumoniae* bloodstream infections after renal transplantation from donation after cardiac death in a Chinese hospital: a case series analysis. *Antimicrob Resist Infect Control*. 2018;7:66. <https://doi.org/10.1186/s13756-018-0355-8>.
16. Gu D, Dong N, Zheng Z, et al. A fatal outbreak of ST11 carbapenem-resistant hypervirulent *Klebsiella pneumoniae* in a Chinese hospital: a molecular epidemiological study. *Lancet Infect Dis*. 2018;18(1):37–46. [https://doi.org/10.1016/s1473-3099\(17\)30489-9](https://doi.org/10.1016/s1473-3099(17)30489-9).
17. Pereira MR, Scully BF, Pouch SM, et al. Risk factors and outcomes of carbapenem-resistant *Klebsiella pneumoniae* infections in liver transplant recipients. *Liver Transpl*. 2015;21(12):1511–9. <https://doi.org/10.1002/lt.24207>.
18. Mouloudi E, Massa E, Papadopoulos S, et al. Bloodstream infections caused by carbapenemase-producing *Klebsiella pneumoniae* among intensive care unit patients after orthotopic liver transplantation: risk factors for infection and impact of resistance on outcomes. *Transplant Proc*. 2014;46(9):3216–8. <https://doi.org/10.1016/j.transproceed.2014.09.159>.
19. Grąt MLJ, Lewandowski Z, Barski K, Hołwko W, Skalski M, Kornasiewicz O, et al. Incidence, pattern and clinical relevance of microbial contamination of preservation fluid in liver transplantation. *Ann Transplant*. 2012;17(3):20–8. <https://doi.org/10.12659/aot.883454>.
20. Bertrand D, Pallet N, Sartorius A, et al. Clinical and microbial impact of screening kidney allograft preservative solution for bacterial contamination with high-sensitivity methods. *Transpl Int*. 2013;26(8):795–9. <https://doi.org/10.1111/tri.12130>.
21. Yu X, Wang R, Peng W, et al. Incidence, distribution and clinical relevance of microbial contamination of preservation solution in deceased kidney transplant recipients: a retrospective cohort study from China. *Clin Microbiol Infect*. 2019;25(5):595–600. <https://doi.org/10.1016/j.cmi.2018.12.040>.
22. Mattana AMMA, Machado AM, Lopes Filho Gde J, Salzedas Netto AA, Gonzalez AM. Evaluation of the presence of microorganisms in solid-organ preservation solution. *Braz J Infect Dis*. 2011;15(6):528–32. <https://doi.org/10.1590/s1413-86702011000600005>.
23. Huang J, Wang H, Fan ST, et al. The national program for deceased organ donation in China. *Transplantation*. 2013;96(1):5–9. <https://doi.org/10.1097/TP.0b013e3182985491>.
24. Lewis JD, Sifri CD. Multidrug-resistant bacterial donor-derived infections in solid organ transplantation. *Curr Infect Dis Rep*. 2016;18(6):18. <https://doi.org/10.1007/s11908-016-0526-9>.
25. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control*. 2008;36(5):309–32. <https://doi.org/10.1016/j.ajic.2008.03.002>.
26. Clinical and Laboratory Standards Institute. M100-S25. Performance standards for antimicrobial susceptibility testing: 28th informational supplement. Wayne: Clinical and Laboratory Standards Institute; 2018.
27. Kirchner VA, Pruett TL. Receiving the unwanted gift: infection transmission through organ transplantation. *Surg Infect*. 2016;17(3):318–22. <https://doi.org/10.1089/sur.2016.009>.
28. Ison MG, Nalesnik MA. An update on donor-derived disease transmission in organ transplantation. *Am J Transplant*. 2011;11(6):1123–30. <https://doi.org/10.1111/j.1600-6143.2011.03493.x>.
29. Wakelin SJ, Casey J, Robertson A, et al. The incidence and importance of bacterial contaminants of cadaveric renal perfusion fluid. *Transpl Int*. 2005;17(11):680–6. <https://doi.org/10.1007/s00147-004-0792-6>.
30. Oriol I, Sabe N, Tebe C, Veroux M, Boin I, Carratala J. Clinical impact of culture-positive preservation fluid on solid organ transplantation: a systematic review and meta-analysis. *Transplant Rev (Orlando)*. 2018;32(2):85–91. <https://doi.org/10.1016/j.trre.2017.11.003>.
31. Ranghino A, Diena D, Simonato F, et al. Clinical impact of bacterial contamination of perfusion fluid in kidney transplantation. *Springerplus*. 2016;5:7. <https://doi.org/10.1186/s40064-015-1658-3>.
32. Chaim FH, Boin IF, Ataide EC, Stucchi RS. Perfusion fluid contamination in relation to recipient survival and acute cellular rejection in orthotopic liver transplantation: retrospective analysis. *Transplant Proc*. 2011;43(4):1313–5. <https://doi.org/10.1016/j.transproceed.2011.02.030>.
33. Audet M, Piardi T, Panaro F, et al. Incidence and clinical significance of bacterial and fungal contamination of the preservation solution in liver transplantation. *Transpl Infect Dis*. 2011;13(1):84–8. <https://doi.org/10.1111/j.1399-3062.2010.00529.x>.
34. Wu TJ, Lee CF, Chou HS, Yu MC, Lee WC. Suspect the donor with potential infection in the adult deceased donor liver transplantation. *Transplant Proc*. 2008;40(8):2486–8. <https://doi.org/10.1016/j.transproceed.2008.07.039>.
35. Martins N, Martins IS, de Freitas WV, et al. Severe infection in a lung transplant recipient caused by donor-transmitted carbapenem-resistant *Acinetobacter baumannii*. *Transpl Infect Dis*. 2012;14(3):316–20. <https://doi.org/10.1111/j.1399-3062.2011.00701.x>.
36. Oriol I, Sabe N, Camara J, et al. The Impact of Culturing the Organ Preservation Fluid on Solid Organ Transplantation: A Prospective Multicenter Cohort Study. *Open Forum Infect Dis*. 2019;6(6):ofz180. <https://doi.org/10.1093/ofid/ofz180>.
37. Veroux M, Corona D, Scriffignano V, et al. Contamination of preservation fluid in kidney transplantation: single-center analysis. *Transplant Proc*. 2010;42(4):1043–5. <https://doi.org/10.1016/j.transproceed.2010.03.041>.
38. Cicora F, Mos F, Roberti J. Kidney transplant recipients infected with blaKPC-2-producing *Klebsiella pneumoniae*. *Nefrologia*. 2015;35(3):340–3. <https://doi.org/10.1016/j.nefro.2015.02.002>.
39. Meatherall BL, Gregson D, Ross T, Pitout JD, Laupland KB. Incidence, risk factors, and outcomes of *Klebsiella pneumoniae* bacteremia. *Am J Med*. 2009;122(9):866–73. <https://doi.org/10.1016/j.amjmed.2009.03.034>.
40. Qian Y, Zhang H, Chen X, Zhang J, Xia Q. Graft hepatic artery rupture due to Carbapenem-resistant *Klebsiella pneumoniae* infection after liver transplant. *Exp Clin Transplant*. 2020;18(4):529–32. <https://doi.org/10.6002/ect.2018.0384>.
41. Ariza-Heredia EJ, Patel R, Blumberg EA, et al. Outcomes of transplantation using organs from a donor infected with *Klebsiella pneumoniae* carbapenemase (KPC)-producing *K. pneumoniae*. *Transpl Infect Dis*. 2012;14(3):229–36. <https://doi.org/10.1111/j.1399-3062.2012.00742.x>.
42. Doucette KE, Al-Saif M, Kneteman N, et al. Donor-derived bacteremia in liver transplant recipients despite antibiotic prophylaxis. *Am J Transplant*. 2013;13(4):1080–3. <https://doi.org/10.1111/ajt.12133>.
43. Tambarello M, Trearichi EM, Corona A, et al. Efficacy of Ceftazidime-Avibactam salvage therapy in patients with infections caused by *Klebsiella pneumoniae* Carbapenemase-producing *K. pneumoniae*. *Clin Infect Dis*. 2019;68(3):355–64. <https://doi.org/10.1093/cid/ciy492>.
44. Satlin MJ, Walsh TJ. Multidrug-resistant Enterobacteriaceae, *Pseudomonas aeruginosa*, and vancomycin-resistant Enterococcus: Three major threats to hematopoietic stem cell transplant recipients. *Transpl Infect Dis*. 2017;19(6). <https://doi.org/10.1111/tid.12762>.
45. Clancy CJ, Chen L, Shields RK, et al. Epidemiology and molecular characterization of bacteremia due to carbapenem-resistant *Klebsiella pneumoniae* in transplant recipients. *Am J Transplant*. 2013;13(10):2619–33. <https://doi.org/10.1111/ajt.12424>.

46. Simkins J, Muggia V, Cohen HW, Minamoto GY. Carbapenem-resistant *Klebsiella pneumoniae* infections in kidney transplant recipients: a case-control study. *Transpl Infect Dis*. 2014;16(5):775–82. <https://doi.org/10.1111/tid.12276>.
47. Wang Z, Qian Y, Bai H, Yang J, Li X. Allograft hemorrhage as a manifestation of carbapenem-resistant *Klebsiella pneumoniae* infection in kidney transplant recipients: case series. *Medicine (Baltimore)*. 2020;99(13):e18982. <https://doi.org/10.1097/MD.00000000000018982>.
48. Miller R, Covington S, Taranto S, et al. Communication gaps associated with donor-derived infections. *Am J Transplant*. 2015;15(1):259–64. <https://doi.org/10.1111/ajt.12978>.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Ready to submit your research? Choose BMC and benefit from:

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

At BMC, research is always in progress.

Learn more biomedcentral.com/submissions

