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Outcomes of transplantation using organs from a donor infected with *Klebsiella pneumoniae* carbapenemase (KPC)-producing *K. pneumoniae*

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Abstract: Transmission of pathogens from donor to recipient is a potential complication of organ transplantation. Herein, we describe the clinical course and outcomes of 4 transplant recipients who received tissues from a donor with multi-organ infection with *Klebsiella pneumoniae* carbapenemase (KPC)-producing *K. pneumoniae*. Recipient 1 underwent simultaneous liver and kidney transplantation for alpha-1 antitrypsin deficiency and alcohol-related cirrhosis, and acute tubular necrosis, respectively. Soon after transplantation, he developed an infected hematoma and peritonitis due to KPC-producing *K. pneumoniae* despite receiving tigecycline prophylaxis. He was treated with a prolonged course of tigecycline, amikacin, and meropenem, in conjunction with surgical evacuation and percutaneous drainage of the infected fluid collections. Recipient 2 underwent living-donor liver transplantation for cholangiocarcinoma and primary sclerosing cholangitis using vein graft from the donor infected with KPC-producing *K. pneumoniae*. Culture of the preservation fluid containing the vein graft was positive for KPC-producing *K. pneumoniae*. The patient received preemptive amikacin and tigecycline, and he did not develop any infection (as evidenced by negative surveillance blood cultures). The isolates from the donor and Recipients 1 and 2 were indistinguishable by pulsed-field gel electrophoresis. Recipients 3 and 4 underwent kidney and heart transplantation, respectively; both patients received perioperative tigecycline prophylaxis and did not develop infections due to KPC-producing *K. pneumoniae*. All transplant recipients had good short-term outcomes. These cases highlight the importance of inter-institutional communication and collaboration to ensure the successful management of recipients of organs from donors infected with multidrug-resistant organisms.

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The transmission of an infection from donor to recipient is a well-recognized risk of solid organ transplantation that can occur despite defined screening and prevention strategies, and may result in increased morbidity and mortality (1). Consequences of donor-derived infections have included sepsis syndromes (2) and arterial anastomotic disruption (3). Given the scarcity of organs, the need to increase the donor pool, and the absence of definitive contraindications to the use of organs from infected donors, the American Society of Transplantation does not specifically preclude organ donation from patients with common

treatable infections (1, 4). For example, some studies show that donors with septic shock, pneumonia, or meningitis due to organisms such as *Neisseria meningitidis* and *Streptococcus pneumoniae* have successfully donated organs without major repercussions, if donors receive treatment for at least 48 h before demise (5, 6), and the recipient is also treated accordingly (7). There are, however, no specific directives about individuals with more resistant and difficult-to-treat organisms, such as multidrug-resistant (MDR) gram-negative bacteria.

Carbapenemase production has recently emerged as an important mechanism for antimicrobial resistance among some members of the Enterobacteriaceae, and this is most commonly reported from centers in Europe and the northeastern United States (8). Infections due to plasmid-borne class A carbapenemases, such as *Klebsiella pneumoniae* carbapenamase (KPC), have resulted in poor clinical outcomes after transplantation (9). To our knowledge, no cases of donor-derived infections due to KPC-producing organisms have been reported. Moreover, it is unclear if organs from individuals with active infections due to KPC-producing organisms can be successfully used for organ transplantation.

Herein, we describe the clinical course and outcomes of 4 patients who received tissues from a donor with active infection due to KPC-producing *K. pneumoniae*. All transplant patients were managed with either 1) a surgical approach and combination antimicrobial treatment, 2) preemptive antimicrobial therapy, or 3) antimicrobial prophylaxis. These cases emphasize that early recognition, treatment, and prevention control measures can be successfully implemented, resulting in successful outcomes of organ transplantation.

Case reports

Donor

Liver, kidneys, heart, and vein grafts were procured in hospital A from a 21-year-old male donor who sustained multiple injuries from a motor vehicle accident and who was hospitalized for about 3 weeks before his demise. While undergoing treatment, he developed pneumonia, an infected subdural hematoma, and meningitis due to KPC-producing *K. pneumoniae*. No blood cultures were positive. The isolate was susceptible to colistin, gentamicin, and tigecycline (Table 1). The donor had received treatment with intravenous (IV) tigecycline for 9 days and 3 doses of intrathecal gentamicin at the time of his death. Cultures were still positive for KPC-producing *K. pneumoniae* 2 days before his demise. Upon the decision for organ donation, as required by policy, the case was reported to the Organ Procurement and Transplantation Network (OPTN).

Recipient 1

In hospital B, the liver and kidney allografts were transplanted into a 63-year-old man with cirrhosis due to alpha-1 antitrypsin deficiency and alcohol. He was critically ill in the intensive care unit and had a model for end-stage liver disease score of 36. He was undergoing hemodialysis because of acute tubular necrosis. During liver allograft procurement, a large hematoma arising in segment VI was noted and was débrided. At that time, the transplant team was notified about the donor's infection history and, before transplantation, the patient and his family were informed of the donor infection history. Subsequently, the patient underwent liver transplantation with standard reconstruction via piggyback technique and bile duct-to-bile duct end-to-end anastomoses. A kidney from the same donor was transplanted in the left lower quadrant. The patient was started on IV tigecycline prophylaxis (initial loading dose of 100 mg, followed by 50 mg IV every 12 h for a planned duration of 2 weeks) based on susceptibility pattern of the donor isolates (Table 1). Basiliximab was given as induction therapy, and thereafter, the immunosuppressive regimen consisted of mycophenolate mofetil (MMF) (1000 mg by mouth [p.o.] every 12 h), prednisone (5 mg p.o. daily), and tacrolimus (4 mg p.o. every 12 h). The patient was a cytomegalovirus donor positive/recipient negative sero-mismatch, and was initiated on valganciclovir (900 mg p.o. once daily) prophylaxis.

Liver ultrasound after transplantation showed good vein and artery Doppler flow, but a large perihepatic hematoma measuring $9.2 \times 13.7 \times 14.2$ cm was noted. On postoperative day (POD) +10, while receiving IV tigecycline, he developed severe abdominal pain, tenderness, and leukocytosis (white blood cell count, 16,000 cells/mm^3). Computed tomography (CT) scan of the abdomen and pelvis showed a right upper quadrant perihepatic hematoma, which was larger than on the previous ultrasound findings, and an increased amount of ascites. Cultures of the ascitic fluid were positive for KPC-producing *K. pneumoniae*. The antimicrobial susceptibility pattern of the organism is listed in Table 1. Testing was performed by agar dilution according to Clinical and Laboratory

Antibiogram with susceptibility patterns for *Klebsiella pneumoniae* carbapemenase (KPC)-producing *K. pneumoniae* isolates obtained from a single donor and 2 transplant recipients

		MERO	ERT	IMIP	CRO	PIP/TAZ	CIPRO	Tigecycline	AMIK	GENT	Colistin
Donor[1]											
Sputum		R			R	R	R	S		S	S
Recipient 1											
Isolate #1[2]	MIC	> 8 R	> 4 R	> 8 R	> 32 R	> 64/2 R	> 2 R	**MIC 4**	**16 S**	<1 S	<2 S
Isolate #2[3]	MIC	> 8 R	> 4 R	> 8 R	> 32 R	> 64/2 R	> 2 R	**MIC > 4**	**32 I**	2 S	<2 S
Recipient 2											
Preservative fluid isolate	MIC	> 8 R	> 4 R	> 8 R	> 32 R	> 64/2 R	> 2 R	MIC 2	16 S	<1 S	<2 S

Bolded values indicate changes in minimum inhibitory concentration (MIC) values.
[1]Susceptibilities provided as a "qualitative" report from the outside facility (Resistant [R] or Sensitive [S]).
[2]Isolate #1 = post-transplant day 10 from hepatic hematoma.
[3]Isolate #2 = post-transplant day 24 from abdominal fluid collection.
MERO, meropenem; ERT, ertapenem; IMIP, imipenem; CRO, ceftriaxone; PIP/TAZ, piperacillin/tazobactam; CIPRO, ciprofloxacin; AMIK, amikacin; GENT, gentamicin.
S (susceptible), R (resistant), I (intermediate) in reference to MIC.

Table 1

Standards Institute (CLSI) guidelines, with the use of a modified Hodge test for confirmation of carbapenemase production, and polymerase chain reaction for confirmation of the presence of bla_{KPC} (10).

Based on the susceptibility pattern of the donor and the recipient isolates, IV amikacin was added to tigecycline. In addition, ciprofloxacin was empirically added for possible synergy. The patient underwent exploratory laparotomy and washout. The intraoperative cultures were positive for KPC-producing *K. pneumoniae*. *Candida albicans* was also isolated from the intraoperative cultures, and the patient was started on fluconazole (200 mg p.o. daily). During the same week, his liver enzymes increased, and a liver biopsy was consistent with mild acute cellular rejection. Because of infection with an MDR bacterium, no specific anti-rejection treatment was given.

On POD +24, a follow-up ultrasound of the abdomen revealed a persistent perihepatic fluid collection. Radiology-guided insertion of a new drainage catheter was performed. Cultures obtained from the drainage procedure were positive for KPC-producing *K. pneumoniae*.

By this time, the isolate had an increased tigecycline minimum inhibitory concentration (MIC) (4 → >4 μg/mL), amikacin MIC (16 → 32 μg/mL), and gentamicin MIC (< 1 → 2 μg/mL). *In vitro* synergy testing from the first isolate was performed using the antibiotic gradient E-test technique, and was reported as the calculated fractional inhibitory concentration index (measuring the *in vitro* activity of each primary drug in the presence of each secondary drug). The result revealed synergy between meropenem and amikacin, but not between ciprofloxacin and amikacin, or amikacin and tigecycline (Table 2). Hence, tigecycline and ciprofloxacin were discontinued, and subsequent treatment consisted of meropenem and amikacin. Amikacin was chosen over gentamicin because of concerns about further development of resistance, as the donor had already received gentamicin. Colistin was reserved as a salvage option, in case the combination therapy with amikacin and meropenem failed and its combination with amikacin has the potential for synergistic nephrotoxicity. The culture of the fluid also revealed penicillin-susceptible *Enterococcus* species, and IV ampicillin was started.

The patient received combination antibiotic therapy with amikacin (500 mg IV q 12 h), meropenem (1 g IV every 8 h), ampicillin (1 g IV every 6 h), and fluconazole (200 mg p.o. daily) for 4 weeks. He was discharged home, and at the end of the treatment, an abdominal CT scan demonstrated resolution of hematoma, with a minimal amount of peritoneal fluid. All percutaneous catheters were removed. Five months after liver transplantation, while off antibiotic treatment, the patient demonstrated no recurrence of infection.

Recipient 2

A vein graft obtained from the infected donor was emergently used in hospital B during the living-donor

Results of antibiotic synergy testing of the first isolate from the kidney-liver transplant recipient (Recipient 1)

ANTIBIOTICS	MIC (mcg/mL) Individual drug	MIC (mcg/mL) Drug in combination	FIC
Colistin	4		
Meropenem	32		
Ciprofloxacin	> 32		
Piperacillin/Tazobactam	> 256		
Tigecycline	3		
Cefepime	> 256		
Amikacin	12		
Amikacin-Cefepime		2/32	0.29
Amikacin-Mepropenem		2/4	0.29
Amikacin-Tigecycline		12/3	2
Amikacin-Ciprofloxacin		12/>32	2
Colistin-Tigecycline		0.5/3	2
Colistin-Meropenem		2/16	1
Colistin-Mepropenem		0.75/3	1
Colistin-Ciprofloxacin		4/32	2
Colistin-Ciprofloxacin		0.5/> 32	1
Colistin-Piperacillin/Tazobactam		4/> 256	2
Colistin-Piperacillin/Tazobactam		0.5/128	1
Ciprofloxacin-Tigecycline		> 32/3	2

Fractional Inhibitory Concentration (FIC) index refers to index interpretation of drug combinations provided by Focus Laboratory.
FIC $\leq$ 0.5 "Synergy detected."
FIC > 0.5 to $\leq$ 1.0 "Additive" or minimal increased activity.
FIC > 1.0 to $\leq$ 4 "Indifference."
FIC > 4.0 "Antagonism detected."
MIC, minimum inhibitory concentration.

Table 2

liver transplantation of a 58-year-old man with hilar cholangiocarcinoma in the setting of primary sclerosing cholangitis. He had received pre-transplant chemotherapy and brachytherapy. During living donor liver transplantation, an iliac vein graft from the infected deceased donor was used for anastomosis between the iliac vein of the recipient and portal vein of the transplanted liver. Culture of the preservative fluid containing the graft vessel was positive for KPC-producing *K. pneumoniae*. The transplant team was aware of these results, but at that time, no other vein graft was available from other donors. Therefore, the patient's family was informed of this finding and they agreed to proceed with the use of the vein graft.

The patient had no immediate surgical complications. He received induction therapy with IV methylprednisolone (500 mg intraoperatively, 250 mg on POD +1, and 125 mg on POD +2). Because of the donor's infection history (and the information gathered from Recipient 1), the patient received perioperative prophylaxis with IV tigecycline (initial loading dose of 100 mg, followed by 50 mg IV every 12 h) and amikacin (600 mg IV every 8 h), and the patient was placed on strict isolation.

On POD +7, a liver biopsy showed mild acute cellular rejection, and he received IV methylprednisolone (1000 mg IV $\times$ 3 doses). Thereafter, his immunosuppression regimen consisted of tacrolimus (0.5 mg p.o. every 12 h), MMF (1000 mg p.o. every 12 h), and prednisone (10 mg p.o. daily). The patient received IV tigecycline and amikacin for 1 week, and completed 7 more days of monotherapy with IV tigecycline (total of 14 days). A CT scan of the abdomen on day 20 after liver transplantation did not reveal any fluid collections or ascites. Surveillance cultures of the patient's blood after completion of antibiotics were

negative. No fevers or other signs of infection were reported as of 5 months after liver transplantation.

Recipient 3

The other kidney from the infected donor was transplanted, in hospital C, into a 50-year-old man with end-stage renal disease due to diabetes mellitus. He had received renal replacement therapy for 8 years before deceased donor renal transplantation. The transplant team was aware of the KPC-producing *K. pneumoniae* infection in the donor, and this was discussed with the patient who agreed to proceed with transplantation. Because of the donor's history, the recipient received a single pre-transplant dose of IV gentamicin (4 mg/kg) and tigecycline (100 mg). The right donor kidney was transplanted into the recipient's right iliac fossa without intraoperative complications. Induction immunosuppression consisted of alemtuzumab (30 mg) and methylprednisolone (500 mg intraoperatively, 250 mg on POD +1, and 125 mg on POD +2). Oral cyclosporine (CsA) and IV MMF were initiated post surgery. Pharmacokinetic profiling for CsA subsequently showed high trough and low peak levels. Therefore, CsA was discontinued and tacrolimus was initiated with doses adjusted to achieve trough target of 6–8 ng/mL. Surveillance cultures from the preservative fluid were negative. A 10-day course of IV tigecycline (50 mg every 12 h) was completed. The postoperative course was complicated by delayed graft function, which necessitated the use of renal replacement therapy for several days. Renal ultrasound was negative for fluid collections. During the post-transplant period, he developed severe but transient cholestasis, with a negative workup, and this slowly resolved by POD +13. The patient had delayed wound healing associated with serous drainage, but this was not thought to be infected and was managed successfully using wound vacuum-assisted closure (VAC). Five months after transplantation, the patient is doing well.

Recipient 4

The heart allograft was transplanted, in hospital D, to a 69-year-old man with a 12-year history of non-ischemic cardiomyopathy. The transplant team was aware of the donor's infection history and the patient consented to proceed with heart transplantation. Based on the donor's infection history, the perioperative antibiotic regimen consisted of IV cefepime (2 g every 12 h) and tigecycline (100 mg loading dose and then 50 mg twice daily), with first doses started before and continued until 72 h after heart transplantation. The immunosuppression regimen included methylprednisolone 500 mg IV twice given intraoperatively (once on induction and again at reperfusion) followed by 125 mg IV every 8 h for 24 h, then p.o. prednisone taper; MMF 1500 mg p.o. twice a day (b.i.d.), and tacrolimus 1-2 mg p.o. b.i.d. Other antimicrobial prophylaxis included valacyclovir (1000 mg p.o. once daily), trimethoprim-sulfamethoxazole (single-strength p.o. daily), and inhaled amphotericin B (100 mg inhalation daily for 2 days, then 50 mg daily for 2 days, then 50 mg weekly until hospital discharge).

Culture of thrombus from the donor heart was negative for bacteria, fungus, or mycobacteria. Thus, no special isolation precaution was implemented. Subsequent cultures were performed based on clinical indications. His first endomyocardial biopsy on POD +7 showed International Society for Heart and Lung Transplantation grade 2R/3A rejection, which was treated with a p.o. prednisone "boost" of 2 mg/kg twice daily for 3 days. The patient was discharged from the hospital without any signs of infection on POD +9.

A week after hospital dismissal, he developed leukocytosis (25,000 cells/mm^3). Evaluation revealed a new pericardial effusion and a stable small right pleural effusion. Cultures of his urine and blood were negative. Subsequently, yellow cloudy fluid started draining from his sternal wound site. He underwent re-exploration of median sternotomy, with evacuation of pericardial effusion and placement of wound VAC. Intraoperative mediastinal cultures yielded *C. albicans*, and all other cultures were negative. Five days later, he underwent further sternal debridement, with exploratory laparotomy and omental flap to the anterior mediastinum. Cultures were negative, but because of concerns of the donor infection, he received gentamicin 5 mg/kg IV during the surgical procedure, and his postoperative antibiotic regimen consisted of micafungin, gentamicin, tigecycline, vancomycin, and ceftazidime. He completed a 6-week total course of p.o. fluconazole.

Microbiology studies

The isolate from the donor was compared with the isolates from the liver-kidney recipient (Recipient 1) and from the preservation fluid containing the vessel graft (which was transplanted into Recipient 2). Figure 1 shows the pulsed-field gel electrophoresis

image of the 3 isolates using *Xba*I restriction enzyme revealing indistinguishable banding patterns for the 3 isolates (Fig. 1). This finding provided proof of donor-derived infection and therefore was reported to the OPTN as required by policy.

Discussion

This report describes the clinical courses of 4 transplant patients who received organs or tissue from a donor infected with KPC-producing *K. pneumoniae*. Numerous cases have been reported documenting transmission of infectious agents from donors to recipients with poor outcomes (11, 12). Much of the emphasis on donor-derived infections has focused on unusual pathogens such as lymphocytic choriomeningitis virus (13), rabies virus (14), and *Balamuthia mandrillaris* (15). Reports of donor transmission of human immunodeficiency virus and West Nile virus have recently emphasized the threat posed by donor-transmitted infections (16, 17). Several instances of transmission of common treatable bacterial pathogens have also been documented with variable results (11, 12). To our knowledge, only one case report has specifically addressed donor-derived infection with MDR organisms (18). Therefore, no data-driven recommendations are available about the use of organs in this setting.

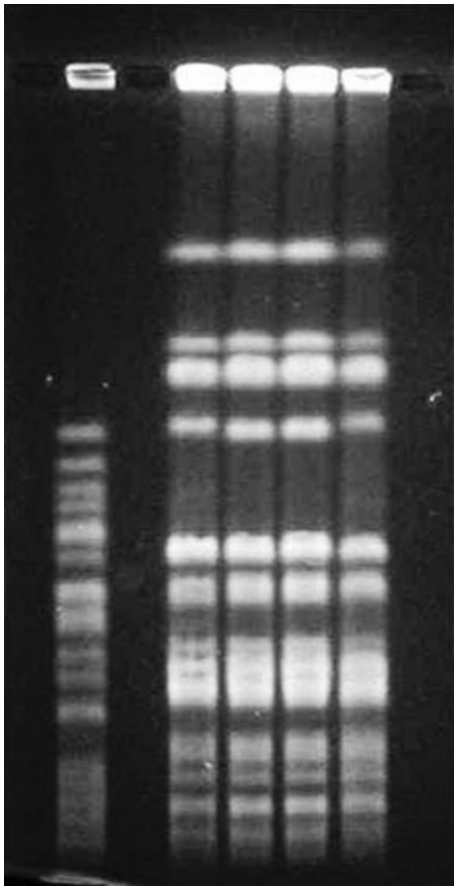

Fig. 1. Pulsed-field gel electrophoresis of *Klebsiella pneumoniae* carbapemenase (KPC)-producing *K. pneumoniae* isolates from an organ donor and 2 transplant recipients. *From left to right*: The first lane (1) is a control *Escherichia coli*, followed by a blank lane. Lane 2 is the isolate from the preservation fluid containing the vessel graft used in Recipient 2. Lanes 3 and 4 were isolates from transplant Recipient 1. Lane 5 is the isolate from the donor.

The first transplant recipient reported here (Recipient 1) represents definitive evidence of KPC-producing *K. pneumoniae* transmission by organ transplantation. Several factors are highlighted herein with regard to the challenges of management of donor-derived KPC-producing *K. pneumoniae* infections, including the critical importance of inter-institutional communication, drainage of infected collections, and infection control precautions.

The major question raised is whether an organ procured from a patient with such an MDR bacterial isolate can safely be used. The difficulties in treating MDR organisms, especially KPC-producing Enterobacteriaceae with limited therapeutic options, are highlighted in this report. Only a limited number of antibiotics (tigecycline, amikacin, gentamicin, and colistin) had *in vitro* activity against the isolate, and in one isolate, the MIC to these drugs increased during the antibiotic use. Therefore, some centers may opt not to use organs from donors infected with such organisms. However, as demonstrated in this report, these organs may be procured and used for transplantation, either because of critical illness of the patient (as in Recipient 1, who may not have survived, had he not received liver and kidney transplantation), or because of pre-defined assessment of low risk of possible transmission from a non-bacteremic donor (as in Recipients 3 and 4, where surveillance cultures remained negative). In all these cases, communication about the donor status among team members and with the patient/family was documented.

As required by policy, these cases were reported to OPTN. As importantly, there was prompt inter-institutional communication that, we believe, was a key component of successful management. For example, the procurement teams were informed of the infection history, while transplant infectious disease physicians from hospital A communicated with other colleagues at the other institutions. All these communications occurred before organ transplantation. Because of the

immediate communication of the isolate's nature and susceptibility pattern, before the acceptance of the organs, appropriate preventive strategies were implemented at the time of transplantation. Contact isolation was implemented or reinforced as needed in patients with positive cultures, and pathogen-directed antibiotic treatment was commenced in all 4 cases (in the form of antimicrobial prophylaxis, and in the case of Recipient 2, preemptive treatment). Indeed, the occurrence of these cases allowed us to revisit our policies and procedures, and it reinforced the presence of existing channels of communication among the transplant surgical teams and infectious disease specialists.

The importance of sharing information between transplant centers has been advocated by the Centers for Disease Control and Prevention (CDC) as a key point in the successful management of cases where directed antimicrobial therapy is required (18). The OPTN/United Network for Organ Sharing (UNOS) has a policy requiring the prompt sharing of culture results between centers and Organ Procurement Organizations, and potential donor-derived infections are tracked by the OPTN/UNOS through the *Ad Hoc* Disease Transmission Advisory Committee. We believe that, although infection was not totally avoided in the liver-kidney recipient (Recipient 1), the timely communication of vital donor-related information mitigated an otherwise potentially catastrophic infection (such as bacteremia, sepsis, and vascular complications) (18–20), by the prompt administration of pathogen-directed therapy. Early pathogen-directed antimicrobial therapy was initiated in all the transplant recipients because of concern for potential donor-derived transmission. Given the MDR nature of the donor infection, it is especially critical to have accurate information about donor cultures at the time of transplantation.

The optimal treatment of infections caused by KPC-producing Enterobacteriaceae is yet to be determined, but should be guided by antimicrobial susceptibility testing. Prior cases of invasive carbapenemase-producing Enterobacteriaceae infection after transplantation have been treated with different antibiotic regimens, including colistin alone or in combination with rifampin and imipenem (continuous infusion) (21), gentamicin and tigecycline (22), or monotherapy with tigecycline (23). The difficulty in treating KPC-producing *K. pneumoniae* was highlighted by the development of an elevated MIC to tigecycline, gentamicin, and amikacin in the isolate from Recipient 1. This phenomenon has been previously reported with the use of tigecycline for treatment of KPC-producing *K. pneumoniae* infection (23, 24). Changes in MIC during treatment emphasize the importance of rechecking susceptibilities, as well as surgical evacuation of contained infections to reduce the microbial burden as much as possible. In our cases, patients with positive cultures received combination amikacin–meropenem therapy (guided by synergy testing), or tigecycline–amikacin.

The impact of gram-negative donor-derived infections on the allograft can be substantial and includes disruption of anastomoses with graft loss and death. Thus far, no evidence of graft failure or non-functioning graft has been seen in all 4 recipients of this study. However, in 3 cases, biopsy-proven acute cellular rejection occurred. This rejection is potentially a result of our cautious approach to immunosuppression in the face of an MDR organism. MDR *Escherichia coli* donor-derived infection has been associated with graft failure in a kidney recipient (18). Likewise, in a study involving liver transplant recipients from infected donors, including gram-negative organisms, there was increased risk for primary non-functioning graft (12), and prior studies on lung transplant recipients have suggested acute and chronic rejection associated with donor infection by MDR organisms (25). In our cases, despite the early rejection episodes, there were no other short-term effects on allograft function. Recipient 3 had delayed graft function, which was attributable to prolonged ischemic time rather than related to infection or rejection.

There are important infection control implications of carbapenemase-producing Enterobacteriaceae, with numerous nosocomial outbreaks reported in the literature. With aggressive infection-control strategies, the spread of this MDR organism can be avoided. At the center where the first 2 patients received their transplant, surveillance cultures were performed among the patients at the same hospital location where the patients with active positive cultures for KPC-producing *K. pneumoniae* were located. In an effort to control this emerging problem of carbapenemase-producing Enterobacteriaceae, the CDC and the Healthcare Infection Control Practices Advisory Committee updated their recommendations to include aggressive infection control strategies, such as the management of infected patients using contact precautions and implementing CLSI guidelines for the detection of carbapenemase production in their respective laboratories (26). Fortunately, no evidence was found of other infection or colonization by carbapenemase-producing Enterobacteriaceae related to the index cases.

In conclusion, this is the first report to our knowledge of donor-derived KPC-producing *K. pneumoniae* infection after transplantation with good clinical

outcomes. Paramount to the success of therapy was effective communication between transplant centers, along with early and aggressive intervention, drainage of an infected collection, plus combined antimicrobial therapy.

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