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Case report

Fatal case of donor-derived colistin-resistant carbapenemase-producing *Klebsiella pneumoniae* transmission in cardiac transplantation

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ABSTRACT

Herein we report a fatal case of donor-derived transmission of XDR-resistant carbapenemase-producing *Klebsiella pneumoniae* (KPC-Kp) in cardiac transplantation. A 59-year-old male patient with non-obstructive hypertrophic cardiomyopathy underwent heart transplantation. On day 5 post-operation, blood cultures from the donor were positive for colistin-resistant carbapenemase-producing *K. pneumoniae* (ColR KPC-Kp) susceptible only to amikacin. Recipient blood cultures were also positive for ColR KPC-Kp with the same sensitivity profile as the donor isolate with an identical PFGE pattern. The patient was treated with double-carbapenems and amikacin. The patient evolved to pericarditis, osteomyelitis, and pulmonary necrosis, all fragment cultures positive for the same agent. The patient developed septic shock, multiple organ failure and died on day 50 post-transplantation. Based on current microbiological scenario worldwide the possibility of transmitting multidrug resistant (MDR) organisms should be considered.

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Introduction

Multidrug-resistant Gram-negative bacterial infections in solid organ transplantation have been increasingly recognized in the past decade.[1] Most recently, XDR-resistant carbapenemase-producing *Klebsiella pneumoniae* (KPC-Kp) infection has emerged as a significant healthcare challenge, especially considering that few antibiotics are effective to treat it.[1] The incidence of donor-derived transmission of infections is <1% in solid organ transplants (SOTs), with morbidity and mortality rates of 40%.[2] This case report aims to address the challenges involved in prevention, detection and management of donor-derived infection by MDR organisms.

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Case report

A 59-year-old male patient with non-obstructive hypertrophic cardiomyopathy was hospitalized to manage cardiac insufficiency with inotropic therapy. The worsening of his clinical picture required the use of an intra-aortic balloon, and he was prioritized for cardiac transplantation. During the pre-transplant period, the patient did not develop any infectious complications. Blood cultures collected 24 and 48 h before transplantation were negative.

The patient underwent bicaval–bipulmonary heart transplantation, which required another surgery in the first 24 h to resolve hemostasis. He remained under mechanical ventilation with the use of vasoactive drugs and an intra-aortic balloon for 48 h, maintaining a good clinical condition. The immunosuppressive regimen included mycophenolate mofetil, cyclosporine and prednisone. On day 3 post-operation, a blood culture from the donor was positive for Gram-negative bacilli. As the donor was receiving piperacillin–tazobactam at the time of transplantation, the same drug was administered to the recipient. Although the recipient was clinically stable without signs of infection, blood cultures and surveillance cultures (from the groin and rectal areas) were collected. On day 5 post-operation, the final identification turned out ColR KPC-Kp.

Blood cultures from the recipient were positive for ColR KPC-Kp on day 7 post-operation with the same sensitivity profile as the donor isolate. The surveillance cultures were negative. At this time, the recipient was afebrile and hemodynamically stable, presenting with 26,000 cells/mm^3 leukocyte count and 6.5 mg/dL C-reactive protein (CRP) levels. Double-carbapenems (meropenem 2 g 8/8 h in a 4-h infusion combined with ertapenem 1 g/day, 1 h before one of the meropenem doses) and amikacin (15 mg/kg once a day) was started. The patient developed pericarditis on day 9 post-operation and required the drainage of 1000 mL of exudate; his pericardial fluid cultures were positive for ColR KPC-Kp. The patient underwent a surgical approach to clean the pericardium, requiring a repeated surgery eight days later; a sternum bone fragment collected at this time was positive for ColR KPC-Kp. On day 37 post-operation, he presented with worsening respiratory signs with chest computed tomography exhibiting consolidation with lung fluid levels suggestive of pulmonary cavitation due to necrosis (Fig. 1). This diagnosis was confirmed by histopathology of lung fragments obtained from lobectomy, in which a pulmonary abscess with liquefactive necrosis and necrotizing arteritis were noted. The patient developed septic shock, and antibiotic coverage was extended to linezolid and fluconazole while maintaining the initial treatment regimen for ColR KPC-Kp (double-carbapenem and amikacin). The patient progressed to multiple organ failure and died on day 50 post-transplantation.

The donor and other recipients

The donor was a 17-year-old male who died from traumatic brain injury after being hospitalized in the intensive care unit (ICU) for seven days having received piperacillin/tazobactam and vancomycin for two days. His blood cultures and surveillance cultures were negative at the time of donation. The culture that was positive for ColR KPC-Kp was collected from the splenic artery at the time of organ removal. The recipients of other organs (two kidneys) did not present any infectious complications or positive cultures and did not receive specific antimicrobial treatment. The liver was not used for transplantation.

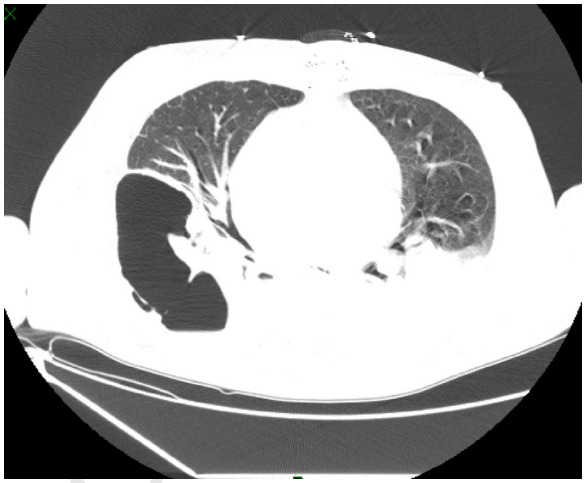

Fig. 1 – Chest TC consolidation with lung fluid levels suggestive of pulmonary cavitation due to necrosis.

Microbiological aspects

Bacterial identification was performed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF/MS) technology, using Vitek MS® system (bioMerieux, Marcy-I'Étoile, France), and the antimicrobial susceptibility profile was determined using the Vitek® 2 system (bioMerieux). The minimum inhibitory concentration (MIC) for polymyxin B was defined by broth microdilution (Probac, Brazil). The two strains evaluated (donor and recipient) exhibited high levels of resistance to cefepime, cefoxitin, ceftazidime, ceftriaxone (MIC ≥ 64 μg/mL), ertapenem (MIC ≥ 8 μg/mL), imipenem (MIC, 8 μg/mL), meropenem (MIC ≥ 16 μg/mL), ciprofloxacin (MIC, ≥4 μg/mL), gentamicin (MIC ≥ 16 μg/mL), and tigecycline (MIC, 4 μg/mL) following the breakpoints established by CLSI, 2016.[3] Resistance to polymyxin B (MIC, >64 μg/mL) was established using the EUCAST breakpoint, 2017.[4] Amikacin was the only susceptible drug (MIC, 4 μg/mL). The presence of the bla_{KPC} resistance gene was detected in all strains evaluated,[5] and the molecular gene identification using Sanger sequencing exhibited 100% homology with the enzyme KPC-2. The genetic similarity between strains was established by pulsed field gel electrophoresis (PFGE) using the *Spe*I restriction endonuclease, demonstrating an identical PFGE pattern (Pattern A) between the donor and recipient strain (Fig. 2). Multilocus sequence typing (MLST) analysis also identified the same sequence type (ST 437) from the donor and the recipient strains, and eBURST analysis (http://eburst.mlst.net) revealed that ST 437 belonged to clonal complex CC 258.

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