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ORIGINAL ARTICLE

Successful kidney transplantation from a donation after cardiac death donor with acute renal failure and bowel infarction using extracorporeal support

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Summary

As a result of the ever widening disparity between organ supply and demand, a resurgence of interest has occurred in kidney recovery from donation after cardiac death (DCD) donors. New techniques of *in situ* extracorporeal support offer the potential to reduce warm ischemic injury and optimize donor management prior to organ recovery. In addition, preliminary outcomes using kidneys from selected deceased donors with rising serum creatinine levels have been promising. However, contraindications to successful organ donation and transplantation may include the presence of abdominal compartment syndrome, generalized bowel infarction, refractory shock with profound metabolic and lactic acidosis, and acute anuric renal failure, particularly in the setting of DCD. We report herein the successful recovery and transplantation of kidneys from an unstable donor with the above constellation of conditions in the setting of extracorporeal support after declaration of death by asystole.

Introduction

As of August 1, 2008, the United Network for Organ Sharing (UNOS) national waiting list for organ transplantation included over 107 000 registrations, of which more than 81 000 were awaiting kidney transplantation (http://www.optn.org). Despite concerted efforts to increase the donor organ supply such as the Organ Donor Breakthrough Collaborative [1], the waiting list continues to grow disproportionately because of the relative shortage of donors and transplantable organs. Any increase in the number of donors or expansion of previous limits of acceptable donors may favorably impact the organ shortage but adversely affect transplant outcomes because current efforts to increase donor utilization target potential donors that historically were considered marginal [2]. Recent initiatives to increase the deceased donor organ pool have incorporated primarily the use of kidneys from expanded criteria donors (ECD) in the setting of donation after brain death (DBD) as well as the use of kidneys from donation after cardiac death (DCD) [1,3–5].

A recent surge of interest has been directed toward the use of organs from DCD donors [6]. However, the main drawback to DCD donation has been the variable period of warm ischemia that occurs prior to organ recovery. The interval from asystole to cross-clamp and organ perfusion (agonal phase) is a straightforward and measurable parameter, but warm ischemia that occurs before or during the withdrawal phase is much more difficult to quantify. It is well established that prolonged warm ischemia is associated with irreversible cell damage leading to either severe delayed graft function (DGF) or primary

nonfunction [7–9]. In the setting of DBD donation, warm ischemia may manifest as the development of acute renal failure (ARF) prerecovery and is usually related to hypoperfusion from shock or rhabdomyolysis [10,11]. Despite these concerns, there are many reports of DCD donor kidneys performing well in the long-term, comparable to DBD kidneys with respect to renal function and patient survival, especially when warm and cold ischemia times can be minimized [9,12–15]. In addition, a number of recent studies have reported good preliminary outcomes using kidneys from DBD donors with either elevated or rising terminal serum creatinine levels [16–18].

These positive reports notwithstanding, there are scarce data to support the use of kidneys from DCD donors that develop ARF prior to withdrawal of support measures. At least one study shows initial promise with ARF kidneys from donors after cardiac death, and others have shown positive results even with prolonged resuscitation times and Maastricht category II uncontrolled DCD donors [19–21]. Herein we report a case of successful kidney recovery and transplantation from a DCD donor who developed ARF and bowel infarction because of refractory shock from an untreated abdominal compartment syndrome prior to withdrawal of ventilatory support.

Case report

Donor history

The donor was a 20-year-old caucasian male with no significant past medical history who attempted suicide by hanging. He was discovered by his parents and immediately brought to the Emergency Department. Upon arrival, the patient was asystolic and underwent immediate intubation and resuscitation for a period of 20 min before the return of a normal sinus rhythm and measurable blood pressure. The amount of time that the patient was asystolic prior to arrival in the Emergency Department was uncertain. Despite management and resuscitative efforts in the Emergency Department, his Glasgow Coma Scale was never higher than 3 and he was transferred to the Medical Intensive Care Unit. Initial blood and urine testing were positive for alcohol and marijuana respectively.

Computerized tomographic imaging demonstrated poor gray-white matter differentiation with diffuse swelling, clinical examination revealed that both pupils were fixed and dilated, and Neurosurgery consultation indicated that the patient had suffered a nonsurvivable anoxic brain injury. However, the presence of spontaneous respirations prevented the immediate diagnosis of brain death. The patient's best prognosis was predicted to be a persistent vegetative state. After considering options, the family voluntarily decided to withdraw ventilatory support. At this time, the parents were approached by the local Organ Procurement Organization to discuss the option of organ donation after declaration of death by cardiac arrest. The parents voluntarily consented to DCD organ donation as well as the use of Extracorporeal Interval Support for Organ Retrieval (EISOR), which included a separate consent for placement of femoral arterial and venous cannulas prior to the withdrawal of ventilatory support.

During initial donor assessment (including blood typing, tissue typing, and serologic evaluation) and attempted placement of extrarenal organs, the donor became hemodynamically unstable despite the administration of intravenous fluids and vasopressors. The donor then developed severe abdominal distension and bladder-pressure monitoring revealed a pressure of 38 mmHg, confirming the clinical diagnosis of an acute abdominal compartment syndrome. A General Surgery consultation was obtained but a decision was made not to proceed with a decompressive laparotomy because of the patient's neurologic prognosis. Over a period of 6 h, the donor developed progressive acute renal failure with hypotension (mean arterial pressure <60 mmHg), profound metabolic and lactic acidosis (pH 6.98, lactate level 6.6 mM/l), coagulopathy (INR 3.28), oliguria/anuria (urine output <20 cc/h), and a rise in serum creatinine level from 1.6 to 4.0 mg/dl (Fig. 1). Additionally, the donor organs endured untreated abdominal compartment syndrome for 3–4 h.

After placement of femoral arterial and venous catheters with local heparinization only, and in accordance with the wishes of the family, ventilatory support measures were withdrawn. The donor arrested within 5 min of withdrawal of support measures and was declared dead by cardiac arrest after 5 min of monitored asystole. At this time, the donor was systemically heparinized and placed on EISOR with flows of 5 l/min as the donor was cooled to 22 °C. The donor was then transported nonurgently to the Operating Room at which time laparotomy resulted in immediate decompression of the abdominal cavity. Abdominal exploration revealed

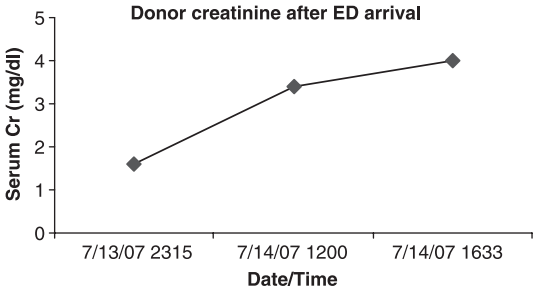

Figure 1 Donor serum creatinine after arrival in the emergency department (ED).

complete bowel infarction from the ligament of Treitz to the rectum. Although there was no evidence for bowel perforation, turbid peritoneal fluid and putrefaction were apparent.

The liver appeared somewhat mottled and a decision was made not to perform a hepatectomy because of its gross appearance, the prolonged instability of the donor (liver enzymes were in the 400–600 U/l range), and the concern for bacterial translocation resulting in both portal and surface contamination. However, because the kidneys were retroperitoneal, a decision was made to perform a bilateral donor nephrectomy with the plan of transplanting the kidneys if both the biopsy findings and pump parameters on the pulsatile perfusion preservation apparatus were acceptable. Similar to the technique used in DBD donors, we performed a standard retroperitoneal dissection, including skeletonization of the intra-abdominal aorta and vena cava with identification of both ureters. Each kidney was partially mobilized and time was taken to permit the EISOR circuit to 'resuscitate' the kidneys.

After approximately 90 min of total EISOR, 5 liters of cold Viaspan® (Belzer UW solution; Bard Laboratories, Pomona, NY, USA) were added to the EISOR circuit and rapidly infused through the femoral arterial cannula after cross-clamping of the supra-celiac aorta as the femoral venous cannula was used to exsanguinate the donor. Iced slush was placed topically on the kidneys. Both kidneys were then removed, washed with Betadine®, placed in cold Viaspan®, cleaned on the back table as the anatomy was verified, and upper pole wedge biopsies were performed for frozen section assessment.

The kidneys appeared grossly normal without evidence for petechiae or other obvious surface changes. The kidney biopsies did not show either cortical necrosis or disseminated intravascular coagulation; there was evidence for mild acute tubular necrosis but no evidence for any background parenchymal or vascular changes. Subsequently, the kidneys were placed on the pulsatile preservation pump (RM3 Renal Preservation System, Waters Medical Systems, Rochester, MN, USA) and perfused with Belzer's hypothermic machine perfusion solution (KPS-1; Organ Recovery Systems, Des Plaines, IL, USA) at 5 °C at an initial pressure of 50 mmHg. Both kidneys had excellent pump characteristics, with flows in the range of 180 ml/min and resistances of 0.11–0.14 mmHg/ml/min. Consequently, a decision was made to proceed with transplanting both the kidneys.

Recipient outcomes

Case 1

Recipient One is a 58-year-old caucasian female with a history of insulin-requiring Type 2 diabetes, obesity, coronary artery disease, and hypertension. She started peritoneal dialysis in January 2005 and then was switched to hemodialysis in September 2005 so that a panniculectomy could be performed. Secondary to problems with dialysis access and not tolerating hemodialysis treatments well, she was called in for a 'medically urgent' transplant in July 2007. Her peak and current panel reactive antibody (PRA) levels were 50% and 30%, respectively, and the donor and recipient were a six-antigen mismatch. However, T and B lymphocyte flow cross-match testing between the donor and recipient were compatible. Her transplant procedure was uneventful and total cold ischemia was 22.5 h with a pump time of 20 h as kidney reperfusion appeared normal. She received induction immunosuppression with a single intra-operative dose of alemtuzumab (30 mg intravenous) and high-dose intravenous immunoglobulin (IVIG, 2 gm/kg) followed by maintenance therapy with mycophenolate mofetil (MMF), the delayed administration of tacrolimus, and tapered steroids. She was placed on vancomycin and ciprofloxacin for peri-operative prophylaxis for 4 days because of the donor history of bowel infarction. The patient experienced DGF but otherwise had an uncomplicated postoperative course and was discharged on the 5th postoperative day on planned hemodialysis.

A kidney biopsy performed at 12 days following transplantation because of DGF revealed severe acute tubular necrosis and minimal intimal arteritis (Banff 1997 IIA) so the patient was readmitted for treatment with five doses of Thymoglobulin, dexamethasone, and high-dose IVIG (2 gm/kg). Following this admission, the patient no longer required dialysis (3 weeks post-transplant) and her serum creatinine level declined to <3.0 mg/dl on day 25. A follow-up biopsy in September 2007 revealed complete resolution of both acute tubular necrosis and intimal arteritis, but a clinically indicated biopsy in April 2008 showed focal acute tubular injury and early chronic changes. No C4d deposition was identified in any of these biopsies.

To date, the patient has had two further hospital readmissions; one for rehydration following several days of diarrhea (that prompted the clinically indicated biopsy for a rise in serum creatinine level from 1.1 to 1.9 mg/dl) and the other for treatment of asymptomatic cytomegalovirus (CMV) viremia identified by polymerase chain reaction (PCR) with a viral load of 33,600 copies/ml in December 2007. The patient was at risk for primary CMV exposure (donor CMV seropositive, recipient CMV seronegative) and was receiving 6 months of low-dose valganciclovir prophylaxis (450 mg every Monday, Wednesday, and Friday because of leukopenia) before developing evidence for CMV viremia at 5 months. The valganciclovir dose was increased to 900 mg twice daily for 1 month, then 900 mg once daily for 2 months, and

then 450 mg daily for 4 months. Serial CMV PCR titers revealed resolution of viremia within 1 month of valganciclovir dose optimization. At 1 year following transplantation, valganciclovir was stopped because of severe leukopenia.

By 7 weeks post-transplantation, the patient reached her new steady state serum creatinine level of 1.3 mg/dl, corresponding to a glomerular filtration rate (GFR) of 45 ml/min by abbreviated Modification of Diet in Renal Disease (MDRD) calculation. Urine protein excretion at 1 year was <100 mg/24 h. She has remained at this level of renal function through 18 months of follow-up (Figs 2 and 3) and is currently doing well on triple maintenance immunosuppression consisting of oral tacrolimus (12-h target trough levels 8–10 ng/ml), MMF 500 mg twice daily, and prednisone 5 mg daily. No additional renal allograft biopsies have been performed, and no further immunosuppressive dose reduction is planned because of a history of a high PRA, six-antigen mismatch, DGF, biopsy-proven early acute rejection, and the administration of half-dose MMF because of neutropenia.

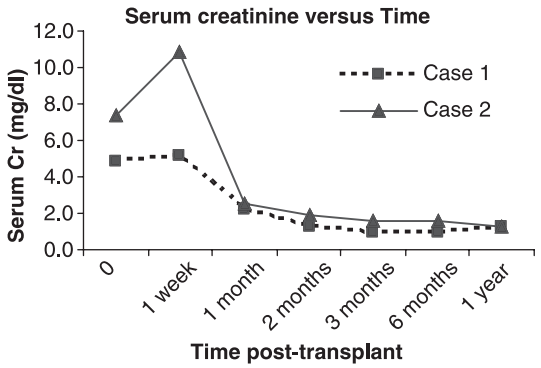

Figure 2 Serum creatinine levels in both recipients from the time of transplant through 1 year of follow-up.

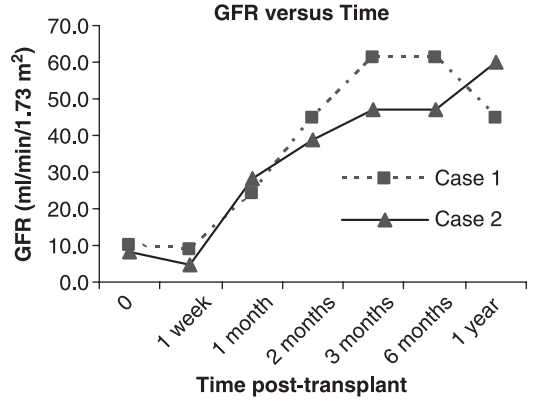

Figure 3 Glomerular filtration rates in both recipients from the time of transplant through 1 year of follow-up.

Case 2

Recipient Two is a 59-year-old caucasian male with a history of low-grade transitional cell bladder cancer, end-stage renal disease caused by IgA nephropathy, and hemodialysis for 4 years. His PRA level was 0%, the donor and recipient were a four-antigen mismatch, and final T and B cell flow cross match were compatible. He received induction immunosuppression with a single intra-operative dose of alemtuzumab (30 mg intravenous) followed by maintenance therapy with MMF, delayed tacrolimus, and tapered steroids. His transplant procedure was uneventful and he was placed on vancomycin and ciprofloxacin for peri-operative prophylaxis for 4 days because of the donor history of bowel infarction. Total cold ischemia time was 17 h with a pump time of 14 h as kidney reperfusion appeared normal. The patient experienced DGF but otherwise had an uncomplicated postoperative course and was discharged on the 6th post-operative day on planned hemodialysis.

A kidney biopsy performed at 12 days because of DGF revealed acute tubular necrosis yet the patient became dialysis-free at 2 weeks following transplantation. He reached a serum creatinine level <3.0 mg/dl on day 25 and was subsequently weaned off of steroids completely at 1 month. He achieved a baseline serum creatinine level of 1.3 mg/dl (GFR 60 ml/min) at 3 months that has remained stable up to 18 month follow-up (Figs 2 and 3). No other biopsies have been performed. He is currently on oral tacrolimus (target 12-h trough levels of 6–8 ng/ml) and MMF (500 mg twice daily) dual therapy because he is over 60 years of age, is low risk with a 0% PRA, and has not had any episodes of acute rejection. He has not had any hospitalizations since the transplant, is working full-time, and has been transferred back to the care of his nephrologist.

Discussion

The burgeoning crisis in organ supply fuels initiatives to expand the limited deceased donor pool. DCD donors offer an innovative approach for increasing the organ pool. In the United States (US), DCD donors account for a small, but growing proportion of deceased donors [1,5]. According to UNOS data from 2000 through 2005, the total number of deceased donor kidney transplants increased by 22% in the US, whereas the number of DCD donor kidney transplants increased by 361% during this same time period. From 2005 to 2006, the actual number of DCD donors in the US increased 16% from 560 to 647 [6].

Most reports of transplantation from DCD donors are exclusive to kidney transplantation and report high rates of DGF [12–15]. DGF, usually defined as the need for dialysis in the first week following kidney transplantation, is a form of ARF following kidney transplantation that results in oliguria, enhanced allograft immunogenicity, and decreased medium-term graft survival [9,22]. It is well-established that DGF is a risk factor not only for graft dysfunction, acute rejection, and poorer graft survival but is also directly related to deceased donor age and category [9,14]. According to UNOS data, the incidence of DGF is highest with DCD kidneys (44%), intermediate with DBD/ECD kidneys (33%), and lowest with DBD standard criteria donor kidneys (21%) [1,6]. The presence of DGF is an early marker of organ quality and preservation that represents a combined response to a series of ischemic, reperfusion, inflammatory, and immunologic injuries [9].

The incidence of DGF has not changed appreciably in the last decade and the major risk factors for DGF appear to be warm and cold ischemia, which account for the hierarchy of DGF among the various donor categories [8,9,13,22]. Warm ischemia is more deleterious to initial organ function than cold ischemia and the requisite warm ischemia inherent in DCD organ donation has been implicated as the major causative factor for the high rate of DGF within this donor category [7–11]. Other risk factors for DGF include donor, procurement, preservation, transplant, and recipient issues [9]. It is important to note, however, that unlike DBD kidney transplantation in which DGF is a known risk factor for reduced medium-term graft survival, DGF does not necessarily portend a poor prognosis after DCD donor kidney transplantation [5,14]. Perhaps the difference in this unusual finding is the requisite terminal warm ischemia characteristic of DCD donor kidneys, which is more than likely responsible for DGF and may be more reversible than preterminal warm ischemic injury that results in DGF in the DBD donor setting.

However, prolonged warm ischemia is associated with reduced graft survival irrespective of donor category [7–11]. EISOR and other strategies designed to minimize warm ischemia might improve initial function of DCD donor kidneys, lower rates of DGF, and permit extrarenal organ recovery and transplantation from DCD donors [5]. EISOR may become the preferred method of managing DCD donors (as well as unstable DBD donors) because it not only minimizes warm ischemia but permits withdrawal of ventilatory support with the option of having the family at the bedside, nonurgent multi-organ recovery using a standard dissection, core cooling during the organ recovery process, and a rapid *in situ* flush and exsanguination of the organs after preservation solution is added to the circuit.

A number of recent reports have demonstrated good short-term outcomes when transplanting kidneys from selected DBD donors with either elevated or rising serum creatinine levels or ARF [16–18,23]. Although the incidence of DGF (28–88%) was high in these studies, excellent outcomes, with comparable renal function and graft survival to DBD standard criteria donor kidneys were noted. However, to our knowledge, the successful use of DCD donor kidneys in the setting of prerecovery ARF, profound metabolic and lactic acidosis, and shock caused by untreated abdominal compartment syndrome resulting in complete bowel infarction has not been previously reported. Although the donor described herein appeared to have a number of contraindications to successful organ donation, we believe that the use of EISOR permitted optimal 'resuscitation' and recovery of the kidneys by preventing ongoing warm ischemia that was already apparent prior to the withdrawal of ventilatory support. It is interesting to speculate not only on the role but the ethics and timing of decompressive laparotomy for the abdominal compartment syndrome in a patient in whom the family has already made the decision to withdraw support measures and consequently has been designated as 'do not resuscitate' but remains a potential 'donor'.

Donor organs can serve as vehicles for the transmission of infection, and the donor described herein had a number of risk factors for and manifestations of sepsis. Untreated abdominal compartment syndrome can lead not only to bowel infarction but bacteremia because of bacterial translocation and peritonitis resulting from perforation and ischemic necrosis. Although we did not identify bowel perforation at the time of organ recovery, turbid peritoneal fluid and putrefaction were apparent. The liver was mottled and we were concerned about portal and surface bacterial contamination of the intra-abdominal organs. However, because the kidneys are retroperitoneal and nonportal in location, we believed that the risk for transmission of infection by kidney transplantation was lower and perhaps more amenable to systemic antibiotic prophylaxis in the donor as well as circulating antibiotics in the EISOR circuit and pulsatile perfusion pump. In addition, at the time of nephrectomy, the kidneys were washed in Betadine® to reduce the risk of surface contamination prior to placement on the perfusion pump. Although it is unclear whether flushing with and pumping antibiotics through the kidneys can reduce the risk of infectious disease transmission, it does afford the opportunity to deliver relatively high local levels of antibiotics during organ preservation. Moreover, in contrast to our standard antibiotic prophylaxis that consists of a first-generation cephalosporin intra-operatively and postoperatively for 24 h, we elected to use

empiric expanded coverage (vancomycin and ciprofloxacin) for a longer duration of time (4 days) in both kidney transplant recipients to further reduce the risk for bacterial transmission.

In addition to risk for transmitting infection, this donor was considered 'high risk' because of shock, terminal ARF, and DCD donation, each of which could result in primary nonfunction. Because appropriate recipient selection is paramount to optimizing outcomes in transplantation, we intentionally chose recipients who were either high risk or medically urgent yet whom we believed could tolerate a prolonged period of DGF because of the unique circumstances associated with this donor. The first recipient had been on dialysis for over 2 years but was not doing well on dialysis, had limited dialysis access, had multiple co-morbidities, and was flow cross-match compatible in spite of a PRA level of 30–50%. The second recipient had been on dialysis for over 4 years and had a history of bladder cancer. Both patients accepted the organs with informed consent, and were specifically apprised of the unique risks of infection and DGF associated with this particular donor. However, because of the fact that the background of the terminal events in this case was an otherwise ideal donor, we believed that it was reasonable to proceed with transplantation provided that the EISOR circuit functioned well and that the kidneys were anatomically and histopathologically normal, flushed well, and pumped well.

Proper management and assessment of DCD donors, optimal methods of recovery and preservation, abrogation of ischemia/reperfusion injury, prevention of disease transmission, and appropriate recipient selection remain key issues in DCD organ transplantation. While there are good initial data supporting the use of kidneys from selected DBD donors with pretransplant ARF, data of the same magnitude are not available with DCD donor kidneys. We believe that the use of extracorporeal support provides a new method of minimizing ischemia and optimizing organ protection during the donation process, even in cases with multiple risk factors for organ damage.

Authorship

JMZ, RJS, RPS and MHH: designed research. JMZ, RJS, RPS and MHH: performed study. ACF and JR: contributed important reagents. JMZ, RJS and RPS: collected data. JMZ, RJS and RPS: analyzed data. JMZ, RJS, ACF and JR: wrote paper.

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