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ORIGINAL ARTICLE

The benefits of initiating continuous renal replacement therapy after brain death in organ donors with oligoanuric acute kidney injury

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Abstract

Acute kidney injury (AKI) in deceased organ donors is increasing due to the escalation in anoxic brain-deaths. The management of an organ donor with oligoanuric AKI is frequently curtailed due to hemodynamic and electrolyte instability. Although continuous renal replacement therapy (CRRT) corrects the effects of AKI, it is rarely started after the diagnosis of brain-death (BD). Since 2017, we have initiated CRRT in organ donors with oligoanuric AKI to allow more time to stabilize the donor and improve the function of the thoracic organs. We now report our experience with the first 27 donors with oligoanuric AKI that received CRRT after the diagnosis of BD, with organs transplanted as the primary outcome. The average duration of CRRT was 30.1 ± 14.4 h and the mean ultrafiltration volume was 5141 ± 4272 ml. The time from BD declaration to cross clamp was significantly longer in the CRRT group versus a historical cohort with oligoanuric AKI that was not dialyzed (62.8 ± 18.3 vs. 37.1 ± 14.9 h; $P < .01$). The mean number of total organs transplanted per donor in the CRRT group was greater than the historical cohort, 2.9 ± 1.7 vs. 1.4 ± .6 ($P = .< 01$), respectively. The mean number of thoracic organs transplanted per donor also increased between the two groups, 1.4 ± 1.2 versus .6 ± .9 ($P = .02$). Thirty-seven percent of the kidneys were successfully transplanted with a mean serum creatinine of 1.4 mg/dl at 6 months. We suggest that OPOs consider starting CRRT in organ donors with oligoanuric AKI to possibly increase the number of organs transplanted.

KEYWORDS
acute kidney injury, anuria, brain death, continuous renal replacement therapy, dialysis, oligoanuria, organ donor, organ recovery center

1 | INTRODUCTION

In the United States, more than 90 000 patients are on the kidney transplant waitlist with a median wait time of greater than 3 years.[1] In 2020, there were 17 583 kidney transplants from deceased donors, demonstrating the enormous imbalance between donors and transplant candidates. The cause of death of deceased organ donors has changed over the last 5 years, with trauma and stroke remaining relatively stable but anoxia increasing from 38% to 47% nationally.[1] The increase in anoxic brain death (BD) is driven primarily by the opioid epidemic and drug overdose.

Brain Death is initially associated with vasoconstriction and then prolonged hypotension which can lead to renal ischemia and acute kidney injury (AKI). In addition to these harmful effects, the anoxic BD

donor has suffered from either severe hypoxemia, shock, or cardiac arrest. These cause irreversible death of the brain tissue and frequently additional ischemic damage to the kidney contributing to AKI. It is estimated that as many as 24%–36% of BD donors experience AKI.[2,3] The proportion of deceased donors with any AKI was 37% in 2020, and the proportion with Stage 3 AKI was 12%, but it is unclear of the proportion of AKI with oligoanuria in this population.[4]

After the declaration of BD, the organ procurement organization (OPO) is responsible for donor management to maximize the number of organs transplanted. AKI with hyperkalemia, acidosis, and fluid retention from oligoanuria can cause significant difficulties in managing the donor, including arrhythmias, intractable shock, and pulmonary edema. If the donor remains unstable, the OPO may curtail donor management and expedite organ procurement, which limits the number of organs transplanted, particularly thoracic organs.

Although dialysis or continuous renal replacement therapy (CRRT) is a standard of care for severe AKI in critical patients, it is rarely started after the diagnosis of BD in potential organ donors.[5] This is due to logistics, lack of equipment in smaller hospitals, cost, and the notion that it is futile in a deceased patient. The limited use of CRRT in this population may also be due to a lack of any descriptive benefit or supporting evidence in the literature. Since 2017, with the increase in AKI in organ donors, we started utilizing CRRT in our organ recovery center (ORC) in donors with severe electrolyte disturbances, metabolic acidosis, volume overload, and oligoanuria. Our primary objective was to stabilize the donor and increase the time for donor management. We surmised that the extended time in the intensive care unit (ICU) would allow us to improve the function of the heart and lungs, with the goal of transplanting more organs. We now report on our retrospective experience with the first 27 donors with oligoanuric AKI in which CRRT was initiated after BD in our ORC.

2 | MATERIALS AND METHODS

Mid-America Transplant is an OPO that has an independent ORC that includes a six-bed ICU, laboratory, CT scanner, and surgical suites. Ninety-five percent of BD donors are transferred to the ORC within 8 h of authorization for donation. To optimize the function of all transplantable organs, donor management included invasive hemodynamic monitoring, ventilatory support, and aggressive diagnostic and therapeutic interventions. The primary vasopressor used was norepinephrine with a target mean arterial pressure greater than 65 mm Hg. An aggressive lung donor management protocol, utilizing a lung protective strategy, was employed on all potential lung donors.[6] All donors received 300 mg hydrocortisone intravenously and then 100 mg every 8 h. Enoxaparin 40 mg subcutaneously daily was used for deep venous thrombosis prophylaxis if dialysis was not started. Heparin 500–1000 units per hour intravenously was used for anticoagulation while on dialysis. Antibiotics were used for identified infections with the dose adjusted for renal failure and dialysis.

2.1 | CRRT group

Between January 2017 and June 2021, all BD organ donors in Mid-America Transplant's ORC with AKI were eligible for the study. Oligoanuria was defined as urine output ≤ 600 ml/24 h. All donors underwent a stroke volume-based fluid resuscitation protocol over 4 h to optimize cardiac output and to wean off vasopressors.[7] Donors with oligoanuric AKI after fluid resuscitation were treated with furosemide 40–120 mg intravenously. Donors with persistent hyperkalemia, acidosis, fluid overload and/or anuria were considered for CRRT (CRRT group) under the direction of an experienced nephrologist (M.R.). A femoral dialysis catheter (Power-Trialysis Short-term Straight Dialysis catheter, Bard Access Systems, Inc., Salt Lake City, UT, USA) was inserted by the ICU nurse. CRRT was started utilizing the NxStage System One (NxStage Medical, Inc., Lawrence, MA, USA) in the continuous venovenous hemodialysis mode with the CAR-170 Renaflow II hemofilter pre-attached to bloodlines, with an effluent rate of 35–45 ml/kg/h, blood flow rate 300–350 ml/h, and with ultrafiltration rates ranging from 100 to 1000 ml/h. The dialysis solution was tailored to the donor's potassium and calcium values, and the range of dialysate was between 2 to 5 L/ h depending upon the clearance required. Ultrafiltration rates varied depending on the volume status of the donor, and was well tolerated with minimal adjustments in vasopressor support and/or intravenous albumin. CRRT was continued until the electrolyte abnormalities and acidosis were corrected and euvolemia achieved with fluid removal. CRRT was then maintained to avoid a recurrence of the electrolyte abnormalities and fluid overload, and was discontinued shortly before procurement.

2.2 | Cohort groups

To evaluate the benefit of CRRT in terms of organs transplanted per donor, two groups of donors with AKI were identified retrospectively for comparison. The first group was a historical oliguric cohort with severe AKI and oligoanuria that was not treated with dialysis. All BD organ donors, greater than 12 years old, managed in our ORC from January 1, 2015, through December 31, 2017, with an initial creatinine < 2.0 mg/dl on arrival to the hospital, a peak creatinine > 3.0 mg/dl, and a total urine output < 600 ml in the last 24 h prior to procurement were included in the historical oliguric cohort.

The second group was a contemporaneous nonoliguric cohort of 27 BD donors with AKI that did not require CRRT. Inclusion criteria for this comparative group were BD donors greater than 12 years old managed in our ORC between January 2017 and June 2021, inclusive, with an initial creatinine less than 2.0 mg/dl and a peak creatinine greater than 3.0 mg/dl, that did not receive dialysis. Forty-one donors fulfilled this criteria. The 27 donors with the highest peak creatinines were chosen as the comparison group to equal the number and severity of donors in the CRRT group.

2.3 | OPTN data

Outcome data including the 30-day and 6-month post-transplant heart and lung graft survival rates from our donors was obtained from an Organ Procurement and Transplantation Network (OPTN) query as of April 8, 2022. To obtain national data on organs transplanted from donors with oligoanuric AKI, the OPTN was queried on January 14, 2022. All national data was from brain-dead organ donors between January 1, 2017, and June 30, 2021, with an initial creatinine < 2.0 mg/dl, peak creatinine > 3.0 mg/dl and less than 600 ml of urine output in 24 h prior to cross-clamp.

2.4 | Measurements

To assess comparability, the CRRT group, historical oliguric cohort and contemporaneous nonoliguric cohort were analyzed based on cause of death, time from admission to BD, time from BD to cross clamp, fourteen demographic and social factors, and nine initial laboratory values. The initial partial pressure of arterial oxygen (PaO_2) on arrival to the ORC and the final PaO_2 prior to procurement were measured. All PaO_2 measurements were on a fraction of inspired oxygen (FiO_2) 1.0 and positive end-expiratory pressure (PEEP) 5 centimeters of water. The difference between the initial and final PaO_2/FiO_2 (P/F) was measured as a surrogate for the clearance of pulmonary edema with ultrafiltration. Echocardiography was performed on all heart-eligible donors, and the ejection fraction (EF) from the first and last scans in the ORC were recorded. The number and type of organs transplanted from each group were recorded.

The duration of CRRT, blood flow rate, effluent rate and ultrafiltration rate were recorded. Kidney biopsies were at the discretion of the transplant surgeon. All biopsies were wedge biopsies and read by a renal transplant pathologist. All kidneys that underwent CRRT and were transplanted were biopsied. Biopsy data including acute tubular necrosis (ATN), glomerulosclerosis (GS), interstitial fibrosis and tubular atrophy (IFTA) and cortical necrosis (CN) were recorded for each kidney. Kidneys were either preserved in static cold storage or pumped with hypothermic (4°C) preservation fluid using a LifePort Kidney Transporter (Organ Recovery Systems, Itasca, IL, USA). Kidney recipient outcome data including graft function and serum creatinine at hospital discharge and at 6 months was obtained from an OPTN query in November 2021.

AKI was defined using the Acute Kidney Injury Network (AKIN) criteria as follows: Stage 1, increase in serum creatinine from admission to the terminal value by ≥.3 mg/dl or 1.5- < 2-fold; Stage 2, 2- < 3-fold; and Stage 3, ≥3-fold, or terminal serum creatinine ≥4.0 mg/dl after a rise of ≥.5 mg/dl from admission or dialysis support.[8] All of the donors in the cohort groups had AKIN Stage 3 AKI.

Since the donors were deceased, the study represented non-human study research and therefore Institutional Review Board approval was not required. The study was approved by the Medical Advisory Board of Mid-America Transplant. Consent from legal next-of-kin for research prior to procurement was obtained for all donors that received CRRT.

2.5 | Statistical analyses

Data were analyzed using IBM SPSS Statistics for Windows, version 28.0.1.0 (IBM Corp., Armonk, NY, USA). The distribution shapes of variables measured on interval or ratio scales were examined using frequency tables and histograms. Outcomes that were normally distributed as determined by the D'Agostino-Pearson test for normal distribution were analyzed using the t-test. Non-normally distributed variables were analyzed using the Mann Whitney U Test. Categorical variables were analyzed using the chi-square test. When observed frequencies in a cell were less than five, Fisher's exact test was used instead of the chi-square test. All differences were examined using two-tailed tests. Unless otherwise stated, the measure of spread of the mean values is the standard deviation.

3 | RESULTS

3.1 | Comparison of CRRT and cohort groups

During the study period 27 out of 764 (3.5%) BD donors received CRRT. The mean age of the CRRT group was 32.0 ± 11.2 years, 59.3% were male, and 29.6% were African-American. The cause of death was trauma in 26% of the donors and anoxia in 67%, consistent with the drug use history in 70% of the donors. Demographic and social factors are displayed in Tables 1A and 1B. The historical oliguric cohort only had 14 donors over a 3-year span that fulfilled the inclusion criteria. Compared to the CRRT group, the historical oliguric cohort was older and had more donors with diabetes and hypertension (Table 1A). The contemporaneous nonoliguric cohort was also older and had a higher BMI than the CRRT group (Table 1B) but otherwise was comparable. The cause of death and drug use history among the three groups were similar.

The mean initial creatinines on admission to the hospital in the historical oliguric cohort, contemporaneous nonoliguric cohort, and CRRT group were 1.4 ± .4 mg/dl, 1.4 ± .3 mg/dl and 2.1 ± 1.4 mg/dl, respectively (Tables 2A and 2B). The mean peak creatinines in the three groups were 5.5 ± 2.0 mg/dl, 5.4 ± 1.6 mg/dl and 6.1 ± 2.3 mg/dl, respectively. Tables 2A and 2B display the other initial laboratory tests after declaration of BD and admission to the ORC for the three groups. Compared to the CRRT group, the historical oliguric cohort had a lower mean serum bicarbonate, but no difference in pH or serum lactate (Table 2A). Compared to the contemporaneous nonoliguric cohort, the CRRT group had greater mean blood urea nitrogen, serum potassium and lactate levels, but no significant difference in pH or serum bicarbonate (Table 2B).

3.2 | CRRT metrics

The mean time to start CRRT after brain-death was 24.6 ± 9.4 h. The average duration of CRRT was 30.1 ± 14.4 h. CRRT was

TABLE 1A Demographic and social factors in the CRRT and cohort groups: Historical oliguric cohort and CRRT group

	Historical oliguric cohort [a]	CRRT group[b]	
	% or Mean (SD)	% or Mean (SD)	Statistical tests
Number of donors	(*n* = 14)	(*n* = 27)	
Time (h) from admission to brain death[c]	88.0 (144.4)	61.2 (52.4)	*P* = .65
Time (h) from brain death to cross-clamp[d]	37.1 (14.9)	62.8 (18.3)	*P* < .01
Age (years)	46.9 (13.7)	32.0 (11.2)	*P* < .01
Gender	Male: 6 (42.9%)	Male: 16 (59.3%)	*P* = .32
	Female: 8 (57.1%)	Female: 11 (40.7%)	
Race			
African American	6 (42.9%)	8 (29.6%)	
Caucasian	7 (50.0%)	18 (66.7%)	
Hispanic		1 (3.7%)	
Other	1 (7.1%)	0%	
Race, Caucasian[e]	7 (50.0%)	18 (66.7%)	*P* = .30
BMI (kg/m^2)	33.0 (13.4)	27.8 (7.2)	*P* = .11
History of diabetes	7 (50%)	3 (11.1%)	*P* = .02
History of hypertension	9 (64.3%)	5 (18.5%)	*P* = .03
History of cocaine use	5 (35.7%)	10 (37.0%)	*P* = .93
History of IV drug use	0	6 (22.2%)	*P* = .08
History of other drug use	8 (57.1%)	19 (70.4%)	*P* = .40
Behavioral risk criteria[f]	5 (35.7%)	9 (33.3%)	*P* = .88
HIV serology positive	0	0	NA
Hepatitis B serology positive	1 (7.1%)	0	NA
Hepatitis C serology positive	1 (7.1%)	3 (11.1%)	*P* = 1.00
Cause of death			
Anoxia	9 (64.3%)	18 (66.7%)	*P* = .11
Stroke	4 (28.6%)	2 (7.4%)	
Trauma	1 (7.1%)	7 (25.9%)	

prescribed at an effluent rate of 30–45 ml/kg/h and hourly ultrafiltration rates of 100–1000 ml/h, with the higher removal rate in subjects with severe volume overload and anasarca. The total mean ultrafiltration volume was 5141 ± 4272 ml. There were no complications noted with the insertion of the dialysis catheter or clinical adverse events with CRRT.

3.3 | Donor management time

As suspected, the time from BD declaration to cross clamp was significantly longer in the CRRT group versus the historical oliguric cohort (62.8 ± 18.3 vs. 37.1 ± 14.9 h; *P* < .01), which allowed for a delay in procurement until the donor was more stable and heart and lung function potentially improved. Compared to the contemporaneous nonoliguric cohort, the time from BD declaration to cross clamp in the CRRT group was also significantly longer but to a lesser extent (62.8 ± 18.3 vs. 50.2 ± 16.7 h; *P* = .01).

3.4 | Organs transplanted

There were 79 organs transplanted from the 27 donors in the CRRT group (2.9 ± 1.7 organs/donor), 20 organs transplanted from 14 donors in the historical oliguric cohort (1.4 ± .6 organs/donor; *P* < .01), and 84 organs transplanted from 27 donors in the contemporaneous nonoliguric cohort (3.1 ± 1.5 organs/donor) (Tables 3A and 3B). Livers were transplanted at the same rate in all three groups. Compared to the historical oliguric cohort, the CRRT group transplanted more kidneys, lungs and hearts. In the CRRT group, 44.4% of the hearts and 48.1% of the lungs were transplanted, compared to 14.3% and 21.4%, respectively, in the historical oliguric cohort (Table 3A). There were twice as many thoracic organs transplanted in the CRRT group as in the historical oliguric cohort, 1.4 ± 1.2 vs. .6 ± .9 organs/donor, respectively (*P* = .02). There was no difference in the number of thoracic organs transplanted between the CRRT group and the contemporaneous nonoliguric cohort (Table 3B). The 6-month post-transplant heart graft survival rate in both the CRRT and contemporaneous nonoliguric

TABLE 1B Demographic and social factors in the CRRT and cohort groups: Contemporaneous nonoliguric cohort and CRRT group

	Contemporaneous nonoliguric cohort[g]	CRRT group[b]	
	% or Mean (SD)	% or Mean (SD)	Statistical test
Number of donors	(*n* = 27)	(*n* = 27)	
Time (h) from admission to brain death[c]	106.0 (191.1)	61.2 (52.4)	*P* = .74
Time (h) from brain death to cross-clamp[d]	50.2 (16.7)	62.8 (18.3)	*P* = .01
Age (years)	36.3 (6.7)	32.0 (11.2)	*P* = .04
Gender	Male: 11 (40.7%)	Male: 16 (59.3%)	*P* = .17
	Female: 16 (59.3%)	Female: 11 (40.7%)	
Race			
African American	9 (33.3%)	8 (29.6%)	
Caucasian	16 (59.3%)	18 (66.7%)	
Hispanic	1 (3.7%)	1 (3.7%)	
Other	1 (3.7%)	0%	
Race, Caucasian[e]	16 (59.3%)	18 (66.7%)	*P* = .57
BMI (kg/m^2)	33.0 (8.9)	27.8 (7.2)	*P* = .02
History of diabetes	5 (18.5%)	3 (11.1%)	*P* = .70
History of hypertension	10 (37.0%)	5 (18.5%)	*P* = .13
History of cocaine use	7 (25.9%)	10 (37.0%)	*P* = .38
History of IV drug use	6 (22.2%)	6 (22.2%)	*P* = 1.00
History of other drug use	17 (63.0%)	19 (70.4%)	*P* = .56
Behavioral risk criteria[f]	7 (25.9%)	9 (33.3%)	*P* = .55
HIV serology positive	0	0	NA
Hepatitis B serology positive	0	0	NA
Hepatitis C serology positive	4 (14.8%)	3 (11.1%)	*P* = 1.00
Cause of death			
Anoxia	20 (74.1%)	18 (66.7%)	
Stroke	5 (18.5%)	2 (7.4%)	
Trauma	2 (7.4%)	7 (25.9%)	*P* = .12

Abbreviations: AKI, acute kidney injury; BD, brain-dead; CRRT, continuous renal replacement therapy; BMI, body mass index; IV, intravenous; kg, kilogram; m, meter; HIV, human immunodeficiency virus; ORC, organ recovery center; SD, standard deviation.

[a]This cohort is a historical group of BD donors with oligoanuric AKI managed at the ORC between January 1, 2015, and December 31, 2017, that did not receive CRRT.

[b]CRRT group is the group of BD donors with oligoanuric AKI managed at the ORC that underwent CRRT between January 1, 2017, and June 30, 2021.

[c]Time from admission to the hospital to declaration of brain death.

[d]Time from declaration of brain death at the hospital to cross-clamp at the organ recovery center.

[e]Caucasian versus non-Caucasian donors.

[f]Public Health Service behavioral factors that increase the risk for HIV, Hepatitis B, and Hepatitis C transmission with organ donation.

[g]This cohort is a concurrent group of BD donors with non-oligoanuric AKI managed at the ORC between January 1, 2017, and June 30, 2021, that did not receive CRRT.

cohorts was 100%. The 30-day post-transplant lung graft survival rate was also 100% in both groups. There was insufficient data to analyze the 6-month lung graft survival rate in the CRRT donors.

3.5 | Kidneys transplanted

Twenty kidneys from ten donors (37%) were transplanted from the CRRT group and 25 kidneys from 13 donors (46%) were transplanted from the contemporaneous nonoliguric cohort group (Tables 4A and 4B). The mean creatinine at discharge from the transplant center was 6.5 ± 3.5 mg/dl and 6.7 ± 2.8 mg/dl for the contemporaneous nonoliguric cohort and CRRT groups, respectively. The mean creatinine at 6 months after transplantation was 1.4 ± .3 mg/dl and 1.4 ± .6 mg/dl in the contemporaneous nonoliguric cohort and CRRT groups, respectively. There were only 3 kidneys transplanted from two donors (11%) in the historical oligoanuric cohort.

TABLE 2A Initial laboratory values after the declaration of brain death and on arrival to the organ recovery center for the CRRT and cohort groups: Historical oliguric cohort and CRRT group

	Historical oliguric cohort [a]	CRRT group[b]	
	% or Mean (SD)	% or Mean (SD)	Statistical test
Number of donors	($n = 14$)	($n = 27$)	
Admission creatinine (mg/dl)[c]	1.4 (.4)	2.1 (1.4)	$P = .16$
Peak creatinine (mg/dl)	5.5 (2.0)	6.1 (2.3)	$P = .43$
Blood urea nitrogen (mg/dl)	46.5 (24.1)	54.9 (29.7)	$P = .65$
Sodium (mEq/L)	143.9 (13.1)	138.8 (7.8)	$P = .13$
Potassium (mEq/L)	4.3 (1.1)	5.0 (1.1)	$P = .08$
Serum bicarbonate (mEq/L)	18.7 (3.2)	21.3 (3.7)	$P = .03$
pH	7.4 (.1)	7.4 (.1)	$P = .73$
Calcium (mg/dl)	7.6 (1.3)	7.8 (1.0)	$P = .60$
Phosphate (mg/dl)	4.6 (2.9)	5.5 (1.9)	$P = .15$
Lactate (mmol/L)	3.2 (1.9)	3.8 (3.5) [$n = 25$]	$P = .95$
PaO_2/FiO_2 (initial)[d]	276 (161)	263 (137)	$P = .80$
PaO_2/FiO_2 (final)[e]	281 (142)	402 (172)	$P = .03$
Cardiac ejection fraction (EF) (initial) (%)	51.0 (8.2) [$n = 5$]	49.6(13.8) [$n = 20$]	$P = .83$
Initial EF < 55%[f]	2 (40%)	11 (55%)	$P = .65$
Initial EF < 55%, final EF ≥ 55%[g]	1 (50%)	6 (54.5%)	

3.6 | Secondary outcomes

There was no difference in the initial P/F among the three groups. The final P/F was significantly higher in the CRRT group than the historical oliguric cohort, suggesting a possible decrease in lung water with ultrafiltration or an improvement in atelectasis with an aggressive lung donor protocol. Five of the thirteen (38%) lung donors in the CRRT group had an initial P/F < 300, and a final P/F > 380 (initial mean P/F 180 ± 67, final mean P/F 453 ± 80). Five of 12 lung donors (42%) in the contemporaneous nonoliguric cohort also increased the P/F ratio from an initial mean P/F 248 ± 56 to a final mean P/F 467 ± 75. The median time between the first and last P/F was 36 (IQR 24–43) h. This suggests that the added time for donor lung management with CRRT may be instrumental in improving the lung function and transplanting the lungs.

There was no difference in the initial EF among the three groups. In the CRRT group there were five of 12 heart donors (42%) with a mean initial EF 43 ± 4% that increased to 57 ± 4% over the course of donor management. There was a similar improvement in five of eleven (45%) heart donors in the contemporaneous nonoliguric cohort with the EF increasing from 43 ± 6% to 58 ± 3%. There were only two heart donors in the historical oliguric cohort and only one had an initial EF < 50%.

3.7 | Kidney biopsies

Twenty-two donors in the CRRT group had 44 renal biopsies (Table 4A). Twenty-eight biopsies from 14 donors (52%) revealed ATN and less than 20% GS. Eighteen (64%) of these kidneys were transplanted. Ten kidneys from five donors had extensive parenchymal or cortical necrosis (CN) and none were transplanted. Although four donors had elevated creatinines and oligoanuria, the eight kidney biopsies did not reveal any significant ATN, GS, nor evidence of chronic changes, and only two of these kidneys were transplanted. Four donors did not have any renal biopsies because there were no provisional kidney acceptances by the transplant centers.

In the contemporaneous nonoliguric cohort, 19 donors had 38 renal biopsies, of which 25 kidneys were transplanted (46%) (Table 4B). All of the biopsies from the transplanted kidneys revealed ATN or mild interstitial fibrosis with tubular atrophy (IFTA) and minimal GS. Two donors had mild CN and mild-moderate IFTA and one donor had 35% GS and moderate IFTA. None of these kidneys were transplanted. Ten of the 20 (50%) transplanted kidneys in the CRRT group were pumped, and 18/25 (72%) of the kidneys in the contemporaneous group were pumped. Tables 4A and 4B list all of the biopsy findings and pertinent clinical parameters from this cohort and the CRRT group. There were 16 kidneys in the CRRT group and 10 kidneys in the contemporaneous non-oligoanuric cohort that had ATN without necrosis or chronic disease that were not transplanted for a variety of reasons, including abnormal visual inspection, excessive cold ischemic time, recipient issues and poor quality.

4 | DISCUSSION

This study demonstrates that CRRT, which was initiated for AKI in an ORC after the diagnosis of BD, is not only feasible but

TABLE 2B Initial laboratory values after the declaration of brain death and on arrival to the organ recovery center for the CRRT and cohort groups: Contemporaneous nonoliguric cohort and CRRT group

	Contemporaneous nonoliguric cohort[h]	CRRT group[b]	
	% or Mean (SD)	% or Mean (SD)	Statistical test
Number of donors	($n = 27$)	($n = 27$)	
Admission creatinine (mg/dl)[c]	1.4 (.3)	2.1 (1.4)	$P = .12$
Peak creatinine (mg/dl)	5.4 (1.6)	6.1 (2.3)	$P = .24$
Blood urea nitrogen (mg/dl)	39.1 (14.2)	54.9 (29.7)	$P = .01$
Sodium (mEq/L)	144.4 (8.9)	138.8 (7.8)	$P = .02$
Potassium (mEq/L)	4.1 (.7)	5.0 (1.1)	$P < .01$
Serum bicarbonate (mEq/L)	22.6 (4.9)	21.3 (3.7)	$P = .30$
pH	7.4 (.1)	7.4 (.1)	$P = .17$
Calcium (mg/dl)	8.2 (.9)	7.8 (1.0)	$P = .11$
Phosphate (mg/dl)	5.2 (3.9)	5.5 (1.9)	$P = .17$
Lactate (mmol/L)	1.8 (.8)	3.8 (3.5) [$n = 25$]	$P < .01$
PaO_2/FiO_2 (initial)[d]	277 (138)	263 (137)	$P = .71$
PaO_2/FiO_2 (final)[e]	336 (129)	402 (172)	$P = .11$
Cardiac ejection fraction (EF) (initial) (%)	51.2 (12.9) [$n = 20$]	49.6 (13.8) [$n = 20$]	$P = .74$
Initial EF < 55%[f]	10 (50%)	11 (55%)	$P = .75$
Initial EF < 55%, final EF ≥ 55%[g]	7 (70%)	6 (54.5%)	

Abbreviations: AKI, acute kidney injury; BD, brain-dead; CRRT, continuous renal replacement therapy; mg, milligrams; dl, deciliter; EF, cardiac ejection fraction; mEq, milliequivalents; L, liter; mmol, millimole; PaO_2, partial pressure of arterial oxygen; FiO_2, fraction of inspired oxygen; mm Hg, millimeters of mercury; ORC, organ recovery center; SD, standard deviation.

[a]This cohort is a historical group of BD donors with oligoanuric AKI managed at the ORC between January 1, 2015, and December 31, 2017, that did not receive CRRT.

[b]CRRT group is the group of BD donors with oligoanuric AKI managed at the ORC that underwent CRRT between January 1, 2017, and June 30, 2021.

[c]The admission creatinine was the first creatinine measured on admission to the donor hospital.

[d]The initial PaO_2/FiO_2 was measured on arrival to the organ recovery center.

[e]The final PaO_2/FiO_2 was the last measurement prior to organ procurement.

[f]The number of donors with an initial ejection fraction < 55% measured by echocardiography.

[g]The number of donors with an initial EF < 55% and a final EF≥55% measured by serial echocardiography.

[h]This cohort is a concurrent group of BD donors with non-oligoanuric AKI managed at the ORC between January 1, 2017, and June 30, 2021, that did not receive CRRT.

beneficial. Comparing the donors with oligoanuric AKI, the total number of organs transplanted per donor from this CRRT cohort was greater than the historical cohort that did not receive CRRT. Compared to the contemporaneous nonoliguric cohort there was no difference in the number of organs transplanted per donor and the CRRT group. The only major difference between these two groups was that the contemporaneous cohort was nonoliguric, in which the volume status could be controlled by diuretics and vasopressin, whereas in the CRRT group the volume status was controlled by ultrafiltration. This suggests that controlling the volume status of the donor may be an important factor in the effectiveness of CRRT in transplanting more organs. In terms of organs transplanted, the benefit of CRRT is the increase in the number of hearts and lungs transplanted. Compared to the historical oliguric cohort, there were twice as many thoracic organs transplanted in the CRRT group.

Nationally, over the same time period as the CRRT group, there were 1374/ 38 554 (3.6%) brain-dead organ donors with oligoanuric AKI. The total number of organs transplanted per donor was 2.0 (2783/1374). From this group, 27.2% were heart donors, 16.2% were lung donors, 70.4% were liver donors, and 35.4% were kidney donors. Compared to donors with AKI in this national sample, we found higher unadjusted rates of total and thoracic organs transplanted per donor in the CRRT group. Although these differences are noteworthy, it is difficult to compare local to national data, given the multiple confounding variables that were not considered.

The number of lungs transplanted per donor for the CRRT group (48.1%) was much greater than the historical oliguric cohort (21.4%), and the national average for BD donors with oligoanuric AKI (16.2%) as well as from all BD donors (27%).[1] This is due in part to an aggressive lung donor management protocol that we have been using since 2008.[6] The salient point is that CRRT allowed us time to remove fluid with ultrafiltration, perform repeated intrapulmonary percussive ventilations and bronchoscopies, and recruit the lungs. Almost 40% of the lung donors in the CRRT group had an initial P/F < 300 that would deem

TABLE 3A Number of organs transplanted from the CRRT and cohort groups: Historical oliguric cohort and CRRT group

	Historical oliguric cohort[a] (% or SD)	CRRT group[b] (% or SD)	Statistical test
Number of donors	(*n* = 14)	(*n* = 27)	
Total organs transplanted/donor (mean)	1.4 (.6)	2.9 (1.7)	*P* < .01
Total kidneys transplanted/donor(mean)	.2 (.6)	.7 (1.0)	*P* = .10
Liver transplanted	12 (85.7%)	20 (74.1%)	*P* = .69
Lungs transplanted[c]	3 (21.4%)	13 (48.1%)	*P* = .18
Heart transplanted	2 (14.3%)	12 (44.4%)	*P* = .08
Total thoracic organs transplanted/donor (mean)[d]	.6 (.9)	1.4 (1.2)	*P* = .02

TABLE 3B Number of organs transplanted from the CRRT and cohort groups: Contemporaneous nonoliguric cohort and CRRT group

	Contemporaneous nonoliguric cohort[e] (% or SD)	CRRT group[b] (% or SD)	Statistical test
Number of donors	(*n* = 27)	(*n* = 27)	
Total organs transplanted/donor (mean)	3.1 (1.5)	2.9 (1.7)	*P* = .67
Total kidneys transplanted/donor(mean)	.9 (1.0)	.7 (1.0)	*P* = .48
Liver transplanted	23 (85.2%)	20 (74.1%)	*P* = .50
Lungs transplanted[c]	12 (44.4%)	13 (48.1%)	*P* = .79
Heart transplanted	11 (40.7%)	12 (44.4%)	*P* = .78
Total thoracic organs transplanted/donor (Mean)[d]	1.3 (.9)	1.4 (1.2)	*P* = .71

Abbreviations: AKI, acute kidney injury; BD, brain-dead; CRRT, continuous renal replacement therapy; ORC, organ recovery center; SD, standard deviation.

[a]This cohort is a historical group of BD donors with oligoanuric AKI managed at the ORC between January 1, 2015, and December 31, 2017, that did not receive CRRT.

[b]CRRT group is the group of BD donors with oligoanuric AKI managed at the ORC that underwent CRRT between January 1, 2017, and June 30, 2021.

[c]Double lung transplanted is counted as one lung donor.

[d]Maximum total thoracic organs included one heart and two lungs.

[e]This cohort is a concurrent group of BD donors with non-oligoanuric AKI managed at the ORC between January 1, 2017, and June 30, 2021, that did not receive CRRT.

the lungs non-transplantable. With the added time for lung donor management afforded by CRRT, these lungs were able to be transplanted. Lungs transplanted from the CRRT group also functioned well in the short term with 100% viability at 30 days. The fact that the number of lung donors between the contemporaneous nonoliguric cohort and CRRT groups was the same is a testament to the need for ultrafiltration in the oligoanuric group to clear any pulmonary edema, since our lung donor management protocol was the same in both groups. This is supported by the lack of an increase in P/F in the historical oliguric cohort.

CRRT did not only correct electrolyte abnormalities and acidosis, but also allowed more time for an evaluation of the heart function. The heart is frequently stunned after BD, demonstrated by a decreased EF.[9] About half of the stunned hearts will improve over time and be transplanted. This may take up to 72 h of observation and repeated echocardiograms. This would not be possible in an anuric patient with AKI. Forty-two (5/12) percent of the heart donors in the CRRT group had a non-transplantable heart based on the initial EF≤45%, but with the additional time provided by CRRT the EF improved and the hearts were transplanted. Hearts were transplanted from 44.4% of our CRRT group, which is greater than the contemporaneous national average of AKI donors (27.2%) and the historical oliguric cohort (14.3%). The hearts transplanted from the CRRT group also had excellent graft survival at 6 months. CRRT prevented us from losing transplantable thoracic organs in our donors with AKI and oligoanuria. Without CRRT in clinically unstable donors with AKI, many OPOs would curb their donor management time and hasten to procurement limiting the number of organs transplanted.

Multiple studies have shown that kidneys that suffered AKI after brain death can be transplanted with good short and long-term outcomes.[2–5,10–16] Using the acute kidney injury network (AKIN) criteria, most of these studies have a low percentage of kidneys with Stage 3 AKI, rarely do they include kidneys requiring CRRT,[17] and the timing of CRRT initiation and outcomes of kidneys from donors treated with CRRT were not specifically reported. In a single center study in Germany over 11 years, 263 kidneys with AKI were transplanted but only ten received CRRT, and it is unclear when the CRRT was started.[5] In 2015, Heilman et al. reported on one of the largest studies of transplanting kidneys with severe AKI.[17] There were 162 AKI kidneys transplanted, and 71% had Stage 3 AKI, 50% were oligoanuric, and 21% were on renal replacement therapy. Delayed graft function was more common in the AKI group, but there was no difference in 1 year graft survival from a cohort group. The study did not describe if the CRRT was started before or after BD. The overall transplant rate of these kidneys with AKI was 25%. Forty-six percent of our kidneys with AKI and 37% of kidneys with AKI requiring CRRT were transplanted, with serum creatinines of 1.4 mg/dl at 6 months. All of the kidneys that received CRRT that were transplanted had ATN and absent chronic disease on biopsy. Five donors that had anuria and received CRRT had severe parenchymal or cortical necrosis from the anoxic event that caused their death. We were still able to transplant nine non-renal organs from these donors. There is still some hesitancy in transplant surgeons accepting kidneys that have received CRRT, possibly because

TABLE 4A Kidney biopsy results from donors in the CRRT group and the contemporaneous nonoliguric cohort: CRRT group

	Demographics			Renal function		Renal biopsy		Kidney pumped[g]		Kidney transplanted	
	Age	DM[a]	HTN[b]	Admission creatinine[c]	Peak creatinine[d]	Diagnosis[e]	Pertinent findings[f]	Right	Left	Right	Left
1	34	N	N	6.4	8.0	ATN	Global glomerulosclerosis < 5%, Mild IFTA, No thrombi, Severe ATN, mild arteriosclerosis				
2	30	N	N	1.5	4.2	ATN	Global glomerulosclerosis < 5%, No IFTA, No thrombi, Severe ATN				
3	24	N	N	3.4	7.4	ATN	Glomerulosclerosis < 2%, No IFTA/thrombi, ATN, pigmented casts			●	●
4	30	N	N	1.0	6.4	Parenchymal necrosis	Extensive parenchymal necrosis, fibrin thrombi, No global sclerosis/IFTA/ thrombi				
5	24	N	N	1.5	5.3	ATN	Global glomerulosclerosis 9–13%, ATN, rare oxalate stones, no thrombi, mild IFTA			●	●
6	23	N	N	1.6	4.4	ATN	focal glomerulosclerosis 5%, mild IFTA, ATN, No fibrin		●	●	●
7	27	N	N	1.9	5.8	ATN	Glomerulosclerosis 11–15%, hypercellular non-sclerotic glomeruli, mild IFTA, ATN				
8	23	N	N	1.3	4.9	ATN	ATN ~ 30%, No glomerulosclerosis, No IFTA, focal glomerular thrombi				
9	30	N	N	1.4	6.2	Cortical necrosis	Cortical necrosis > 60 % cortex, no glomerulosclerosis/ IFTA, glomerular fibrin thrombi +				
10	45	Y	Y	3.5	4.2	***	***				
11	29	N	N	1.5	8.2	ATN	No glomerulosclerosis, No IFTA, no arteriolar hyalinosis, cortical necrosis, no thrombi, ATN	●	●	●	●
12	34	N	N	1.7	5.2	ATN	No glomerulosclerosis, No IFTA, no arteriolar hyalinosis, no cortical necrosis, no thrombi, ATN	●		●	●
13	60	N	Y	1.1	4.6	***	***				
14	48	N	Y	1.2	13.5	ATN	No glomerulosclerosis, mild atherosclerosis, no interstitial fibrosis, ATN				
15	18	N	N	2.9	8.7	ATN	Glomerulosclerosis (2%), no interstitial fibrosis, no arteriosclerosis	●	●	●	●
16	36	Y	N	3.7	2.8	***	***				

(Continues)

TABLE 4A (Continued)

	Demographics			Renal function		Renal biopsy		Kidney pumped[g]		Kidney transplanted	
	Age	DM[a]	HTN[b]	Admission creatinine[c]	Peak creatinine[d]	Diagnosis[e]	Pertinent findings[f]	Right	Left	Right	Left
17	12	N	N	1.3	7.4	Mild interstitial fibrosis	No glomerulosclerosis, no fibrosis, no fibrin thrombi, no ATN			●	●
18	25	N	N	.8	4.8	Cortical necrosis	Mild interstitial fibrosis and vascular changes, cortical necrosis (-90%), focal global glomerulosclerosis, glomerular fibrin thrombi				
19	30	N	N	1.3	3.2	***	No glomerulosclerosis, no cortical necrosis, no IFTA, no thrombi	●	●		
20	21	N	N	1.4	7.5	Completely necrotic	Clinically necrotic, no biopsy performed				
21	41	Y	Y	4.4	8.3	***	***				
22	43	N	N	1.6	5.1	ATN	ATN, no glomerulosclerosis, no IFTA, no arteriosclerosis, no thrombi, no cortical necrosis			●	●
23	35	N	N	1.8	3.8	ATN	ATN, yellow-green tubular casts, glomerulosclerosis < 2% (2/135), no IFTA, no arteriosclerosis, no cortical necrosis, no thrombi			●	●
24	22	N	N	1.2	5.71	cortical necrosis	global glomerulosclerosis (3%), mild IFTA, mild arteriosclerosis, cortical necrosis involving ~80% of renal cortex, fibrin thrombi				
25	24	N	N	2.1	6	***	global glomerulosclerosis (3%), minor discoloration, no IFTA, no arteriosclerosis, no thrombi	●	●		
26	51	N	Y	1.5	9.8	Mild interstitial fibrosis	global glomerulosclerosis (2%), mild IFTA, no arteriosclerosis, no fibrin thrombi				
27	46	N	N	1.4	7.6	ATN	ATN, mild arteriosclerosis, no glomerulosclerosis, no cortical necrosis, no fibrin thrombi			●	●

of the increased incidence of delayed graft function. There were 12 kidneys with ATN from donors less than 40 years old without diabetes or hypertension that were not transplanted. Hopefully as more transplant centers appreciate the beneficial outcome of kidneys transplanted with ATN and receiving CRRT fewer of these kidneys will be discarded.

CRRT is rarely started in an organ donor after the diagnosis of BD. There is one published study by Park et al. that describes their experience in South Korea in aggressively managing 54 BD organ donors over 41 months, of which 22 (40.7%) received CRRT for AKI.[18] They do not describe any details of the AKI nor CRRT other than it was performed at an early stage and 21 of 22 donors were able to donate kidneys.

TABLE 4B Kidney biopsy results from donors in the CRRT group and the contemporaneous nonoliguric cohort: Contemporaneous nonoliguric cohort

	Demographics			Renal Function		Renal Biopsy		Kidney Pumped[g]		Kidney Transplanted	
	Age	DM[a]	HTN[b]	Admission Creatinine[c]	Peak Creatinine[d]	Diagnosis[e]	Pertinent Findings[f]	Right	Left	Right	Left
1	23	N	Y	1.2	6.8	***	***				
2	48	N	Y	1.6	4.4	***	***				
3	34	N	N	1.3	3.7	ATN	ATN, mild IFTA, no glomerulosclerosis, no arteriosclerosis, no thrombi	●		●	●
4	32	N	N	1.2	5.5	IFTA	mild IFTA, no glomerulosclerosis, no arteriolar hyalinosis, no thrombi				
5	34	N	N	.9	4.7	IFTA	glomerulosclerosis (2%), mild cortical necrosis, mild IFTA, no arteriosclerosis, no thrombi				
6	42	N	Y	.9	4.1	ATN	ATN, no global glomerulosclerosis, no IFTA, no thrombi	●	●	●	●
7	36	N	N	1.4	4.2	ATN	minimal glomerulosclerosis, mild IFTA, ATN, no thrombi		●	●	●
8	36	N	Y	1.3	6.1	IFTA	moderate IFTA, mild arteriosclerosis, cortical necrosis, fibrin thrombi, no arteriosclerosis				
9	47	N	Y	2.0	4.0	***	***				
10	45	Y	N	1.5	3.6	IFTA	Glomerulosclerosis (20%), no ATN, mild IFTA, mild arteriolar thickening, no fibrin thrombi	●	●	●	
11	34	Y	Y	1.7	5.2	***	***				
12	33	N	Y	1.1	4.0	ATN	ATN, focal global glomerulosclerosis (35%), moderate IFTA, + glomerular thrombi, no arteriosclerosis	●	●		
13	37	Y	N	1.4	5.4	***	global glomerulosclerosis (2%), no IFTA, no arteriosclerosis, no thrombi, no cortical necrosis	●	●	●	●
14	32	N	N	1.8	9.1	***	***				
15	41	N	Y	1.7	6.1	***	***				
16	30	N	N	1.0	9.3	ATN	ATN, glomerular fibrin thrombi, no global glomerulosclerosis, no IFTA				
17	47	Y	Y	1.2	5.8	***	***				

(Continues)

TABLE 4B (Continued)

	Demographics			Renal Function		Renal Biopsy		Kidney Pumped[g]		Kidney Transplanted	
	Age	DM[a]	HTN[b]	Admission Creatinine[c]	Peak Creatinine[d]	Diagnosis[e]	Pertinent Findings[f]	Right	Left	Right	Left
18	35	N	N	1.7	4.4	ATN	focal global glomerulosclerosis (2%), ATN, no IFTA, no arteriosclerosis, no thrombi, no cortical necrosis			●	●
19	26	N	N	1.6	5.7	ATN	acute tubular injury with focal necrosis, no sclerotic glomeruli			●	●
20	31	N	N	1.6	6.6	ATN	global glomerulosclerosis (3%), mild IFTA, no arteriosclerosis, no thrombi, no cortical necrosis	●	●	●	●
21	29	N	N	1.5	3.9	ATN	ATN, focal global glomerulosclerosis (2%), no IFTA, no cortical necrosis, no thrombi	●	●	●	●
22	40	Y	Y	1.8	4.4	***	***				
23	39	N	N	1.5	4.7	ATN	ATN, global glomerulosclerosis (6%), no IFTA, no thrombi				
24	46	N	N	1.2	8.1	ATN	glomerulosclerosis (2%), ATN, mild interstitial edema, no interstitial fibrosis			●	●
25	38	N	N	1.3	5.7	***	Global glomerulosclerosis (1%), no IFTA, no arteriosclerosis, no thrombi	●	●	●	●
26	27	N	N	.9	4.4	IFTA	mild IFTA, mild arteriosclerosis, no global glomerulosclerosis, no cortical necrosis, no thrombi	●	●	●	●
27	37	N	N	1.1	7.0	IFTA	mild IFTA, no glomerulosclerosis			●	●

Abbreviations: ATN, acute tubular necrosis; DM, diabetes mellitus; HTN, hypertension, IFTA, interstitial fibrosis and tubular atrophy.
[a]DM, any history of diabetes mellitus; Y = yes, N = no.
[b]HTN, any history of hypertension; Y = yes, N = no.
[c]The admission creatinine was the first serum creatinine measured on arrival to the donor hospital.
[d]Peak creatinine was the greatest serum creatinine measured either at the hospital or the organ recovery center.
[e]The pathological diagnosis from the renal biopsy. An absent diagnosis indicates no abnormality was detected on the biopsy or no biopsy was performed.
[f]Details from the renal biopsy are displayed. An absent result indicates that no biopsy was performed.
[g]Kidneys were pumped with hypothermic preservation solution at 4°C in a LifePort Kidney Transporter (Organ Recovery Systems) at the discretion of the transplant surgeon.

4.1 | Limitations

Although our study demonstrates the feasibility and practicality of CRRT in BD donors with oligoanuric AKI in an ORC, it does have some limitations. This study is observational and retrospective. The historical oliguric cohort was a retrospective 3-year cohort with AKI and oligoanuria before we fully implemented CRRT as a protocol. The historical cohort also had older donors with more hypertension and diabetes which could decrease the chance of transplanting hearts and kidneys. There were some small differences in the initial lab values between the groups, and although statistically significant the differences were probably not clinically significant. Although the

contemporaneous nonoliguric cohort was a retrospective cohort over the same time period with AKI that did not have oligoanuria, it was fairly well-matched to the CRRT cohort except for mildly increased age. The study is also limited by its small sample size, and lack of adjustment for both known confounders such as age, and unmeasured confounders, such as the amount of fluid resuscitation used in the historical cohort. There also may have been some bias in selecting more stable donors for CRRT in the beginning of the study as we were developing our protocol, training our staff and evaluating short-term outcomes.

There is no financial disincentive to starting CRRT since Medicare will reimburse any associated cost with CRRT and the benefit would be the additional revenue from any additional organ transplanted. The study was also performed at a single OPO with a 20-year history of aggressively managing BD donors in an independent ORC. Other independent ORCs may not have the capability, expertise or training to perform CRRT. This may also present logistical challenges for the OPO that manages donors in small hospitals that do not have easy access to CRRT. OPOs that have a transplant center-based ORC should not have any difficulty performing CRRT, utilizing the equipment and staff from the hospital. In spite of these limitations, many donor hospitals have the capability to perform CRRT and we suggest that OPOs consider starting CRRT in the oligoanuric donor to increase the number of organs transplanted.

5 | CONCLUSION

The main goal of our study was to demonstrate the feasibility and benefit of starting CRRT in a donor with oligoanuric AKI after the diagnosis of BD, which currently is rarely performed. This allows more time to manage the donor and optimize the function of the thoracic organs, increasing their chance of being transplanted. Although oligoanuric AKI in BD donors is not a frequent occurrence, it does provide the opportunity to transplant more organs. We are encouraged by these results and suggest that OPOs consider initiating CRRT in their donors with oligoanuric AKI. This is the first study at an independent ORC to describe in detail the process of starting CRRT in deceased donors after the diagnosis of BD, which was associated with more organs being transplanted. Although these preliminary results are compelling, they need to be confirmed by a larger randomized trial with contemporaneous treatment and control groups.

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CONFLICT OF INTEREST

The authors of this manuscript have no conflict of interest to disclose.

AUTHOR CONTRIBUTIONS

Gary F. Marklin and Marcos Rothstein: Contributed to study concept and design; Gary F. Marklin, W.D. Klinkenberg, Marcos Rothstein, Steven J. Bander: Contributed to data analysis/interpretation; Gary F. Marklin and Marcos Rothstein: Contributed to drafting article; Gary F. Marklin, Marcos Rothstein, Steven J. Bander: Contributed to critical revision of article; W. Dean Klinkenberg: contributed to statistics; Laura Ewald and Christina M. Joy: Contributed to data collection; Gary F. Marklin, Laura Ewald, W. Dean Klinkenberg, Steven J. Bander, and Marcos Rothstein: Gave final approval to article.

DATA AVAILABILITY STATEMENT

The donor data are not publicly available due to privacy and ethical restrictions.

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