The Impact of Diabetes on Ethnic Disparities Seen in Kidney Transplantation

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Graft failure rates following kidney transplant is disproportionately higher in African American (AA) renal transplant recipients. The aim of our study was to measure the impact of diabetes and other known confounding risk factors on this disparity. This was a long-term cohort study of adult kidney transplant recipients between 2000 and 2008 comparing AA transplant recipients to White recipients. 987 patients were included and patients were followed for up to 12 years post-transplant. Univariate analysis demonstrated AA recipients were more likely to have diabetes (35% vs 23%, $P<.001$), hypertension (97% vs 94%, $P=.029$), human leukocyte antigen mismatches (4 vs 3, $P<.001$), and receiving dialysis for a longer period prior to transplant (3.9 vs 2.0 yrs, $P<.001$). AA patients were also less likely to receive a living donor transplant (7% vs 31%, $P<.001$). Multivariable modeling established both AA ethnicity (HR 1.32 [95% CI 1.04–1.68]) and pre-existing diabetes (1.58 [95% CI 1.25–2.00]) as important predictors of graft failure. Diabetes was a significant modifier on the influence of AA ethnicity as a risk factor for graft loss (19% HR reduction); tight glycemic control, which was less common in AA recipients (35% vs 51%, $P=.013$), additionally attenuated the ethnic disparities seen in graft loss (28% risk reduction). In the final fully adjusted model, which included sociodemographic, immunologic, and cardiovascular risk factor as variables, the influence of AA ethnicity on graft failure was essentially nullified (HR 1.09 [.81–1.48]). In conclusion, AA ethnicity continues to be an important risk factor for graft loss, which can be significantly attenuated by controlling for pre-existing diabetes, glycemic control, and other transplant and cardiovascular variables. (*Ethn Dis.* 2013;23[2]:238–244)

Key Words: Kidney Disease, Renal Transplantation

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Introduction

Despite significant advancements in immunosuppressant medications and regimens, improved diagnostic testing for allograft dysfunction and rejection, and more potent treatment regimens for infectious complications, African American (AA) renal transplant (RTx) recipients continue to experience a disproportionately higher incidence of graft failure; a disparity that becomes even more striking as the time from transplant increases. Based on the most recent published scientific registry of transplant recipients (SRTR) data, AA RTx recipients have a 2.7% absolute lower rate of graft survival at one year post-transplant (93.7% vs 91%); this difference balloons to 9.8% at 5 years (76.1% vs 66.3%) and 13.8% at 10 years (50.9% vs 37.1%). This means the average transplanted kidney will survive for only 8 years in AA RTx recipients, while this number is over 14 years for all other RTx recipients.[1] Many studies have been conducted over the past two decades unraveling the likely mechanisms involved with this disparity, including immunologic mechanisms (eg, greater variation in human leukocyte antigen (HLA) polymorphisms, stronger immune response, different pharmacokinetics and pharmacodynamics of the immunosuppressants), and non-immunologic mechanisms (eg, comorbidities, longer time on dialysis prior to RTx, less living donors, lower socioeconomic status, poorer medication adherence, and access to health care). Well-designed and conducted interventional studies to reduce or remove this disparity are mostly lacking, but have focused on improved early access to transplantation and modifying immunosuppression regimens and monitoring.[2–8]

Although it is well-known that AA RTx are significantly more likely to have pre-existing comorbidities, including hypertension or diabetes (DM),[2] at the time of transplant, there is very little research analyzing the direct effects these comorbidities contribute to the overall disparity seen in AA RTx after transplant. Several recent large-scale published analyses have demonstrated that DM is a major contributor to graft loss after transplant in the overall RTx population, primarily due to death with a functioning graft from cardiovascular disease.[9–11] The aim of our study was to assess the impact of pre-existing DM on acute rejection and graft survival seen within the AA RTx population.

Methods

Study Population

This was an IRB-approved single-center cohort study of 987 renal transplants conducted between 2000 and 2008. Patients were excluded if they were aged <18 years, received a multi-organ transplant, were of an ethnicity of non-White or non-AA, or

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were lost to follow-up. Patients were followed from time of kidney transplant to death, loss to follow-up or end of the study (April 2012). For the initial univariate analysis, patients were divided into two groups: AAs and Whites. Following this, multivariable models were conducted in a sequential fashion, starting with an unadjusted model, and adding additional controlling variables (as defined below) to determine the impact of DM has on the ethnic disparities seen with acute rejection and graft loss.

Variable Definitions

Pre-existing DM was defined as patients who had a clinical diagnosis of DM prior to transplant. The diagnosis of DM was documented in the medical record and included patients with both diet-controlled and those who required oral hypoglycemic agents or insulin for DM management.

New onset diabetes after transplant (NODAT) was defined as any patient with a hemoglobin A1C>6.5%, diagnosis of new onset diabetes after transplant based on medical record, or if the patient began treatment with insulin or an oral hypoglycemic agent post-transplant.

Controlled DM was defined as patients with either an average hemoglobin A1C<7.0% or an average blood glucose≤150 mg/dL throughout the study period (for those who did not have hemoglobin A1C recorded in the medical records).

Cardiovascular disease risk factor control with medications was defined as use of beta-blockers, ACE inhibitors/ARBs, or statins. The use of these agents was analyzed as a continuous variable and defined as percentage of time receiving these agents after transplant.

Graft failure was defined as return to chronic dialysis, re-transplant, or death.

Biopsy-proven acute rejection was defined as either Grade 1A or higher or if a patient received treatment for borderline rejection. Delayed graft function was defined as requiring dialysis within seven days post-transplant.

Immunosuppression

Patients received induction therapy with either thymoglobulin 1.5 mg/kg IV daily for 3 to 5 doses, daclizumab 1 mg/kg IV on day 0 and day 7 post-transplant, or basiliximab 20 mg IV on day 0 and day 4 post-transplant. Choice of induction therapy was based on protocols that utilized thymoglobulin for high immunologic risk patients (re-transplantation, cold ischemic time [CIT] >24 hours, or panel reactive antibody [PRA]>20%), and an IL2 receptor antibody in all other patients. Maintenance immunosuppression from 2000 to 2004 consisted of cyclosporine adjusted to maintain target trough whole blood concentrations between 200 and 275 ng/mL for weeks 1 through 6, 125 to 225 ng/mL for weeks 7 through month 12, and >70 ng/mL after 1 year. Tacrolimus became the agent of choice in 2005. Tacrolimus was adjusted to maintain target trough whole blood concentrations between 8 and 12 ng/mL for weeks 1 through 6, 6 to 12 ng/mL for weeks 7 through month 12, and >5 ng/mL after 1 year. In addition, all patients received mycophenolate mofetil 1 gram twice daily and prednisone titrated to 5 mg daily by day 90 post-transplant, with tapers below 5 mg occurring rarely. Very few patients were taken off corticosteroids.

Outcome Measures

The primary outcomes were overall graft survival and acute rejection. Comparisons for these primary outcomes were initially conducted in a univariate fashion between ethnicities, and then were used as the time dependent variable for the multivariable modeling.

Key Covariates

Based on the literature,[9–11] covariates were grouped as sociodemographic, clinical, peri-transplant and post-transplant.

Sociodemographic markers included age (treated as a continuous variable), ethnicity (AA, categorized as yes/no), sex (male, categorized as yes/no), body mass index (BMI, measured at the time of transplant and treated as a continuous variable), years on dialysis prior to transplant (treated as a continuous variable).

Clinical covariates were baseline comorbidity and surgical interventions included congestive heart failure (CHF), coronary artery disease (CAD), dyslipidemia, cerebral vascular accident (CVA), coronary bypass grafting (CABG), heart catheterization with intervention, acute myocardial infarction (AMI), peripheral vascular disease (PVD) and hypertension. These were based on past medical history at the time of transplant and were coded as yes (present) vs no (not present at baseline). Baseline smoking status from the medical history was categorized at time of transplant, categorized as yes (ever smoked) vs. no (never smoked).

Peri-transplant data included: living donor (categorized as yes/no), extended criteria donor (ECD, categorized as yes/no), re-transplant (categorized as yes/no), HLA mismatch (treated as a continuous variable, 0–6 mismatches), panel reactive antibody (PRA, treated as a continuous variable, 0–100%), cold ischemic time (CIT, treated as a continuous variable, time in minutes), warm ischemic time (WIT, treated as a continuous variable, time in minutes).

Post-transplant records included: acute rejection (defined as above, categorized as yes/no), delayed graft function (defined as the need for dialysis within 7 days after transplant, categorized as yes/no), NODAT (categorized as yes/no), acute MI (after transplant, categorized as yes/no), CVA (after transplant, categorized as yes/no), CHF (after transplant, categorized as yes/no), CABG or cardiac catheterization with intervention (after transplant, categorized as yes/no), control of DM

(defined as above after transplant, categorized as yes/no), use of CVD risk factor medications (defined as above after transplant, treated as continuous variable).

STATISTICAL ANALYSIS

Sample characteristics were compared by ethnicity using *t*-tests for continuous variables and chi-square for categorical variables. Cox Proportional Hazard Regression was used to examine the effect of ethnicity on time to graft loss and acute rejection controlling for sociodemographic, clinical, peri-transplant and post-transplant factors. Covariates were entered sequentially in groups into the model to examine their contribution to the outcomes. The initial model included only the independent variable of ethnicity, then the independent variable of pre-existing DM; subsequent models were then adjusted for: 1) AA ethnicity adjusted for pre-existing DM, 2) AA ethnicity, pre-existing DM, and glycemic control, and 3) a fully adjusted model, including demographic characteristics including age, sex, and race, baseline clinical characteristics including body mass index (BMI), years on dialysis prior to transplant, and pre-existing CVD, and CVD risk factors, baseline peri-transplant characteristics including donor type, re-transplant, HLA mismatch, PRA, cold ischemic time, and warm ischemic time, and post-transplant events including acute rejection, delayed graft function (DGF), NODAT, and post-transplant CVD events. Kaplan-Meier survival analysis was used to compare graft survival for the two groups (AA and White patients) for patients with and without pre-existing DM. The log rank test was used to calculate statistical significance. Statistical significance was based on a $P<.05$. SPSS (Version 20, SPSS Inc. Chicago, Ill) was used for statistical analysis.

Table 1. Baseline characteristics by ethnicity, *N*=987

Characteristic	AA *n*=578	White *n*=409	*P*
Male	56%	62%	.011
Age, years	48 ± 13	50 ± 14	<.001
Body mass index	26 ± 9	25 ± 8	.039
Peak PRA	9 ± 22	12 ± 25	.146
Cold ischemia time, min	990 ± 523	999 ± 574	.869
Warm ischemia time, min	37 ± 20	36 ± 10	.873
HLA mismatch	4 ± 1	3 ± 2	<.001
Years on dialysis	3.9 ± 2.7	2.0 ± 2.1	<.001
Preexisting diabetes	35%	23%	<.001
History of hypertension	97%	94%	.029
Smoking history	25%	35%	.001
History of heart failure	4%	2%	.047
History of heart disease	20%	24%	.179
History of stroke	8%	5%	.120
History of acute MI	3%	6%	.017
Donor Type			
Living	7%	31%	<.001
Extended criteria	7%	6%	.431
Retransplant	10%	14%	.026
Induction therapy			
Antilymphocyte antibody	31%	25%	.012
Interleukin-2 antagonist	56%	56%	
Calcineurin inhibitor			
Cyclosporine	45%	50%	.174
Tacrolimus	55%	50%	
Mycophenolic acid	92%	91%	.551
Insulin use pre-txp	25%	18%	.022
Oral antiglycemic pre-txp	5%	3%	.054

Data are means ± SD unless indicated otherwise.
AA, African American; PRA, panel reactive antibody; HLA, human leukocyte antigen; MI, myocardial infarction.

RESULTS

A total of 987 patients were included in this analysis (578 AAs, 409 Whites), after removing excluded patients and patients of non-AA or non-White ethnicity. Table 1 displays the baseline characteristics of the cohort between ethnicities. Demographic comparisons across ethnicity reveal AA renal transplant recipients, on average, were less likely to be male (56% vs 62%, $P=.011$), younger (48 vs 50 years, $P<.001$) and had a higher BMI (26 vs 25, $P=.039$). Baseline transplant characteristics show AA renal transplant recipients had higher HLA mismatches (4 vs 3, $P<.001$), waited a longer time on dialysis prior to transplant (3.9 vs 2.0 years, $P<.001$) and less living donor transplants (7% vs. 31%, $P<.001$). Finally, comparisons of baseline comorbidities reveal AA recipients had a lower incidence of smoking (25% vs 35%, $P<.001$), but a larger burden of significant comorbidities, with a higher incidence of heart failure (4% vs. 2%, $P=.047$), hypertension (97% vs. 94%, $P=.029$) and pre-transplant DM (35% vs. 23%, $P<.001$).

The primary outcome analysis of patient and graft survival compared between ethnicities is shown in Table 2. Patient survival was similar at all time points between ethnicities (75% in AAs and 74% in Whites at 10-years post-transplant, $P=.895$). However, graft survival was significantly lower in AA transplant recipients, with statistically significant differences starting at three

Table 2. Outcomes based by ethnicity, *N*=987

Characteristics	AA *n*=578	White *n*=409	*P*
Patient survival			.895
One year	97%	96%	
Three year	92%	93%	
Five year	89%	89%	
Ten year	75%	74%	
Graft survival			.011
One year	91%	93%	
Three year	82%	88%	
Five year	76%	82%	
Ten year	60%	66%	
Acute rejection	37%	24%	<.001
Post-transplant CV event	9%	10%	.653
Post-transplant tight glycemic control	35%	51%	.013

AA, African American; CV, cardiovascular.

years post-transplant (82% vs 88%, *P*=.012). Acute rejection rates were higher in AA patients (37% vs 24%, *P*<.001), and AA patients with DM had a considerably lower likelihood of obtaining tight glycemic control after transplant compared with White patients with DM (35% vs 51%, *P*=.013).

Table 3 displays the multivariable models for death, graft loss and acute rejection. Initially, analyses were conducted for unadjusted ethnicity and unadjusted pre-existing DM, demonstrating that AA ethnicity is a significant risk factor for graft loss (HR 1.32 [95% CI 1.04–1.68]) and acute rejection (HR 1.69 [95% CI 1.33–2.15]), but not death (HR .98 [95% CI .73–1.31]). Pre-existing DM was a significant risk factor for death (HR 2.25 [95% CI 1.68–3.00]) and graft loss (HR 1.58 [95% CI 1.25–2.00]), and trended to be protective for acute rejection (HR .81 [95% CI0.63–1.04]). When AA ethnicity included the confounding variable of pre-existing DM, its independent predictive effect on graft failure was diminished and statistical significance was lost (HR 1.26 [95% CI .98–1.60]). However, AA ethnicity continued to be a significant risk factor for acute rejection (HR 1.76 [95% CI 1.38–2.24]). Adding the confounding variable of glycemic control continued to reduce the influence of AA ethnicity on graft failure (HR 1.23 95% CI .95–1.59), and in the fully adjusted model, which included demographics, transplant characteristics, pre-transplant comorbidities, and post-transplant cardiovascular events, the influence of AA ethnicity on graft failure was essentially nullified (HR 1.09 [95% CI .81–1.48]). Multivariable analyses for death demonstrate that AA ethnicity was not a significant or independent predicator of this outcome within any of the models. Conversely, multivariable analyses for acute rejection demonstrate that AA ethnicity remains a robust and independent risk factor for this outcome, with the fully adjusted model only modestly influencing ethnicity (HR 1.57, 95% CI 1.17–2.10).

Figure 1 displays two Kaplan-Meier analyses with the outcome variable of graft loss, and comparisons made between ethnicity. The first analysis displayed is in patients with pre-existing DM, demonstrating that AA transplant recipients with DM have a substantially higher rate of graft failure compared to White transplant recipients with DM (graft survival at 12-years post-transplant 42% vs 56%, *P*=.049). The second analysis compares graft survival rates across ethnicity in patients without DM, demonstrating equivalent rates in AA vs White recipients (51% versus 48%, *P*=.389). Multivariable modeling with the covariates of AA ethnicity, pre-existing DM, and the interacting term (AA ethnicity × pre-existing DM) did not demonstrate there to be an interaction between these two variables (*P*=.271).

DISCUSSION

This large, long-term cohort study in kidney transplant recipients demonstrates that AA ethnicity is a significant risk factor for graft failure and acute rejection, but not patient survival. The influence of ethnicity on graft loss is significantly modified by pre-existing DM and can be essentially nullified when controlled for baseline demographics, transplant characteristics, glycemic control, comorbidities, and pre- and post-transplant cardiovascular events. To our knowledge, this is the first large-scale study to specifically assess the impact of DM and cardiovascular risk factors on the disparities in graft survival with AA renal transplant recipients.

Consistent with our study and previous analyses, AA patients coming into renal transplantation have a number of important differences compared to non-AA patients, including a longer time on dialysis, a higher prevalence of DM and other cardiovascular risk factors and events, and more immunologic risks (less living donors and more HLA mismatches).[2,4,6–8] It is clear from this analysis that these confounding variables are the predominant factors driving the graft survival disparity. Univariate Kaplan-Meier and multivariable Cox proportional hazard regression analysis demonstrates that one of the most important variables for the higher rates of graft failure seen in AA

Table 3. Sequential multivariable modeling for death, graft loss, and acute rejection

Model	Reference Value	Mortality Hazard Ratio (95% CI)	Graft Loss Hazard Ratio (95% CI)	Acute Rejection Hazard Ratio (95% CI)
Unadjusted AA ethnicity	White	.98 (.73–1.31)	1.32 (1.04–1.68)	1.69 (1.33–2.15)
Unadjusted pre-existing DM	No DM	2.25 (1.68–3.00)	1.58 (1.25–2.00)	.81 (.63–1.04)
AA ethnicity adjusted for pre-existing DM	White	.87 (.65–1.18)	1.26 (.98–1.60)	1.76 (1.38–2.24)
AA ethnicity adjusted for DM and glycemic control	White	.82 (.60–1.11)	1.23 (.95–1.59)	1.76 (1.38–2.25)
Race fully adjusted model[a]	White	.70 (.48–1.02)	1.09 (.81–1.48)	1.57 (1.17–2.10)

AA, African American; DM, diabetes.

[a] Adjustments in the model include age, sex, body mass index, years on dialysis prior to transplant, pre existing congestive heart failure, coronary artery disease, dyslipidemia, stroke, coronary bypass grafting, percutaneous transluminal coronary angioplasty, acute myocardial infarction, peripheral vascular disease, valve replacement, hypertension, smoker, donor (living or expanded criteria), re-transplantation, human leukocyte antigen mismatch, panel reactive antibody, cold ischemic time, and warm ischemic time.

recipients is DM, which can be somewhat attenuated with glycemic control. In fact, the Kaplan-Meier curves for graft survival in patients without DM are nearly superimposed for AA and White transplant patients, while the curves in DM patients separate 18 months after transplant, and continued to diverge throughout the entire follow-up period. It is interesting to note that in both our study and large registry data,[1,11] ethnic disparities for graft survival do not appear until 2 years after transplant, mimicking the curves seen in the DM patients. This data, taken in context with the analysis presented here, leads to a potential conclusion that pre-transplant DM is one of the most important factors driving the graft survival disparities. Importantly, AA patients have a higher prevalence of pre-transplant DM and, because they referred later and are on dialysis for a substantially longer time before transplant, the DM is at a more advanced stage.[2,7] Thus, AA ethnicity is not necessarily the primary underlying mechanism driving the graft failure disparities, but rather a surrogate marker for the higher prevalence and more advanced state of cardiovascular risk factors known to influence graft survival.

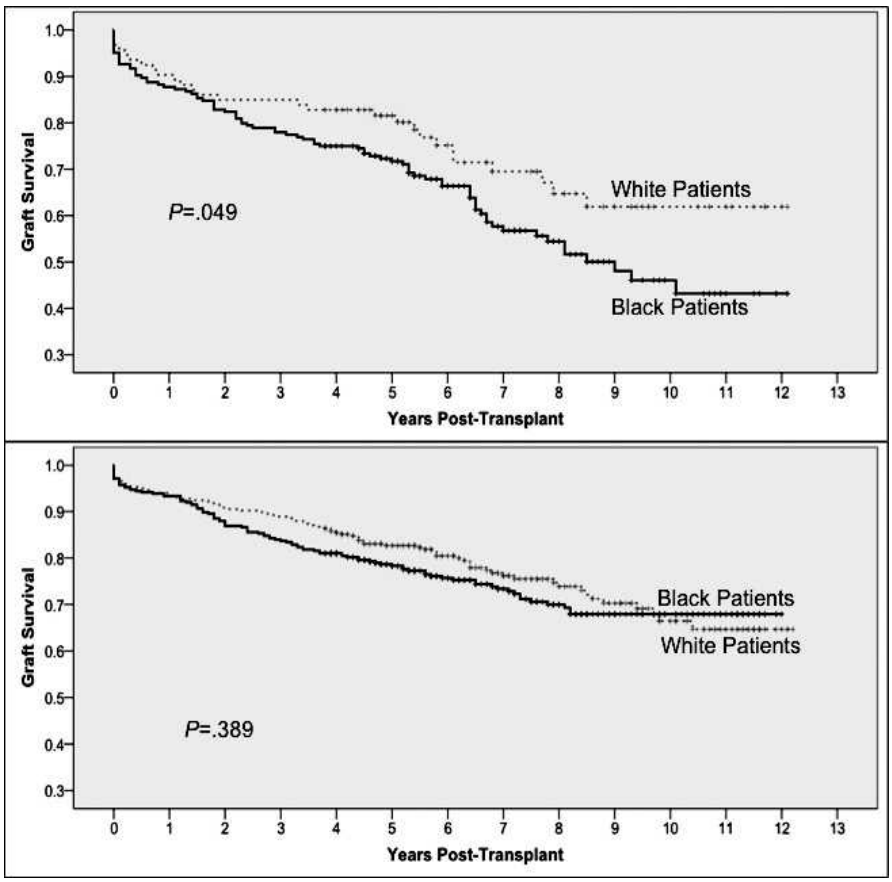

Fig 1. Survival curves for graft survival between ethnicities in patients with DM (top) and without DM (bottom)

This large, long-term cohort study in kidney transplant recipients demonstrates that AA ethnicity is a significant risk factor for graft failure and acute rejection, but not patient survival.

There are a number of well-conducted studies that have assessed the underlying mechanisms behind the disparities seen in AA kidney transplant recipients.[7,8] These risk factors are consistent with some of the data presented here. African American kidney transplant recipients spend a significantly longer time on dialysis prior to transplant; this likely reflects the fact that these patients are often referred to transplantation later than other ethnicities. African American patients also have less suitable living donors, which is a known significant predictor of improved graft survival. Interventions to reduce these two factors have been largely unsuccessful, but include a Centers for Medicare and Medicaid Services (CMS) mandate to refer all eligible dialysis patients for evaluation for transplant and the use of culturally sensitive focused efforts on identifying suitable living donors.[12] Unfortunately, these efforts have been largely unsuccessful. A review of the most recent US Renal Data System (USRDS) data demonstrates that 47% of AA patients placed on the transplant list were still waiting for a kidney after three years of listing; this compares to a rate of 31% in Whites. The rate of living donor transplantation per 100 dialysis patient years was .5 for AAs and 1.9 for Whites. Even more alarming, the number of living donations in AA patients has dropped from a high of 1,000 donations per year in 2003, to approximately 800 donations in 2007.[13]

Studies investigating the impact of cardiovascular risk factors and events on graft survival disparities within AA patients are surprisingly lacking. A few studies have focused on the severity and treatment of hypertension,[14,15] but we could not find any studies assessing DM or cardiovascular events on this disparity. Since efforts to improve early referral, transplantation, and living donation among AA end-stage renal disease patients have largely failed to decrease time to transplantation or increase the number of living donations, clinicians should focus on modifying the known variables that are driving the graft failure disparities after transplant. A focus on reducing acute rejection rates and improving disease state management, especially the important cardiovascular risk factor of DM, is desperately needed. Unfortunately, there are no well-conducted randomized controlled intervention trials demonstrating improved graft survival by modifying these risk factors in AA transplant patients. One small, short-term randomized control trial in hypertensive AA renal transplant patients demonstrated that a clinical pharmacist could improve blood pressure control through improved access to medications and adherence to regimens.[16] However, the study was not designed to determine the impact of these interventions of clinical outcomes, including graft survival.

There are several limitations in our study. First, because this was not a prospective study, we were not able to capture all possible confounding variables, including socioeconomic status, so it is possible that the results would be influenced if additional confounders were added to the models. Nevertheless, the consistency of our findings with those of prior studies suggests that our conclusions are valid. Second, data collection was performed by chart review and relied on patient's recall of family and medical history for baseline data, which makes our findings prone to recall bias. Third, hemoglobin A1C was not available for all patients; therefore, tight glycemic control was determined by either mean A1C or average serum glucose if the A1C was not available for analysis. By using average blood glucose for some patients, we likely underestimated actual glycemic control since there were only 1–2 values yearly for patients who were more than one year from transplant. Finally, CV events were likely underreported in this analysis because some of our patients may have had an event that was treated at an outside hospital and not captured with this analysis.

In conclusion, the results of our study demonstrate the strong and important impact of DM on the graft failure disparities seen in AA renal transplant recipients. Additionally, models that control for sociodemographic and pre- and post-transplant variables can nullify the impact of AA ethnicity of graft failure disparities. These data suggest that focusing on post-transplant cardiovascular risk factor control may be an important mechanism to reduce or eliminate the disparities within graft survival for AA transplant recipients.

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