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High mortality in diabetic recipients of high KDPI deceased donor kidneys

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Abstract

Background: Deceased donor (DD) kidney quality is determined by calculating the Kidney Donor Profile Index (KDPI). Optimizing high KDPI (≥85%) DD transplant outcome is challenging. This retrospective study was performed to review our high KDPI DD transplant results to identify clinical practices that can improve future outcomes.

Methods: We retrospectively calculated the KDPI for 895 DD kidney recipients transplanted between 1/2002 and 11/2013. Age, race, body mass index (BMI), retransplantation, gender, diabetes (DM), dialysis time, and preexisting coronary artery disease (CAD) (previous myocardial infarction (MI), coronary artery bypass (CABG), or stenting) were determined for all recipients.

Results: About 29.7% (266/895) of transplants were from donors with a KDPI ≥85%. By Cox regression older age, diabetes, female gender, and dialysis time >4 years correlated with shorter patient survival time. Diabetics with CAD who received a high KDPI donor kidney had a significantly increased risk of death (HR 4.33 (CI 1.82–10.30), *P*=.001) compared to low KDPI kidney recipients. The Kaplan-Meier survival curve for diabetic recipients of high KDPI kidneys was significantly worse if they had preexisting CAD (*P*<.001 by log-rank test).

Conclusion: Patient survival using high KDPI donor kidneys may be improved by avoiding diabetic candidates with preexisting CAD.

KEYWORDS
deceased donor, diabetes, kidney transplant, patient outcomes, survival

1 | INTRODUCTION

The availability of deceased donor kidneys in the United States is not keeping pace with the demand resulting in an ever-increasing number of candidates accruing on the waiting list.[1] One initiative to maximize available deceased donor kidneys to meet this demand is using higher risk, or "marginal" donor kidneys for transplantation. Pre-transplant identification of these higher risk donor kidneys is currently determined by UNOS using a calculated kidney donor profile index (KDPI) that includes clinical parameters and donor demographics.[2] Recipients of these higher risk kidneys have reduced rates of patient and graft survival.[3,4] In the current setting of transplant program outcomes oversight by CMS and UNOS, there may be a perceived disincentive to transplant these organs, even though survival outcomes are risk adjusted, as they will adversely impact unadjusted survival outcomes.

Transplant programs trying to successfully meet the challenge of simultaneously maximizing kidney utilization and survival outcomes would benefit from knowing which recipients have the best, and worst, outcomes after receiving a higher risk donor kidney. Even higher risk recipients, such as diabetic and older candidates, enjoy significantly improved survivals with transplantation compared to

remaining on dialysis.[5] Yet, this fact bears no direct consequence on UNOS and CMS estimation of acceptable program outcomes. Rather, post-transplant outcomes are risk stratified based on currently collected donor and recipient parameters.[6] However, there are significant recipient comorbidities that are known at the time of transplantation and that are not included in Scientific Registry of Transplant Recipients (SRTR) risk stratification analyses including pulmonary, cardiac and liver disease.

Because the presence of such comorbidities is currently transparent in SRTR outcomes analyses, transplant programs must determine for themselves which candidate cohorts have the best outcomes after receiving higher risk donor kidneys. Thus, we performed these analyses to enhance the ability of our program to achieve excellent outcomes while maximizing our use of higher risk donor kidneys.

2 | MATERIALS AND METHODS

2.1 | Patients

Complete data were available to retrospectively calculate the kidney donor risk index (KDRI) for 895 of 1221 recipients of deceased donor kidneys transplanted between 1/2002 and 11/2013. Recipient age, race, gender, body mass index (BMI), retransplantation, diabetes (DM), dialysis time, and preexisting coronary artery disease (CAD=previous myocardial infarction [MI], coronary artery bypass [CABG], or coronary artery stenting) were determined for all recipients.

2.2 | KDRI calculation and KDPI≥determination

Kidney donor risk index was calculated using the methods described in the "Guide to calculating and interpreting KDPI" found on the OPTN Web site.[1] KDPI ≥85% was determined using the OPTN KDRI to KDPI mapping table found on the OPTN Web site[2] (reference population of all 2013 US deceased donors).

2.3 | Statistical analyses

Student's *t* test, Fisher's exact test, and Pearson chi-square test were used for statistical comparison of means and proportions between groups, respectively, where appropriate. Cox regression models were used where indicated. Kaplan-Meier survival curves were compared by log-rank test. All statistical analyses were performed using IBM SPSS version 22.0.0 statistical software (Armonk, NY, USA) and Stata statistical software version 13.1 (College Station, TX, USA).

3 | RESULTS

3.1 | Recipient demographics

The mean age of the 895 transplant recipients was 52.2±11.9 years (range 14.5–82.8 years). This recipient cohort includes 543 (60.7%) Caucasians, 302 (33.7%) African Americans, and 50 (5.6%) other races, and there are 543 (60.7%) males and 352 (39.7%) females. Their mean BMI was 30.0±6.4 (range 14.6–52.3). Of the 895 recipients, 837 were on dialysis pre-transplant with a mean time of 1289±917 days (range 30–8491). Eighty-nine (9.9%) of the recipients were retransplants, and 280 (31.3%) were diabetic. One hundred and eleven recipients (12.4%, 111/895) had known pre-transplant CAD as defined in the methods. The KDPI of the kidney donor was ≥85% in 266 (29.7%) of all transplants (Table 1). Comparing recipient demographic variables between donor kidney KDPI<85% recipients (low KDPI group) and donor KDPI ≥85% recipients (HiKDPI group), the HiKDPI group recipients were older (*P*<.001) and had fewer African Americans (*P*=.05) (Table 1). This study was approved by our IRB (study #2014H0199).

3.2 | Clinical outcomes

3.2.1 | Renal function

The MDRD GFR was calculated for recipients at 1, 6, 12, 24, and 36 months post-transplant. As expected, the GFR was consistently 10–12 cc/min lower for the HiKDPI vs low KDPI group (*P*<.001

TABLE 1 Recipient demographics stratified by donor kidney KDPI status

Variable	All recipients (n=895)	Low KDPI grp (n=629)	HiKDPI grp (n=266)	Significance
Age (y)	52.2±11.9	50.3±11.9	56.6±11.0	<.001
BMI	30.0±6.4	29.8±6.5	30.6±6.1	ns
Female gender	352 (39.7%)	243 (38.6%)	109 (41.0%)	ns
AA race	302 (33.7%)	225 (35.8%)	77 (28.9%)	.05
Retransplant	89 (9.9%)	68 (10.8%)	21 (7.9%)	ns
Diabetes	280 (31.3%)	185 (29.4%)	95 (35.7%)	ns
Dialysis (d)	1289±917	1315±937	1225±862	ns
Pre-tx CAD	111 (12.4%)	78 (12.4%)	33 (12.4%)	ns

BMI, body mass index; Cauc, Caucasian; AA, African American; Pre-Tx, pre-transplant; CAD, coronary artery disease; grp, group.

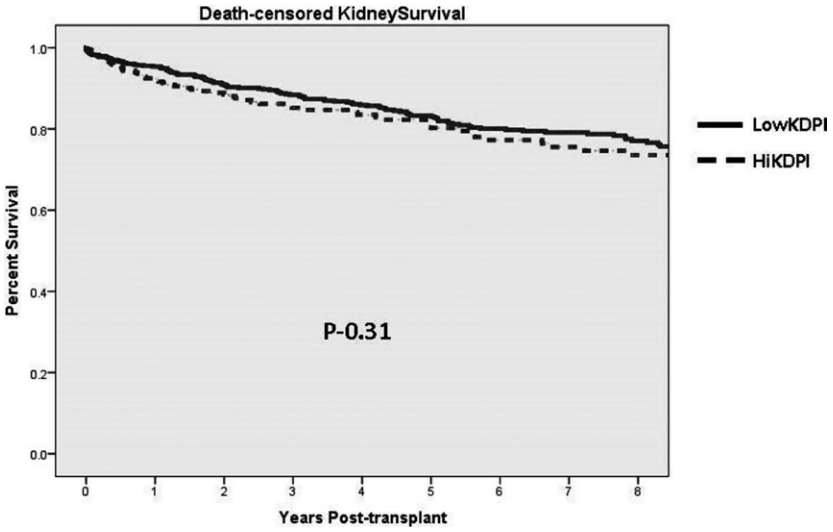

FIGURE 1 Comparison of Kaplan-Meier death-censored kidney survival curves for low KDPI (<85%) vs HiKDPI (≥85%) donor kidney recipients (*P*=.31 by log-rank test)

for all time points) (43.5±19.8 vs 32.4±15.9 at 1 month, 49.6±20.3 vs 38.0±16.3 at 6 months, 46.9±18.6 vs 36.2±15.6 at 12 months, 46.2±18.1 vs 35.4±15.1 at 24 months, and 45.9±19.0 vs 35.5±15.0 at 36 months).

3.2.2 | Survival

Recipient survival and death-censored kidney survival were compared between the low KDPI and HiKDPI groups. The death-censored Kaplan-Meier kidney survival curves were similar between groups (*P*=.31, log-rank test) (Fig. 1); however, the patient survival curves were significantly different (*P*<.001, log-rank test) (Fig. 2).

3.2.3 | Patient survival correlates

Although the HiKDPI group has a higher incidence of patient death, other clinical variables such as age, gender, diabetes, preexisting CAD, and >4 years on dialysis also correlate with a significantly higher incidence of death by univariate analysis (Table 2). A Cox regression model of patient survival with these variables identified older age, female gender, diabetes, and dialysis time >4 years as correlating with a shorter survival time (Table 2). All possible interactions between DM, CAD, and KDPI group were forced into another Cox regression model of patient survival. This model revealed that diabetics with CAD that received a high KDPI donor kidney had a significantly increased risk of death (HR 4.33 [CI 1.82–10.30], *P*=.001) over those who received a low KDPI kidney (Table 3). Other Cox models that included all possible interactive terms between KDPI, gender, age, and dialysis duration revealed that these interactive terms were not significant explanatory variables for patient death. Comparison of patient survival curves for diabetic recipients stratified by KDPI group and preexisting CAD demonstrates a significantly worse survival for diabetics with previously treated CAD in the HiKDPI group (Fig. 3). Patient survival for non-diabetic recipients without preexisting CAD in the low KDPI group is included for reference.

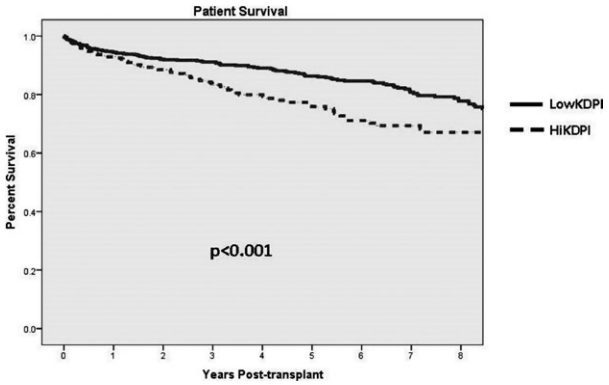

FIGURE 2 Comparison of Kaplan-Meier patient survival curves for low KDPI (<85%) vs HiKDPI (≥85%) donor kidney recipients (*P*<.001 by log-rank test). A reference cohort (DM (diabetics) on dialysis) is included for visual comparison

3.2.4 | Cause of death

The cause of death was determined for all 223 deaths that occurred in the patient cohort. Compared to a reference group consisting of non-diabetic recipients without preexisting CAD that received a kidney from a KDPI <85% donor, diabetic recipients had a significantly higher percent of deaths related to cardiovascular disease (*P*<.001, chi-square analysis, data not shown). Cardiovascular disease and all-cause mortality were stratified by recipient diabetic status, preexisting CAD, and donor KDPI group (Table 4). The highest percentage of cardiovascular disease mortality is seen in the HiKDPI group with CAD.

4 | DISCUSSION

Deceased donor KDPI is incorporated into current UNOS kidney allocation. Post-transplant patient[3] and graft[3,4] survivals worsen as

TABLE 2 Correlation of clinical variables and patient death by univariate and multivariable analysis

	Univariate[a]			Multivariable[b]	
Variable	No death (n=672)	Death (n=223)	Significance	HR	Significance
Recipient age	50.3±11.9	56.6±11.0	<.001	1.05 (1.03–1.06)	<.001
BMI	29.8±6.8	30.6±6.1	.060	0.99 (0.97–1.02)	.623
Female gender	41.7% (280)	32.3% (72)	.013	0.73 (0.55–0.97)	.028
AA ethnicity	34.8% (234)	30.5% (68)	.236	0.79 (0.58–1.05)	.108
Retransplant	10.9% (73)	7.2% (16)	.111	1.00 (0.59–1.69)	.999
Diabetes	26.9% (181)	44.4% (99)	<.001	1.65 (1.26–2.17)	<.001
Dialysis ≥4 y	26.9% (181)	34.1% (76)	.041	1.92 (1.44–2.56)	<.001
Pre-tx CAD	10.4% (70)	18.4% (41)	.002	1.18 (0.83–1.69)	.362
Donor KDPI ≥85%	27.5% (185)	36.3% (81)	.013	1.28 (0.96–1.72)	.095

BMI, body mass index; AA, African American; Pre-Tx, pre-transplant; CAD, coronary artery disease; KDPI, kidney donor profile index; HR, hazard ratio.
[a]Univariate analysis by Student's *t* test or chi-square test.
[b]Multivariable Cox proportional hazards regression model.

TABLE 3 Analysis of patient survival by Cox regression analysis forcing recipient diabetes, preexisitng CAD, and KDPI ≥85% variable interactions

KDPI ≥85% (Yes vs No)			
Diabetes	CAD	HR (95% CI)	Significance
No	No	1.01 (0.66–1.55)	.962
No	Yes	1.05 (0.34–3.20)	.75
Yes	No	1.23 (0.77–1.97)	.384
Yes	Yes	4.33 (1.82–10.30)	.001
Female gender		−0.33 (−0.61 to −0.04)	.024
Age		0.05 (0.03–0.06)	<.001
Dialysis >4 years		0.69 (0.40–0.99)	<.001
AA race		−0.31 (−0.61 to −0.01)	.044

KDPI, kidney donor profile index; HR, hazard ratio; CI, confidence interval; CAD, coronary artery disease; AA, African American.

KDPI quintiles increase. Given the regulatory oversight of transplant programs and the consequences of these programs being identified as "underperforming," transplant professionals are challenged with the task of maximizing the use of "low-quality" high KDPI organs while maintaining excellence in patient and graft survivals. This study was performed to assist our program in meeting this challenge.

Initial analyses of our patient cohort indicated that patient survival but not death-censored graft survival was significantly worse for recipients of high KDPI (≥85%) donor kidneys (Fig. 2). Examining our HiKDPI group, we found they were significantly older and non-African American compared to the low KDPI group (Table 1). However, when analyzing patient death by univariate analyses and survival by Cox regression models, we identified a number of explanatory variables including recipient age, gender, diabetes, and dialysis time >4 years by univariate and Cox regression analyses plus preexisting CAD and high KDPI by univariate analysis (Table 2). Many of these

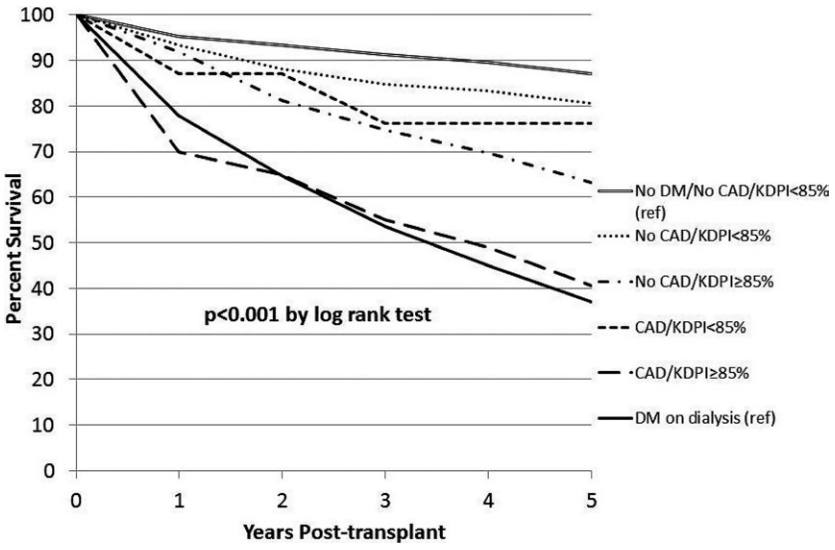

FIGURE 3 Comparison of patient survival curves for diabetic recipients stratified into four groups based on preexisting CAD and kidney donor KDPI (No CAD/KDPI <85%; No CAD/KDPI ≥85%; CAD/KDPI <85%; CAD/KDPI ≥85%) (*P*<.001, by log-rank test). Reference cohorts (No diabetes/No CAD/KDPI <85%) and (DM [diabetics] on dialysis) are included for visual comparison

TABLE 4 Recipient CVD and all-cause mortality stratified by diabetes, preexisting CAD, and KDPI ≥donor kidney status

DM/CAD/KDPI ≥85%	CVD mortality	All-cause mortality
No/No/No	**2.5%** (10/397)	**17.9%** (71/397)
Yes/No/No	**11.0%** (17/154)	**31.2%** (48/154)
Yes/Yes/No	**12.9%** (4/31)	**29%** (9/31)
Yes/No/Yes	**12.0%** (9/75)	**38.7%** (29/75)
Yes/Yes/Yes	**30.0%** (6/20)	**65%** (13/20)

DM, diabetes; CAD, preexisting coronary artery disease; KDPI, kidney donor profile index; CVD, cardiovascular disease. No/No/No = No DM/No CAD/No KDPI ≥85%.

variables have been previously reported to correlate with patient survival. One-, 5-, and 10-year patient survival is lower for males and has been shown to decline as recipient age increases, consistent with the outcomes in our study cohort.[7] Cosio et al.[8] previously reported a significantly worse post-transplant survival and a higher rate of CVD cause of death for diabetics vs non-diabetics as noted in our study. Goldfarb-Rumyantzev et al.[9] reported a worsening patient survival as the duration of ESRD increases, and Meier-Kriesche et al.[10] found longer dialysis times, diabetes, and older age increase the risk of post-transplant death, all consistent with the findings in our study.

Interestingly, HiKDPI group designation and preexisting CAD alone were not significant explanatory variables in the Cox regression model. However, a Cox model that included all interactions between DM, CAD, and KDPI group demonstrated that diabetics with preexisting CAD had a significantly worse survival when they received a kidney from a KDPI ≥85% vs <85% donor (Table 3). Heaphy et al.[11] recently reported a significant interaction between recipient diabetes and donor KDRI on the risk of recipient death and graft loss following DDRT in a cohort of 109,392 adult recipients from the SRTR database. Our analyses may extend their findings by demonstrating that the risk of death is disproportionately higher for diabetics with preexisting CAD.

Wolfe et al.[5] reported a nearly 50% reduction in death rate for patients with diabetes transplanted vs those remaining on the waiting list. Left ventricular hypertrophy has been shown to significantly improve after successful renal transplantation.[12,13] Diabetic recipients have a significant reduction in the risk of hospitalization for acute coronary syndromes following transplantation.[14] However, after graft loss, acute coronary syndrome (using ICD-9 codes), with the associated increased mortality, has been reported to occur at a rate 2–3 times higher vs prior to graft loss.[15] Taken together, these data suggest that adequate renal function ameliorates the increased risk of post-transplant cardiovascular disease-related death in diabetic recipients. Conversely, poor renal function may fail to do so. Several mechanisms have been proposed to explain this relationship including improvement of pretransplant hyperparathyroidism, reduced homocysteine levels, and better volume control, (reviewed in[14]).

In our patient cohort, the renal function for the HiKDPI group was significantly worse during the first 3 post-transplant years. This retrospective study cannot determine whether renal dysfunction associated volume overload contributed to the higher observed mortality in the HiKDPI group. Previous studies have reported higher all-cause mortality[16,17] and cardiovascular-cause mortality[16] associated with greater fluid overload in dialysis patients. Also diabetic patients with larger dialysis-associated weight loss indicating more severe volume expansion and contraction have been reported to have a higher risk of mortality.[18] The post-transplant cumulative incidence of de novo CHF in the US has been reported to be 10.2% increasing to 18.3% at 1 and 3 years post-transplant.[19] The USRDS annual report shows for Medicare patients transplanted from 2007 through 2009 hospitalization for CHF was 21% in the first post-transplant year,[20] suggesting post-transplant clinically significant volume overexpansion is relatively common. Currently, there are no reports specifically linking post-transplant volume expansion with higher recipient death rates or cardiac-related complications.

As might be expected, the death rate was higher in our diabetic recipients with or without preexisting CAD. However, the highest mortality rate (65%) was seen for those with preexisting CAD that received a high KDPI donor kidney (Table 4). Lentine et al.[21] found, using USRDS registry data, that both recipient factors (age, diabetes, MI, and PVD) and donor factors (age and DGF) were risk factors for post-transplant MI and that post-transplant MI, in turn, was a risk factor for death. Our results are consistent with these findings with the addition of demonstrating an interaction between recipient factors (diabetes) and donor factors (high KDPI).

Poorer post-transplant renal function has been shown to correlate with a higher risk of death. Using 1995–2003 USRDS data, Schnitzer et al.[22] reported a higher risk of death as the 1 year post-transplant renal function worsened with an adjusted hazard ratio of 2.33 for death between 1 and 3 years post-transplant for recipients with an estimated GFR (eGFR) <30 mL/min/1.73 m^2 at the end of the first year. Meier-Kriesche et al.[23] reported similar findings for cardiovascular cause of death using 1988–1998 USRDS data; the adjusted death hazard ratio was 2.26 if the serum creatinine was between 2.6 and 4.0 mg/dL at the end of the first year. Our results are consistent with these studies. Our HiKDPI group (with a worse mean eGFR) had a 3-year mortality of 17.7% vs 10.2% for the low KDPI group (P=.002, data not shown).

Limitations of this study include a small sample population from a single center, and it is retrospective and thus non-randomized. However, there is currently no multicenter, prospective study comparing outcomes of diabetic candidates stratified by donor KDPI. Also, the cause of death could not be determined in 30% of cases (68/223) limiting our ability to definitively quantitate and analyze cardiovascular-cause mortality. Finally, there is a possibility of a selection bias regarding candidates chosen to receive kidneys from less ideal donors (high KDPI donor organs) in this cohort. Such an historical selection bias would not be captured by this retrospective study.

5 | CONCLUSION

Given the 76% 5-year patient survival for diabetics with preexisting CAD that were transplanted with a kidney from a <85% KDPI

donor in this study compared to 36.8% unadjusted 5-year survival reported by the USRDS in 2013 for diabetics on dialysis,[7] we feel such high comorbidity recipients should continue to be provided access to renal transplantation at our center. However, in view of the higher mortality for diabetics with preexisting CAD who received a kidney from a ≥85% KDPI donor, we currently avoid this donor/recipient combination while continuing to refine our strategy for the optimal use of such higher risk kidneys.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHORS' CONTRIBUTIONS

Ronald P. Pelletier participated in concept and design of the study, data collection, writing of the manuscript, and data analysis. Todd E. Pesavento participated in writing, approval, and critical review of the manuscript and data analysis. Amer Rajab participated in writing, approval, and critical review of the manuscript and data analysis. Mitchell L. Henry participated in concept and design of the study, data analysis, and critical review of the manuscript.

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