And Then There Were Three: Effects of Pretransplant Dialysis on Multiorgan Transplantation

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INTRODUCTION

The number of multiorgan transplants (MOTs) is rising. A large proportion of MOTs are simultaneous liver-kidney (SLK) and simultaneous heart-kidney (SHK) transplants. These transplants treat the kidney as a “secondary” organ, offered to individuals with a failing nonrenal organ and concomitant kidney disease. According to United Network of Organ Sharing (UNOS) data, 6% of deceased donor kidneys in 2018 and 2019 were allocated as “secondary” organs in this manner. The rise of SLK and SHKs against a background of organ shortage has been the center of a vigorous controversy. In 2019, UNOS released a white paper, “Ethical Implications of Multi-Organ Transplants,” recommending, among other things, that “[d]ata for each MOT combination should be made publicly available to foster transparency.”

A prerequisite to fostering transparency in MOT is the proper selection of metrics that reflect what they measure. In the case of MOTs using kidney as a secondary organ, the current kidney outcome metric, observed kidney allograft survival, is flawed. In contrast to orthotopic liver and heart transplants, requiring removal of the native liver or heart, the native kidneys remain in place in heterotopic kidney transplants. Consequently, when allograft failure is defined as returning to dialysis or requiring another kidney transplant, what it reflects is failure of both the transplant kidney and the native kidneys (Figure 1). In the setting of deceased donor kidney transplantation, especially in the current era of prolonged wait time on dialysis, residual kidney function at the time of multiorgan transplantation are less likely to have apparent failure of the kidney allograft. Whether residual kidney function facilitates function of the allograft or whether some SLK and SHK recipients have 3 functional kidneys is unknown. Sustained kidney function after SLK and SHK transplants does not necessarily indicate successful MOT.
is allowed in kidney-alone transplantation. In the OPTN/UNOS medical eligibility criteria for simultaneous liver-kidney (SLK) transplants, for instance, a patient may become eligible for SLK if he/she had an estimated glomerular filtration rate (eGFR) <60 mL/min for 3 months and 1 eGFR value <35 mL/min. Such liberal criteria can create a lead time bias, as has been proposed in preemptive kidney transplantation. Furthermore, the cause of kidney disease in SLK and SHK transplants is frequently acute kidney injury, the reversibility of which is difficult to predict. Nuclear imaging studies have previously demonstrated significant native kidney function in selected SLK recipients, but these findings have not been generalized to the larger transplant population. The extent to which native kidney function may skew observed kidney allograft survival is therefore unknown.

In addition to carrying implications for monitoring programs, the extent to which observed kidney allograft outcome reflects native kidney function also indirectly estimates the extent of “prophylactic” transplant under current practice. Prophylactic transplants refer to SLK or SHK in nondialysis-dependent patients, in an attempt to improve overall patient survival posttransplant. In an era of worsening organ shortage, directing kidneys away from dialysis-dependent patients who nearly always would benefit from a transplanted kidney to nonrenal organ candidates who may benefit from a transplanted kidney is controversial. Quantifying the magnitude of prophylactic kidney transplantation, which are perhaps premature or even “unnecessary,” helps inform more rational MOT policy development.

We hypothesize that residual (native) kidney function attenuates the risk of apparent kidney allograft failure, as defined as return to dialysis or a low level of eGFR qualifying for kidney retransplantation. Because information on residual kidney function is not readily available, we use pretransplant dialysis duration as a practical, albeit imperfect, proxy. Apparent kidney allograft failure is typically blamed on allograft failure, but it may represent allograft failure plus accelerated loss of native kidney function after SLK and SHK. Studying the association between pretransplant dialysis duration and apparent kidney allograft failure may help quantify the extent of this phenomenon.

**MATERIALS AND METHODS**

**Data Source and Cohort Assembly**

We used the Scientific Registry of Transplant Recipients (SRTR), which contains deidentified data on all solid organ transplant donors, candidates, and recipients in the United States. The SRTR incorporates dialysis start dates from the Centers of Medicare and Medicaid Services, thus ensuring accurate ascertainment of apparent kidney allograft failure.

We identified all adult SLK and SHK recipients from January 1, 1995, through December 31, 2014, who had complete kidney outcome data (either death, kidney allograft failure, or a reported serum creatinine during follow-up) to enable us to calculate eGFR by the CKD-EPI formula at 6 and 12 months. We defined SLK and SHK transplantation as deceased donor liver/heart and kidney transplantations occurring within 2 days in the same recipient. We chose the study start date as a full complement of donor characteristics became available in 1995. We chose the study end date as our database version is dated November 2017: the end date of December 2014 therefore allowed enough time to complete nearly 3 years of follow-up before censoring of survival time. Exclusion criteria included other concomitant solid organ transplantations, duplicate entries in the dataset, omission of primary outcome (eGFR at 6 and 12 mo), and omission of model covariates (Figure 2).

**Exposure and Covariates**

We used pretransplant dialysis duration as a proxy for residual kidney function, dividing pretransplant dialysis duration into 3 categories: ≥90 days (low residual kidney function), <90 days (intermediate residual kidney function), and no dialysis (high residual kidney function). We stratified each analysis by SLK and SHK and, unless stated otherwise, adjusted all analyses for the following covariates:

1. **Era:** year of transplant;
2. **Donor characteristics:** Kidney Donor Recipient Index, a marker of donor kidney quality, calculated from 10 donor characteristics, normalized to the 2017 median;
3. **Recipient comorbidities:** Age, sex, race, insurance status, kidney diagnosis, liver/heart diagnoses, nonkidney life support at time of transplant;
4. **Transplant characteristics:** Kidney and liver/heart cold ischemia times. Where cold ischemia time is missing for 1 organ but not the other, we imputed missing time using the assumption that the liver and heart precede the kidney implantation by 5 and 10 hours, respectively. (Five and 10 h were the mean difference in heart/kidney and liver/kidney cold ischemia time, respectively, in the nonmissing data.)
Primary Outcome

Our primary outcome was the difference in the probability of apparent kidney allograft failure between the groups with no dialysis exposure (presumed high residual kidney function) and >90 days dialysis exposure (presumed low residual kidney function) at 6- and 12-month posttransplant, adjusted for differences in baseline characteristics including disease severity. This outcome was our best indirect estimate for the extent of “prophylactic” transplantation. To calculate the frequency of this outcome, we examined mutually exclusive occurrences of death, apparent kidney allograft failure (reported kidney allograft failure or eGFR < 20 mL/min), and no event (alive with eGFR ≥ 20 mL/min) at 6 and 12 months by a multinomial regression model. The multinomial regression model is an extension of the logistic regression model allowing for >2 outcomes. In the 25% of cases where both death and allograft failure occurred by month 6 or 12, we chose to adjudicate the event as death although allograft failure may precede death. We made this decision because disease severity confounds the relation between pretransplant dialysis duration and apparent allograft failure. We therefore made the conservative estimate that all episodes of allograft failure shortly preceding death were due to disease severity rather than residual kidney function. Such a choice biased our primary outcome toward the null, underestimating the true association of residual kidney function with apparent allograft survival. We used predicted probabilities from the multinomial model to estimate the difference in apparent kidney allograft failure between the different pretransplant dialysis duration (residual kidney function) groups, with and without adjustment for covariates.

Secondary Outcomes

Our secondary outcomes included:

(1) Delayed kidney allograft function: Results among the 3 groups were compared using the chi-square test.
(2) eGFR at 1 year in patients without death or apparent allograft failure: Results among the 3 groups were compared using the t-test.
(3) Death and apparent allograft failure after 1 year: In a landmark analysis, we restricted our analysis to patients who survived to 1 year without death or apparent kidney allograft failure. We applied the Fine and Gray extension to the traditional proportional hazards (Cox) regression model, employing the proportional subdistribution hazards model to examine the association between pretransplant dialysis exposure and death or apparent kidney allograft failure in a competing risk framework. We examined Schoenfeld residuals to examine the validity of the proportional hazards assumption.

RESULTS

Cohort and Baseline Characteristics

Our final cohort consisted of 5723 adult MOT recipients (4875 SLK recipients and 848 SHK recipients). Of the SLK
recipients, 1533 (31%) received ≥90 days of dialysis, 1567 (32%) received <90 days of dialysis, and 1775 (36%) received no dialysis before transplant. Of the SHK recipients, 304 (36%) received ≥90 days of dialysis, 95 (11%) received <90 days of dialysis, and 449 (53%) received no dialysis before transplant. Table 1 outlines their baseline characteristics. Markers of disease severity, as manifested by nonkidney life support and biochemical indices (available for SLK), were highest in the groups receiving <90 days of dialysis and lowest in the groups receiving no dialysis. Acute kidney injury (often hepatorenal syndrome in the case of SLK) was the primary kidney diagnosis in 36% and 24% of SLK and SHK recipients, respectively. Diabetes mellitus and prior solid organ transplants, on the other hand, were most common in SLK patients receiving no dialysis and not significantly different among the SHK groups. Other comorbidities were generally present in <10% of all groups.

**Primary Outcome**

At 6 and 12 months, the likelihood of event-free survival (no death or apparent kidney allograft failure) was highest in SLK and SHK recipients with no pretransplant dialysis exposure (Figure 3). Figure S1 (SDC, http://links.lww.com/TXD/A303) shows results before multivariable adjustment. When the events were parsed into death and apparent kidney allograft failure, no pretransplant dialysis exposure was associated with a 1%–3% lower probability of apparent kidney allograft failure at 12 months in SLK and SHK recipients (Table 2). Intermediate dialysis exposure, or <90 days of dialysis exposure, was associated with a statistically nonsignificant 1%–3% increase in likelihood of death at 12 months in SLK and SHK recipients (Table 2, also in Table S1, SDC, http://links.lww.com/TXD/A303, where P values are available in parentheses). In 25% of all events before 1 year, apparent kidney allograft failure preceded death by 47 ± 70 days (Table S2, SDC, http://links.lww.com/TXD/A303).

**Secondary Outcomes**

**Delayed Graft Function**

After SLK, delayed kidney allograft function occurred in 26%, 25%, and 10% of patients requiring ≥90 days of dialysis, <90 days of dialysis, and no dialysis pretransplant, respectively (P < 0.001). After SHK, delayed kidney allograft function occurred in 38%, 26%, and 16% of patients requiring ≥90 days of dialysis, <90 days of dialysis, and no dialysis pretransplant, respectively (P < 0.001).

eGFR at 1 year in patients with no events: 3978 SLK and 706 SHK recipients survived to 1 year without apparent kidney allograft failure. eGFR was highest in SHK recipients with <90 days dialysis exposure (Table 3; P = 0.004).

Death and apparent kidney allograft failure beyond 1 year: over a median follow-up of 5.7 years, 365 (22%) SLK recipients died and 292 (17%) experienced kidney allograft failure. No pretransplant dialysis exposure (presumed high residual kidney function) was associated with a higher risk of death and a lower hazard for apparent kidney allograft failure (Table 3). Over a median follow-up of 6.2 years, 131 (19%) SHK recipients died and 61 (9%) experienced kidney allograft failure. In SHK recipients, a similar trend in apparent kidney allograft failure to SLK was observed, but the association was not statistically significant after multivariable adjustment (Table 3).

**DISCUSSION**

In this registry-based study, we demonstrate that pretransplant dialysis duration, an imperfect proxy of residual kidney function at the time of transplant, is associated with apparent kidney allograft failure after multiorgan transplantation involving the kidneys, that is, SLK and SHK. We find a 1%–3% lower risk of kidney allograft failure at 12 months in SLK/SHK recipients who did not require dialysis compared with SLK/SHK recipients who required >90 days of dialysis before transplant. This association remains significant after adjusting for donor, recipient, and transplant characteristics, including markers of disease severity. We submit that at least 1%–3% SLK/SHK transplants in patients who did not require pretransplant dialysis may be considered “prophylactic”; that is, even without the kidney component of these MOTs, these recipients may have had sufficient residual kidney function to stave off dialysis at 1-year posttransplant. While the estimated effect size is small, we feel that it is a lower bound for the extent of “prophylactic” kidney transplants, for reasons we will discuss below. More studies are warranted to uncover the upper bound of the true effect.

Our results complement prior single-center nuclear imaging studies examining native kidney function in SLK recipients. In the largest of these studies, in 39 of 78 SLK recipients (51%) had native GFRs >20 mL/min at an average of 1-year posttransplant. Given the high geographic variability in the SLK/SHK use, extrapolating from single-center studies is challenging. Our estimate of 1%–3% should be regarded as the lowest estimate of prophylactic kidney transplant, as native kidneys may be contributing concurrently with the transplant kidney and thus evade recognition by the endpoint of apparent kidney allograft failure. Indeed, the lower long-term apparent kidney allograft failure rate in SLK recipients without pretransplant dialysis exposure suggests that a subset of SLK recipients have 3 functional kidneys. We also used a very conservative estimate of allograft failure, adjudicating cases in which both allograft failure and death occurred within 1 year as death. An alternative adjudication will likely increase the estimate of prophylactic kidney transplant.

While several studies have addressed these issues in SLK transplantation, we are not aware of prior studies examining the association between dialysis exposure and apparent kidney allograft failure in SHK recipients. Our findings in SHK are similar in direction and magnitude to those observed in SLK transplantation; the precision of our estimates is limited owing to much smaller sample size. Thus, our analyses confirm and extend previously published findings from single-center studies of SLK recipients to the entire SLK/SHK transplant population in the United States.

Our analysis has several strengths. We utilized a national dataset (the SRTR), improving generalizability relative to single-center or regional studies. The sample size was relatively large, increasing the power to detect modest associations. We conducted multivariable analyses to account for confounding by factors related to kidney function over time.

There are also several important limitations to our analysis. First and foremost, the SRTR data had no direct measure of residual kidney function. The need for, and duration of, preoperative dialysis are the closest proxies, but are clearly imperfect. For instance, if in the throes of end-stage liver or heart failure a patient with normal or near normal kidney function at experiences an episode of reversible acute kidney injury
## TABLE 1
Baseline characteristics in SLK and SHK transplants, stratified by pretransplant dialysis exposure

| Baseline characteristic | SLK (N = 4875) | SHK (N = 848) |
|-------------------------|---------------|---------------|
|                         | Pretransplant dialysis duration | Pretransplant dialysis duration |
|                         | ≥90 d (low) | <90 d (intermediate) | None (high) | ≥90 d (low) | <90 d (intermediate) | None (high) |
| Age (y) | 56 (49–62) | 57 (51–63) | 58 (52–63) | <0.001 | 52 (43–60) | 56 (45–62) | 59 (52–64) | <0.001 |
| Sex (% female) | 508 (33%) | 561 (36%) | 621 (35%) | 0.3 | 63 (21%) | 20 (21%) | 101 (22%) | 0.8 |
| Race (%) | White 1177 (77%) | 1296 (83%) | 1429 (81%) | <0.001 | 190 (63%) | 69 (73%) | 321 (71%) | 0.1 |
| Black 265 (17%) | 196 (13%) | 275 (15%) | 97 (32%) | 24 (25%) | 115 (26%) | 31 (7%) | 31 (7%) |
| Others 91 (6%) | 75 (5%) | 71 (4%) | 17 (6%) | 2 (2%) | 13 (3%) |
| Ethnicity (% Hispanic) | 275 (18%) | 274 (17%) | 235 (13%) | <0.001 | 32 (11%) | 6 (6%) | 29 (6%) | 0.1 |
| Education (% college or higher) | 600 (39%) | 548 (35%) | 604 (34%) | 0.006 | 123 (40%) | 34 (36%) | 177 (39%) | 0.7 |
| Primary payer (% private/self-pay) | 581 (38%) | 836 (53%) | 953 (54%) | <0.001 | 98 (32%) | 56 (59%) | 250 (56%) |
| Primary kidney disease diagnosis (%) | AKI/Hepatorenal syndrome* | 341 (22%) | 849 (54%) | 580 (33%) | <0.001 | 55 (18%) | 38 (40%) | 108 (24%) |
| Diabetic nephropathy | 309 (20%) | 230 (15%) | 318 (18%) | 58 (19%) | 17 (18%) | 92 (29%) |
| Glomerulonephritis | 244 (16%) | 124 (8%) | 232 (13%) | 40 (13%) | 7 (7%) | 31 (7%) |
| PKD/Hypertension | 313 (21%) | 109 (7%) | 266 (15%) | 79 (26%) | 8 (8%) | 79 (18%) |
| Unknown | 95 (6%) | 95 (6%) | 146 (8%) | 16 (5%) | 7 (7%) | 36 (8%) |
| Primary liver disease diagnosis (%) | Alcohol | 271 (18%) | 305 (19%) | 236 (13%) | 17 (6%) | 6 (6%) | 21 (5%) |
| Viral | 622 (41%) | 581 (37%) | 726 (41%) | 211 (69%) | 79 (83%) | 337 (75%) |
| Crypotogenic | 112 (7%) | 163 (10%) | 155 (9%) | 33 (2%) | 21 (1%) | 12 (1%) |
| Other | 424 (28%) | 353 (23%) | 499 (29%) | 13 (4%) | 8 (8%) | 28 (6%) |
| INR at transplant | 1.3 (1.1–1.6) | 1.8 (1.4–2.7) | 1.5 (1.2–1.9) | <0.001 | 1.3 (1.1–1.6) | 1.8 (1.4–2.7) | 1.5 (1.2–1.9) | <0.001 |
| Bilirubin at transplant | 8.7 (3.0–27.0) | 14.0 (7.3–37.0) | 1.0 (0.6–1.6) | <0.001 | 8.7 (3.0–27.0) | 14.0 (7.3–37.0) | 1.0 (0.6–1.6) | <0.001 |
| Primary heart disease diagnosis (%) | Ischemic cardiomyopathy | 17 (6%) | 6 (6%) | 21 (5%) |
| Dilated cardiomyopathy | 257 (85%) | 74 (78%) | 362 (81%) |
| Other | 30 (10%) | 15 (16%) | 66 (15%) | 110 (7%) | 387 (25%) | 134 (8%) |
| Nonkidney life support at transplant* | Alcohol | 271 (18%) | 305 (19%) | 236 (13%) | 17 (6%) | 6 (6%) | 21 (5%) |
| Viral | 622 (41%) | 581 (37%) | 726 (41%) | 211 (69%) | 79 (83%) | 337 (75%) |
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Covariates included in the multivariable analysis are underlined. Continuous variables are represented as median (25–75th percentile range). Categorical variables are represented as count (percentage). Data missingness is <1% unless stated otherwise.

*1–10% missing.

+11–20% missing.

+21–30% missing.

AKI, acute kidney injury; COPD, chronic obstructive pulmonary disease; cPRA, calculated panel reactive antibodies; HCV, hepatitis C virus; HLA, human leukocyte antigen; INR, international normalized ratio; KDRI2017, Kidney Donor Risk Index, normalized to the 2017 scale; NASH, nonalcoholic steatohepatitis; PKD, polycystic kidney disease; SHK, simultaneous heart-kidney; SLK, simultaneous liver-kidney.
none vs ≥90 d dialysis

|                  | SLK | SHK |
|------------------|-----|-----|
| 6-mo             |     |     |
| Apparent allograft failure | -1% | +1% |
| Death            | +4% | +3% |
| 12-mo            |     |     |
| Apparent allograft failure | -1% | +1% |
| Death            | +3% | +1% |
| 6-mo             |     |     |
| Apparent allograft failure | -3% | -3% |
| Death            | +4% | +5% |

Dialysis ≥90 d (presumed lowest residual kidney function) is the reference group. Results are represented as the difference in event risk. Bolded cells denote statistical significance (P < 0.05). SHK, simultaneous heart-kidney; SLK, simultaneous liver-kidney.

FIGURE 3. Adjusted event rate at 6- and 12-mo in SLK and SHK recipients, by presumed residual kidney function at time of transplant. Gray, death; black, apparent allograft failure; white, event-free survival. Dialysis ≥90 d (presumed lowest residual kidney function) is the reference group. P values refer to the test for difference between the likelihood of event-free survival compared with the reference group, in dialysis <90 d group (shorter line) and no dialysis group (longer line). SHK, simultaneous heart-kidney; SLK, simultaneous liver-kidney.

Our study does not answer the question of whether the extent of seemingly “prophylactic” kidney transplants is appropriate. Some degree of kidney impairment is almost ubiquitous in end-stage heart and liver disease. A functioning kidney allograft does more than avert dialysis in the immediate perioperative setting; it also carries out complex metabolic and immunomodulatory roles. In other words, the kidney allograft, in reducing the need for dialysis and kidney failure-associated complications, may also have “protected” the native kidneys postoperatively in SHK and SLK. From the individual SHK and SLK candidate’s perspective, having 3 functioning kidneys is thus preferable to incurring the risk of having none. Although these salutary effects are doubtlessly present clinically, the incremental benefit of the kidney in MOT is less well-established at a population level. For SLK, the best available study, a propensity-matched cohort study, reported a significantly lower mortality (roughly half) in the first-year posttransplant of SLK compared with liver-alone transplant, but this only translated to a 1–4 month gain in 5-year mean posttransplant survival owing to the overall high
mortality of SLK recipients. This gain in quite modest compared with the years gained for kidney-alone transplant as estimated by Wolfe et al. For SHK, the results are similar (as reviewed by Cheng et al). Furthermore, all studies comparing the mortality of SLK/SHK to liver-/heart-alone transplant recipients have been subject to confounding by indication: the difference in survival was the most marked in the first-day posttransplant, suggesting confounding by indication where some of the sickest patients received only liver-/heart-alone because they were too sick to undergo dual-organ transplant. Our study does not directly address many of the clinical challenges in the management of patients with dual-organ failure. Rather, our study sheds light on the population level and policy question of how many kidney transplants used in MOT should be offered is a supply-side question—the answer may depend on the availability of deceased donor kidney transplant recipients. The results are not yet known.

In summary, we find an association between the need for, and duration of, dialysis in advance of SLK and SHK transplant recipients and access for kidney-alone transplant candidates is not yet known. Such changes would reduce the likelihood of seemingly prophylactic transplants and shift kidneys toward patients who unambiguously need them. As a move in this direction, UNOS’s 2017 SLK policy set criteria for SLK (where none existed before) and implemented a Safety Net option; whether and how this policy change altered outcomes for liver transplant recipients and access for kidney-alone transplant candidates is not yet known.

As a nontransplant option to kidney failure exists in the form dialysis, a lifetime approach to management of heart and liver transplant candidates with kidney disease would be to reserve kidney transplants for when residual kidney function is truly exhausted, that is, when the patient becomes truly dialysis-dependent. Changes to the MOT allocation system to reflect this would include:

1) mandated reporting of measured native and transplant kidney GFR to the SRTR after MOT, as discussed previously, and incorporation of these data into assessments of the center’s posttransplant kidney allograft survival;

2) more stringent criteria for SLK/SHK;

3) a Safety Net option to enable liver/heart transplant recipients to obtain allocation priority for deceased donor kidneys.

Such changes would reduce the likelihood of seemingly prophylactic transplants and shift kidneys toward patients who unambiguously need them. As a move in this direction, UNOS’s 2017 SLK policy set criteria for SLK (where none existed before) and implemented a Safety Net option; whether and how this policy change altered outcomes for liver transplant recipients and access for kidney-alone transplant candidates is not yet known.

In summary, we find an association between the need for, and duration of, dialysis in advance of SLK and SHK transplantation, with a suggestion of seemingly “prophylactic” kidney transplantation in some SLK and SHK recipients. The estimated effect sizes are statistically significant but clinically modest and warrant verification in future studies with better quantifications of residual kidney function. Our findings challenge the validity of apparent kidney allograft failure as a metric of kidney transplant program quality in multiorgan transplantation. Whether the extent of seemingly prophylactic kidney transplantation is warranted in the age of critical organ shortage deserves vigorous debate and discourse in the ethics and policy arenas.

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