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Acute Kidney Injury in Deceased Organ Donors: Risk Factors And Impacts on Transplantation Outcomes

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Background. Acute kidney injury in deceased donors (D-AKI) is one of the common causes of donor kidney discard. The risk factors for D-AKI and its impact on kidney transplantation outcomes are not yet fully understood. **Methods.** This single-center, retrospective cohort study included 388 donors referred between June 2021 and December 2022. D-AKI was defined and staged according to kidney disease: Improving global outcomes criteria, and donor clinical variables were analyzed to identify risk factors for D-AKI. Delayed graft function and estimated glomerular filtration rate (eGFR) at 6 mo were evaluated in 369 kidney grafts transplanted from donors with and without D-AKI. **Results.** AKI was present in 171 deceased donors (44.1%), with 117 (30.2%) classified as AKI stage 1 and 54 (14%) as AKI stages 2 or 3. Donor history of hypertension (odds ratio [OR] 1.93; 95% confidence interval [CI], 1.21-3.10; $P = 0.005$), history of diabetes (OR 2.2; 95% CI, 1.21-3.98; $P = 0.008$), and anoxia as the cause of death (OR 2.61; 95% CI, 1.5-4.61; $P < 0.001$) were independently associated with an increased risk of D-AKI. Multivariable mixed models identified donor age (β –0.49; 95% CI, –0.71 to –0.28; $P < 0.001$) as the only independent risk factor for lower eGFR at 6 mo. D-AKI was not associated with delayed graft function or lower eGFR at 6 mo. **Conclusions.** Hypertension, diabetes, and anoxia as the cause of death were identified as risk factors for AKI in deceased donors. D-AKI should not be used as the sole criterion to assess the risk of poor graft outcomes. A broader range of donor variables should be considered when evaluating graft viability.

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The scarcity of available organs for transplantation necessitates changes in donor acceptance criteria.[1] Acute kidney injury in deceased donors (D-AKI) is often a reason for discarding donor kidneys, yet the evidence supporting this practice is not well established.[2,3]

In clinical practice, decisions about accepting deceased donor kidneys consider not only the presence and severity of AKI but also other factors. This approach can ultimately lead to selection bias, as kidneys from "healthier" donors are more likely to be accepted even if D-AKI is present. As a result, study outcomes are inconsistent; some studies report no impact of D-AKI or its severity on kidney allograft outcomes,[4] whereas other studies describe detrimental effects, including increased rates of delayed graft function (DGF) and primary nonfunction.[5,6]

Up to 30% of hospital-acquired AKI is preventable, and many cases are reversible if detected early.[7] Therefore, identifying risk factors of D-AKI could aid in modifying donor management practices and reducing the incidence of D-AKI. Despite extensive knowledge of risk factors, detection, prevention, and management of AKI in the general population,[8,9] the specific circumstances of deceased donors due to brain-death-associated changes[10] present unique challenges, and the available evidence remains limited.

In this study, we aimed to identify risk factors for the development of D-AKI and to evaluate its impact on transplant outcomes by assessing the rates of DGF and eGFR at 6 mo.

MATERIAL AND METHODS

Study Design and Cohort

This single-center, retrospective cohort study included deceased donors (both after brain and circulatory death, n = 462) referred to our center from June 11, 2021, to December 1, 2022. Donors younger than 18 y (n = 17) and donors with only 1 serum creatinine level available (n = 57) were excluded. This left 388 donors with complete clinical data. From these donors, 776 kidneys were potentially available for transplantation. Transplantation outcomes were not assessed for discarded allografts (n = 195; reasons for discard are detailed in **Table S1** (**SDC,** http://links.lww.com/TXD/A712) or for allografts transplanted at other transplant centers (n = 198). Consequently, we analyzed risk factors for DGF in 383 recipients and eGFR at 6 mo in 369 recipients, as 14 recipients (10 had graftectomy, 4 died) had shorter follow-ups (see Figure 1).

AKI Definition

To assess risk factors for D-AKI, we categorized donors into cases (with AKI) and controls (without AKI). D-AKI was defined using the kidney disease: Improving global outcomes (KDIGO) classification.[11] Baseline and peak creatinine levels

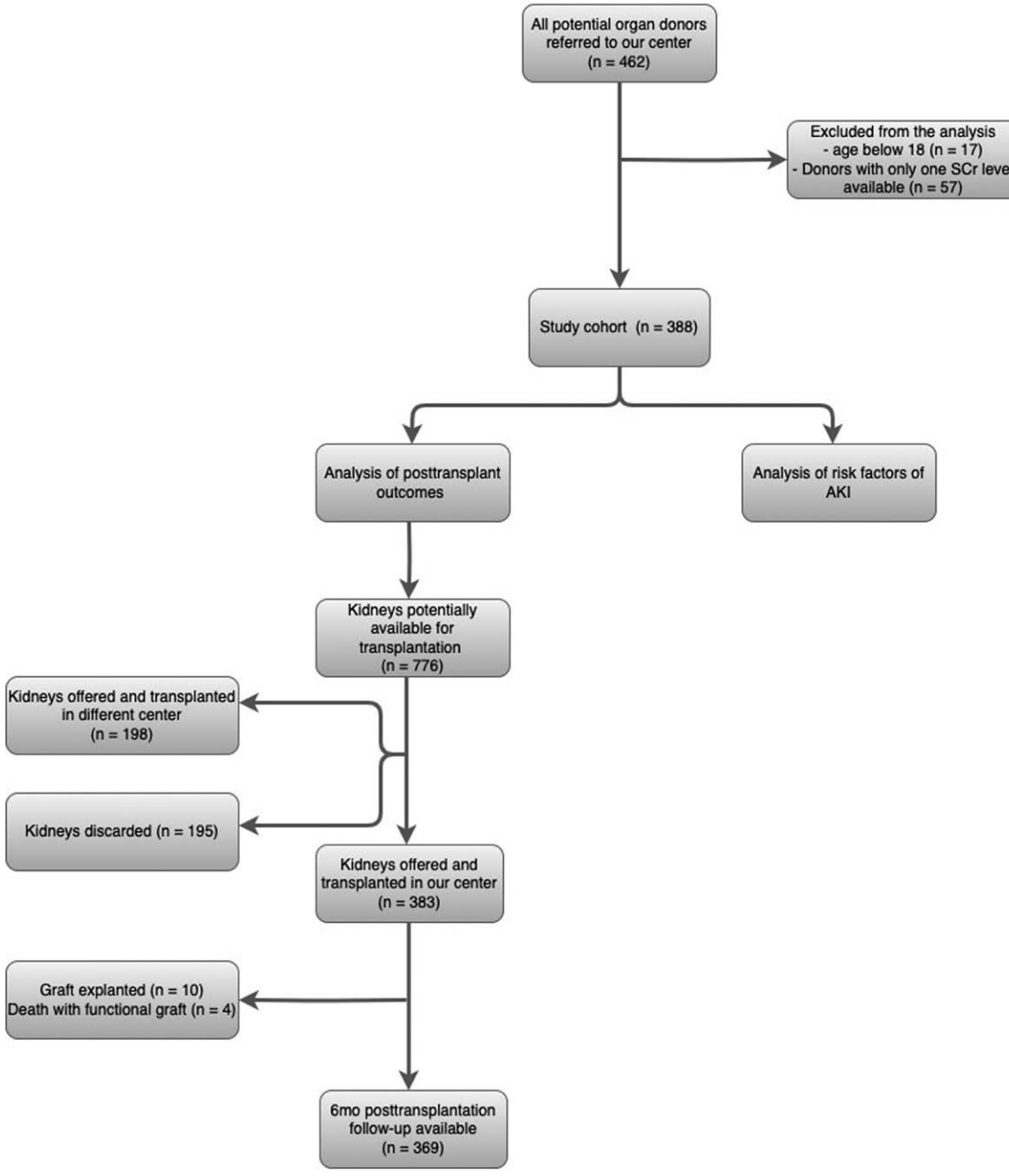

FIGURE 1. Study flowchart. Of the 476 potential donors referred to our center, 388 were included in the analysis. Clinical data were collected to assess risk factors of AKI in these donors. From the 776 kidneys potentially available for transplantation from these donors, we selected 383 kidneys that were transplanted at our center to assess posttransplant outcomes. Ten recipients had an early graftectomy and 4 recipients died within 6 mo of transplantation and thus were not included in the final analysis. The reasons for graftectomy are detailed in **Table S8** (**SDC,** http://links.lww.com/TXD/A712). Ultimately, 369 recipients with 6-mo follow-up were included in the analysis. AKI, acute kidney injury; SCr, serum creatinine.

were determined as the lowest/highest values recorded during the donor's hospital admission. The baseline creatinine level was either the lowest achieved during the hospital admission or the lowest value from the donor's health records up to 3 mo before admission. Donors with D-AKI were further classified into 2 categories based on the KDIGO stage: D-AKI stage 1 and D-AKI stage 2 + 3, the latter combined into a single group to reflect more severe pathophysiological changes.

Donor Data Collection

Donor clinical data including age, sex, body mass index, Kidney Donor Risk Index, history of hypertension, diabetes, cause of death, donor type (standard criteria donor, extended criteria donor or donor after circulatory death), number of admission days, and use of vasopressors were prospectively collected by the organ procurement team. The Kidney Donor Risk Index was calculated according to previously described methods.[12]

Posttransplantation Assessments

The impact of D-AKI on transplant outcomes was assessed by evaluating rates of DGF, primary nonfunction (PNF), and estimated glomerular filtration rate (eGFR, mL/min/1.73 m^2) at 6 mo.

DGF was defined as the need for hemodialysis within the first 7 d posttransplantation.[13] PNF was defined as dependence on dialysis from the time of transplantation up to 90 d. eGFR at 6 mo posttransplantation was calculated using the chronic kidney disease epidemiology collaboration formula.[14]

Statistics

Statistics were calculated using R, version 4.1.1. Continuous variables are reported as medians with interquartile ranges (IQRs), whereas categorical variables are reported as proportions. The Kruskal-Wallis test and the chi-square test were used to compare differences between groups. Several regression techniques were used: ordinal logistic regression was used to model risk factors for AKI development, with D-AKI levels categorized as separate ordinal categories; binary logistic regression mixed model was used to assess risk factors for DGF; and a linear mixed model was used to evaluate factors associated with eGFR at 6 mo posttransplantation. Mixed effects models accounted for the relatedness of paired kidneys (ie, kidneys transplanted to different recipients from the same donor). Variables were selected on the basis of domain knowledge. All statistical tests were conducted at the 5% level of significance.

Ethical Approval and Consent to Participate

The study was approved by the Institutional Review Boards (Ethics Committee of the Institute for Clinical and Experimental Medicine and Thomayer Faculty Hospital) under approval number No.13340/2020, in accordance with the Declaration of Helsinki. All patients were informed about the potential use of their personal data for research purposes.

RESULTS

Donor Demographics

We included 388 deceased donors, of whom 171 (44.1%) developed D-AKI. Among these, 68% (n = 117) of donors had KDIGO stage 1, 19% (n = 32) had KDIGO stage 2, and 13% (n = 22) had KDIGO stage 3. Donor characteristics are detailed in Table 1. Compared with the AKI-free group, donors with AKI had a higher body mass index (median 27.7 in AKI stage 1, 27.8 in AKI stages 2/3 versus 26.2 in the AKI-free group, $P = 0.028$). They also had higher rates of hypertension (35% in AKI stage 1, 48% in AKI stage 2/3 versus 29% in the AKI-free group, $P = 0.026$) and diabetes (20% in both AKI stages 1 and 2/3 versus 8.3% in the AKI-free group, $P = 0.004$). Additionally, anoxia was more common among donors with AKI compared with other causes of death (28% in AKI stage 1, 44% in AKI stages 2/3 versus 18% in the AKI-free group, $P = 0.006$).

TABLE 1.

Demographics of donors with and without AKI

Donor stage AKI	All donors (N = 388)	No AKI (N = 217)	AKI stage 1 (N = 117)	AKI stage 2/3 (N = 54)
Donor age, y, median (IQR)	52 (42–66)	53 (42–66)	51 (41–67)	53 (43–67)
Admission days, median (IQR)	4.0 (3.0–6.0)	4.0 (3.0–6.0)	3.0 (3.0–6.0)	4.0 (3.0–6.8)
KDRI index, median (IQR)	1.41 (1.08–2.04)	1.36 (1.08–1.91)	1.45 (1.03–2.15)	1.55 (1.22–2.15)
Male sex, n (%)	242 (62)	129 (59)	83 (71)	30 (56)
Body mass index, kg/m^2, median (IQR)	27.0 (24.2–31.2)	26.2 (23.4–30.1)	27.7 (24.7–32.1)	27.8 (24.8–31.9)
Donor type, n (%)				
Standard criteria donor, n (%)	187 (48)	104 (48)	56 (48)	26 (48)
Expanded criteria donor, n (%)	178 (46)	102 (47)	52 (44)	25 (46)
Donor after circulatory death, n (%)	19 (4.9)	10 (4.6)	9 (7.6)	1 (1.9)
Hypertension, n (%)	130 (34)	63 (29)	41 (35)	26 (48)
Diabetes, n (%)	52 (13)	18 (8.3)	23 (20)	11 (20)
Cause of death, n (%)				
- Anoxia	96 (15)	39 (18)	33 (28)	24 (44)
- Hemorrhagic stroke	155 (40)	92 (42)	43 (37)	20 (37)
- Ischemic stroke	35 (9.0)	22 (10)	10 (8.5)	3 (5.6)
- Other	15 (3.9)	12 (5.5)	1 (0.9)	2 (3.7)
- Traumatic brain injury	69 (18)	42 (19)	24 (21)	3 (5.6)
Use of vasopressors, n (%)	330 (93)	192 (95)	99 (91)	39 (93)

AKI, acute kidney injury; IQR, interquartile range; KDRI, Kidney Donor Risk Index.

Model for Risk Factors of D-AKI

Risk factors for D-AKI were assessed using an ordinal logistic regression model. The multivariable model (Table 2) identified the following independent risk factors for D-AKI: a history of hypertension (odds ratio [OR] 1.93; 95% confidence interval [CI], 1.21-3.10; $P = 0.005$), diabetes (OR 2.2; 95% CI, 1.21-3.98; $P = 0.008$), and anoxia as a cause of death (OR 2.61; 95% CI, 1.5-4.61; $P < 0.001$); the univariable associations are detailed in **Table S2** (**SDC,** http://links.lww.com/TXD/A712).

Posttransplant Outcomes

Recipient characteristics are detailed in Table 3. The impact of donor and recipient factors on eGFR at 6 mo was evaluated using a linear mixed model. The multivariable model identified donor age as the only independent risk factor for lower eGFR at 6 mo (β = –0.49; 95% CI, –0.71 to –0.28, $P < 0.001$). D-AKI was not associated with impaired eGFR at 6 mo (β = 4.92; 95% CI, –1.69 to –11.53, $P = 0.144$ for AKI stage 1, β = 2.45; 95% CI, –8.15 to –13.05; $P = 0.650$ for AKI stage 2 + 3). The multivariable model is presented in Table 4. Univariable associations for donor and recipient factors are shown in **Tables S3 and S4** (**SDC,** http://links.lww.com/TXD/A712).

Risk factors for DGF were assessed using a binary logistic mixed model. The multivariable model identified the following independent risk factors for DGF: a history of donor hypertension (OR 1.77; 95% CI, 1.03-3.04; $P = 0.038$), recipient male sex (OR 1.77; 95% CI, 1.03-3.04; $P = 0.038$), number of donor admission days (OR 1.08; 95% CI, 1.01-1.15; $P = 0.023$), and recipient history of diabetes (OR 1.99; 95% CI, 1.17-3.41; $P = 0.012$). Notably, a history of diabetes in donors was associated with a reduced risk of DGF (OR 0.25; 95% CI, 0.09-0.69; $P = 0.008$). The multivariable model is presented in Table 5, and univariable associations are detailed in **Tables S5 and S6** (**SDC,** http://links.lww.com/TXD/A712). Seven recipients experienced PNF; 4 of these recipients had transplantation from D-AKI, and 3 did not. The identified causes of PNF are listed in **Table S7** (**SDC,** http://links.lww.com/TXD/A712).

DISCUSSION

In this single-center observational cohort study, we found that donor's history of hypertension, diabetes, and anoxia as cause of death are risk factors for AKI in deceased donors

TABLE 2.

Multivariable ordinal logistic regression model for donor AKI development

Donor characteristics	OR	95% CI	*P*
Donor BMI <30 kg/m²	0.84	0.54-1.31	0.449
Hypertension	1.93	1.21-3.10	**0.005**
Diabetes	2.2	1.21-3.98	**0.008**
Donor age	0.98	0.97-1.00	0.08
Cause of death			
Stroke	1.07	0.64-1.82	0.77
Anoxia	2.61	1.5-4.61	**<0.001**

P values in bold indicate statistical significance ($P < 0.05$).
BMI, body mass index; CI, confidence interval; OR, odds ratio.

TABLE 3.

Demographics of recipients

Recipients (N = 369)	
Recipient age, median (IQR)	54 (45–65)
Male sex, n (%)	248 (67)
Body mass index, kg/m², median (IQR)	26.8 (23.6–29.8)
Retransplantation, n (%)	44 (12.4)
HLA mismatch, median (IQR)	3 (2–4)
DSA at transplantation, n (%)	38 (10)
Peak pretransplant PRA, median (IQR)	8 (3–18)
T cell–depletive induction, n (%)	200 (54)
Cause of recipient ESRD, n (%)	
Glomerulonephritis	142 (38.5)
Diabetes	73 (19.8)
Hypertension	43 (11.7)
Polycystic kidney disease	56 (15.2)
Tubulointerstitial nephritis	19 (5.1)
Other cause	36 (9.8)

DSA, donor-specific antibody; ESRD, end-stage renal disease; IQR, interquartile range; PRA, panel-reactive antibody.

TABLE 4.

Multivariable linear mixed model for estimated glomerular filtration rate at 6 mo after transplantation

Parameter	Coefficient	95% CI	*P*
Donor male sex	0.87	–5.26 to 6.99	0.781
Donor age, y	–0.49	–0.71 to –0.28	**<0.001**
Donor – hypertension	–1.45	–8.39 to 5.49	0.682
Donor – diabetes	2.85	–7.55 to 13.25	0.590
Recipient age, y	–0.02	–0.21 to 0.17	0.813
HLA mismatch	–0.79	–2.85 to 1.28	0.453
Recipient – DSA	–1.76	–10.03 to 6.52	0.676
Recipient male sex	1.36	–4.08 to 6.80	0.622
Recipient BMI <30 kg/m²	–0.68	–6.47 to 5.11	0.817
Donor AKI – stage 1	4.92	–1.69 to 11.53	0.144
Donor AKI – stage 2 + 3	2.45	–8.15 to 13.05	0.650
Cold ischemia time, h	–0.003	–0.01 to 0.00	0.355

P values in bold indicate statistical significance ($P < 0.05$).
AKI, acute kidney injury; BMI, body mass index; CI, confidence interval; DSA, donor-specific antibody.

TABLE 5.

Multivariable logistic regression model for DGF development after transplantation

Parameter	OR	95% CI	*P*
Donor male sex	0.90	0.54-1.49	0.677
Donor age	1.01	0.99-1.03	0.249
Donor – hypertension	1.77	1.03-3.04	**0.038**
Donor – diabetes	0.25	0.09-0.69	**0.008**
Donor AKI – stage 1	0.93	0.53-1.61	0.793
Donor AKI – stage 2 + 3	1.15	0.50-2.63	0.742
Recipient age	1.01	0.99-1.03	0.249
Recipient – diabetes	1.99	1.17-3.41	**0.012**
HLA mismatch	1.01	0.84-1.22	0.907
Recipient – DSA	1.30	0.59-2.84	0.514
Recipient male gender	1.77	1.03-3.04	**0.038**
Donor admission days	1.08	1.01-1.15	**0.023**
Cold ischemia time	1.00	1.00-1.00	0.055

P values in bold indicate statistical significance ($P < 0.05$).
AKI, acute kidney injury; CI, confidence interval; DSA, donor-specific antibody; OR, odds ratio.

(D-AKI). However, our findings indicate that donor AKI was not associated with DGF or impaired kidney graft function at 6 mo.

Anoxia as a cause of death was notably associated with a higher risk of D-AKI. This finding corroborates other studies that have reported an increased risk of AKI in survivors of cardiopulmonary resuscitation, given the association between cardiac arrest and ischemic kidney injury.[15,16]

Interestingly, in our study, donor age was not identified as a significant risk factor for D-AKI. Although tissue aging has been linked to an increased AKI risk, chronological age alone does not necessarily reflect biological aging.[17] Comorbidities, such as diabetes, which we found to be associated with D-AKI, may, however, accelerate biological aging.[18]

Our observation that donor age did not influence D-AKI risk factors could be partly attributed to selection bias. Younger donors with AKI tend to be more likely accepted for transplantation. To make up for this potential selection bias, we included all the referred donors, including those for whom kidneys were not accepted for transplantation. Despite this, selection bias cannot be entirely excluded, as older donors with AKI might not even be referred to transplant centers by donor hospitals based on the referral policies of hospitals.

Although some studies have reported D-AKI as a risk factor for posttransplant outcomes,[2] recent research suggests that D-AKI may not significantly affect transplant outcomes. Liu et al[4] found no impact of D-AKI on posttransplant outcomes in a cohort with a median age of 42 y. Similarly, our study with an older cohort (median age 52 y) found no adverse effects of D-AKI on transplant outcomes. This aligns with a recent study suggesting that AKI stages 1 and 2 in native kidneys may not lead to long-term eGFR decline.[19] It is likely that the long-held belief that AKI is associated with an eGFR decline and with the progression of chronic kidney disease will become increasingly challenged. A recently published meta-analysis also found no association between D-AKI and posttransplant outcomes. However, the authors recommend caution with donors exhibiting AKI stages 2 or 3 and higher kidney donor profile indices.[20] Thus, it appears that graft function is more influenced by chronic parenchymal changes related to comorbidities rather than by the presence of D-AKI itself.

Contrary to other studies, we did not find an association between donor AKI and DGF.[21] Recipients with diabetes were at higher risk of DGF, consistent with other reports.[22] Unexpectedly, donor diabetes was associated with a reduced risk of DGF in our study, possibly due to center-specific allocation policies. The definition of DGF, a binary assessment, is subject to clinical judgment, which may affect its reliability.

One limitation of our study is its retrospective and observational design. Additionally, the donor selection process is specific to our center and is inherently subject to selection biases at multiple levels, ranging from the initial hospital referral to the transplant center's decision to accept the organ. However, we sought to mitigate this bias by including all donors referred to our center in the analysis.

In conclusion, a history of hypertension, diabetes, and anoxia as a cause of death were identified as risk factors for AKI in deceased donors, consistent with patterns observed in the general population.[8,9] In our cohort, D-AKI did not significantly impact the development of DGF or eGFR at 6 mo posttransplant. This suggests that older donors with AKI may represent an underused donor resource. Consequently, overly stringent organ acceptance policies based solely on the presence of D-AKI may result in the unnecessary discard of kidney grafts that are otherwise of acceptable quality.

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