scientific reports

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Impact of donor's acute kidney injury on graft outcomes of deceased donor kidney transplantation

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Deceased donor kidney transplantation (DDKT) has a serious problem of donor organ shortage, particularly in Asia. Consequently, donor kidneys with acute kidney injury (AKI) have been utilized for transplantation. This study aimed to evaluate the prognosis of DDKT using AKI kidneys. We analyzed the data of 8,523 adult patients who underwent DDKT between 2008 and 2022 using the Korean nationwide DDKT database. The association between donor AKI and death-censored graft failure was assessed using competing risk analysis, with adjustment through inverse probability of treatment weighting (IPTW). The AKI group included 3,453 patients (40.51%) and had a higher death-censored graft failure risk than the no-AKI group (hazard ratio: 1.27; 95% confidence interval: 1.05–1.53). Especially, AKI stage 3 subgroup had a higher death-censored graft failure risk than the no-AKI group. Furthermore, the AKI group had a higher death-censored graft failure risk than the no-AKI group in the subgroup with a Korean-kidney donor profile index (K-KDPI) ≥ 70 or the stable- or increasing trend of serum creatinine. In conclusion, using AKI kidneys with a K-KDPI < 70% or a decreasing creatinine trend, or at stage 1 and 2, could help in combating organ shortage, especially in areas with a long waiting time.

Keywords Acute kidney injury, Deceased donor kidney, Kidney transplantation, Organ shortage

Although kidney transplantation (KT) is the treatment of choice for end-stage kidney disease (ESKD)[1,2], the supply of donor organs remains insufficient to meet the global demand[3]. Deceased donation rates in Asian countries are much lower than in Western countries. For example, the donation rates in Iran and Korea were 11.50 and 8.56 per million population (pmp), whereas those of USA and Spain were 41.60 and 40.80 (pmp) in 2021[4]. However, both ESKD incidence and the its yearly change were higher in Asian countries than in Western countries; for example, the top two increasing rates from 2010 to 2020 were 19.7 and 18.8 pmp/year in Thailand and Korea, respectively[5]. Therefore, donor organ shortage is a more serious problem in Asian countries and maximal utilization of deceased donors' kidneys is very important issues to improve this problem[6,7].

One of the efforts employed is the active utilization and efficient allocation of expanded criteria donor (ECD) kidneys to minimize disposal of donors' kidneys[8–10]. The USA developed Kidney Donor Profile Index (KDPI) beyond dichotomous ECD criteria to define the prognostic status of a donor's kidneys in more detail[11,12]. Furthermore, suitable recipient candidates whose deceased donor kidney transplantation (DDKT) using ECD kidneys had survival benefits over waiting for DDKT using standard criteria donor (SCD) were proposed to efficiently utilize the ECD kidneys[13,14].

Another effort to minimize donor kidney disposal is to use kidneys that have acute kidney injury (AKI) before procurement. US studies found that donor AKI did not decrease graft survival[15,16]. Contrary to

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the above, a UK study reported that the donor's AKI increased 1-year graft failure and mortality[17], and a French study reported donor's AKI was associated with decreased graft survival[18]. These conflicting results suggest that the outcomes of DDKT using AKI kidneys would be varied according to an individual country's characteristics. Therefore, there is a need to assess the outcomes of DDKT using AKI kidneys based on each country's own data. This Korean nationwide study aimed to investigate the impact of donor AKI based on its subgroups, such as subgroups based on AKI stages, K-KDPI categories, and trends of creatinine change, on allograft outcomes to propose criteria for adequate use of donors' kidneys with AKI to ensure the maximal utilization and good outcomes of DDKT.

Materials and methods

Study population

We used a nationwide cohort of solitary adult DDKT patients from the Korean Network for Organ Sharing (KONOS) and the National Health Insurance Data Sharing Service (NHISS) database. A total number of 11,113 patients received DDKT between 2008 and 2022. Recipients under 19 years of age (n = 351), and those with missing or invalid information regarding height, serum creatinine, diabetes mellitus, dialysis duration (n = 2,239), were excluded from this study. Eventually, 8,523 DDKT patients were used in the final analysis (Fig. 1). This study was approved by the Institutional Review Board of Yonsei University Severance Hospital (4–2021-1358), and the review board waived the requirement for informed consent. This study was performed in accordance with the Helsinki Declaration and the Declaration of Istanbul of 2008[19].

Definitions and classification of AKI according to severity, K-KDPI, and changing trends in serum creatinine levels

Donor AKI was defined according to AKI Network (AKIN) criteria and classified as follows; stage 1, increase in serum creatinine from admission to the terminal by ≥ 0.3 mg/dL or 1.5 to < 2-fold; stage 2, 2 to < 3-fold; and stage 3, ≥ 3-fold, or terminal serum creatinine ≥ 4.0 mg/dL after an increase of ≥ 0.5 mg/dL from admission or dialysis support[20]. Further, donors were categorized into low and high K-KDPI groups using two different cutoffs: the Korean threshold of 70% as the primary threshold and the US threshold of 85% for sensitivity analysis[15,21]. A previous Korean study defined ECDs as those with a K-KDPI ≥ 70%, as this cutoff demonstrated good diagnostic performance and a significant survival difference compared with K-KDPI < 70%[21].

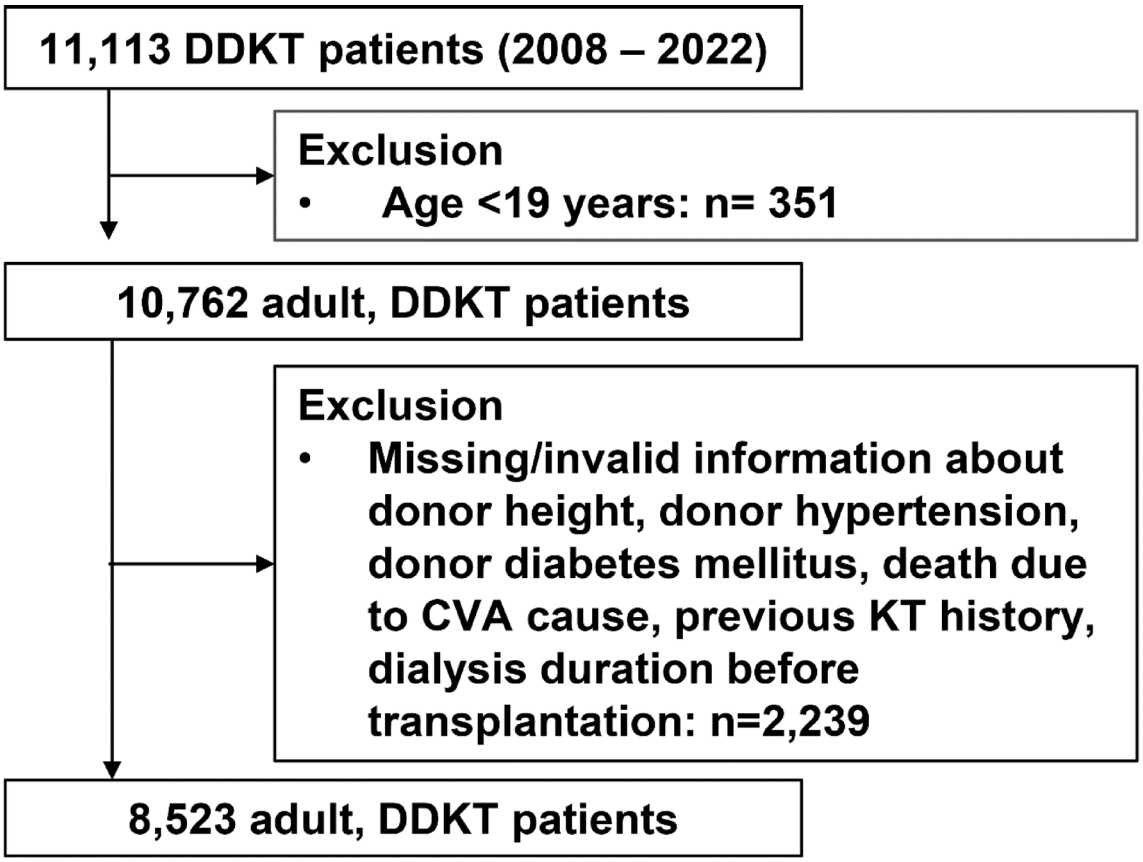

Fig. 1. Flow chart for study population. *CVA* cerebrovascular accident, *DDKT* deceased donor kidney transplantation, *KT* kidney transplantation.

Serum creatinine concentrations were measured at least three times before procurement, and creatinine levels at admission was considered as the baseline levels[16,17,22]. By changes from the peak creatinine concentrations to final creatinine concentrations just before procurement, changing trends in serum creatinine were defined as follows; stable or increasing trend (change > − 0.3 mg/dl), and decreasing trend (change ≤ −0.3 mg/dl)[23,24]. Estimated glomerular filtration rate (eGFR) was calculated using chronic kidney disease-epidemiology collaboration equations[25].

Outcomes

The primary outcome was death-censored graft failure, defined as a reinitiating dialysis, or going through re-transplantation. Death-uncensored graft failure and all-cause mortality were used as secondary outcomes in sensitivity analysis.

Statistical analysis

The association between donor AKI and death-censored graft failure was assessed using competing risk analysis. The primary analysis was conducted using cause-specific Cox proportional hazards models with adjustment for donor-, recipient-, and transplantation-related covariates in Model 1. In Model 2, the center effect was incorporated into the multivariable analysis by using a frailty function and donor-related covariates (weight, sex, death due to cerebrovascular accident [CVA], hypertension, and donation after circulatory death [DCD]) were further adjusted using stabilized and trimmed inverse probability of treatment weighting (IPTW). The results were expressed as a hazard ratio (HR) with a 95% confidence interval (CI). Statistical significance was set at $P < 0.050$. The adequacy of IPTW adjustment was confirmed by assessing the absolute mean difference (Fig. S1).

Sensitivity analyses were conducted using Fine and Gray subdistribution hazard models and multiple imputations (Table S3 and S4). Multiple imputations using chained equations were performed to address missing variables, which were used as covariates in the main analysis with missingness < 20% (donor hypertension, donor diabetes mellitus, death due to CVA, previous KT history, and dialysis duration before KT). Predictive mean matching with k-nearest neighbors (k = 5) was applied, and 10 independent imputed datasets were generated. Multivariate cause-specific hazard models were then fitted across the imputed datasets, and estimates were pooled using Rubin's rules.

All analyses were conducted using SAS (version 7.1, SAS Institute Inc., Cary, NC, USA), R (version 4.4.2, R Core Team, 2022, R Foundation for Statistical Computing, Vienna, Austria; URL: https://www.R-project.org/), and its packages (version 2024.12.0, RStudio Team, PBC, Boston, MA, USA; URL: http://www.rstudio.com). More detailed description of statistical methods was described in the supplementary methods.

Results

Comparison of clinical characteristics between AKI and no-AKI groups

Table 1 compares the clinical characteristics of donors and recipients between AKI and no-AKI groups. Among 8,523 DDKT patients, the no-AKI and the AKI groups included 5,070 (59.49%) and 3,453 (40.51%), respectively. Compared with donors without AKI, donors with AKI were older and had more males, diabetes mellitus, and hypertension. Donors in the AKI group had lower eGFR (41.75 ± 24.67 vs. 100.11 ± 31.52 ml/min/1.73 m^2, $P < 0.001$) and higher K-KDPI (69.21 ± 32.90 vs. 53.37 ± 36.65, $P < 0.001$) compared to those in the no-AKI group. Serum creatinine levels at admission were lower in the AKI group compared to the no-AKI group. Recipients in the AKI group were older and had a lower proportion of previous KT history.

Table S1 shows the comparison of the clinical characteristics among different AKI stages (AKI stage 1, $n = 1,472$, 42.63%; AKI stage 2, $n = 733$, 21.23%; and AKI stage 3, $n = 1,248$, 36.14%). With respect to the donor factors, there were differences in age, serum creatinine, BMI, and K-KDPI, as well as in the prevalence of diabetes mellitus, hypertension, use of continuous renal replacement therapy (CRRT), proportion of male donors, DCD, and CVA. Likewise, concerning the recipient factors, there were differences in age and history of KT. The number of HLA mismatches was higher in the AKI stage 3 group compared to stages 1 and 2.

Impact of AKI and AKI subgroups on graft outcomes

A total of 471 death-censored graft failure occurred during a median follow-up of 6.09 years. Furthermore, when AKI was subgrouped into stage 1 ($n = 1,472$), stage 2 ($n = 733$), and stage 3 ($n = 1,248$), death-censored graft failure occurred in 87, 44, and 69, in stages 1, 2, and 3 groups, respectively. The risk of death-censored graft failure remained higher in the AKI group compared to the no-AKI group in the multivariable analysis (Model 1, HR, 1.28; 95% CI, 1.06–1.54, $P = 0.010$). In the IPTW-adjusted multivariable model with incorporation of center effects, the AKI group showed a significantly higher risk of death-censored graft failure (Model 2, HR, 1.27; 95% CI, 1.05–1.53, $P = 0.015$) (Table 2; Fig. 2A). The variance of the random effect for center was 0.137, indicating minimal variation across transplant centers. In subgroup analysis, stage 3 groups had a higher risk for graft failure compared to the no-AKI group after adjustment for covariates, including center effects and IPTW (AKI stage 3, HR: 1.37, 95% CI: 1.04–1.81, $P = 0.023$) (Table 2; Fig. 2B).

Variables	No-AKI ($n=5,070$)	AKI ($n=3,453$)	P value
Donor-related factors			
Age, year	45.25 ± 15.62	47.77 ± 13.61	< 0.001
sCr at admission, mg/dL	1.06 ± 0.47	1.05 ± 0.62	0.138
sCr just before procurement, mg/dL	0.87 ± 0.41	2.41 ± 1.38	< 0.001
eGFR just before procurement	100.11 ± 31.52	41.75 ± 24.67	< 0.001
Diabetes mellitus	381 (7.51%)	390 (11.29%)	< 0.001
Gender, male	3,217 (63.45%)	2,477 (71.73%)	< 0.001
Hepatitis C virus	14 (0.27%)	14 (0.42%)	0.305
Hepatitis B virus	158 (3.12%)	106 (3.07%)	0.903
K-KDPI	53.37 ± 36.65	69.21 ± 32.90	< 0.001
K-KDPI ≥ 70, n	2,188 (43.16%)	2,151 (62.29%)	< 0.001
K-KDPI ≥ 85, n	1,522 (30.02%)	1,669 (48.33%)	< 0.001
US-KDPI	52.13 ± 26.02	69.09 ± 21.03	< 0.001
DCD	12 (0.24%)	14 (0.41%)	0.165
Cause of death, CVA	2,039 (40.22%)	1,622 (46.97%)	< 0.001
Donor AKI stage			< 0.001
No AKI	5,070 (100.00%)	NA	
Stage 1 AKI	NA	1,472 (42.63%)	
Stage 2 AKI	NA	733 (21.23%)	
Stage 3 AKI	NA	1,248 (36.14%)	
CRRT	0 (0.00%)	452 (13.09%)	< 0.001
Recipient-related factors			
Age, year	49.49 ± 10.81	51.52 ± 10.70	< 0.001
Gender, male	3,007 (59.31%)	2,135 (61.83%)	0.019
Dialysis duration, year	6.56 ± 3.95	6.62 ± 3.90	0.475
Diabetes mellitus	4,330 (85.40%)	2,985 (86.45%)	0.175
Hepatitis C virus	88 (1.74%)	61 (1.77%)	0.915
Hepatitis B virus	338 (6.67%)	223 (6.46%)	0.991
History of KT	485 (9.57%)	283 (8.20%)	0.030
Ischemic heart disease	3,483 (68.70%)	2,460 (71.24%)	0.012
Cerebrovascular accident	1,853 (36.55%)	1,280 (37.07%)	0.624
Cancer	1,633 (32.21%)	1,071 (31.02%)	0.245
Transplantation-related factors			
Numbers of HLA mismatch	3.69 ± 1.60	3.78 ± 1.54	0.007
CIT, min	269.00 ± 179.30	270.26 ± 150.33	0.786
Positivity of PRA	1,059 (20.89%)	785 (22.73%)	< 0.001
Outcomes			
Death	553 (10.91%)	421 (12.19%)	0.067
Death-censored graft failure	271 (5.35%)	200 (5.79%)	0.108

Table 1. Clinical characteristics of kidney transplant patients according to presence of donor AKI. *AKI* acute kidney injury, *CIT* cold ischemic time, *DCD* donation after circulatory death, *HLA* human leukocyte antigen, *K-KDPI* Korean-kidney donor profile index, *N* number, *NA* not applicable, *sCr* serum creatinine, *US-KDPI* United States-kidney donor profile index.

As sensitivity analysis, Fine and gray competing risk analysis demonstrated that the AKI group showed a higher risk of death-censored graft failure (SHR, 1.20; 95% CI, 1.00–1.44.00.44, $P=0.047$) after adjustment for covariates (Table S2, Fig. S2). Comparison between the analysis set and multiple imputation set revealed no significant differences except in donor age, donor and recipient BMI, dialysis duration, history of KT, and follow up duration (Table S3). As another sensitivity analysis, cause-specific Cox analysis after multiple imputation also showed that the risk of graft failure remained higher in the AKI group in the model 1 (HR, 1.21; 95% CI, 1.02–1.43, $P=0.026$), and in the model 2 (HR, 1.15; 95% CI, 1.15–1.37, $P=0.035$, Table S4).

Impact of AKI on graft outcomes according to K-KDPI groups

Donor kidneys were categorized into low and high K-KDPI groups using two different cutoffs: a threshold of 70% (low < 70%, high ≥ 70%) as the primary threshold and a threshold of 85% (low < 85%, high ≥ 85%) for sensitivity analysis. The incidence of death-censored graft failure was higher in the AKI group than in the no-AKI group, but only among recipients with high K-KDPI groups ≥ 70% (Table 3) and ≥ 85%

Group (event number)	Event per 1000 patient-years (95% CI)	Unadjusted			Model 1			Model 2			
		HR	95% CI	*P* value	HR	95% CI	*P* value	HR	95% CI	*P* value	V
No-AKI (*n* = 271)	196.805 (174.068–221.685)	Ref			Ref			Ref			
AKI (*n* = 200)	191.938 (166.258–220.462)	1.18	0.98–1.42	0.072	1.28	1.06–1.54	0.010	1.27	1.05–1.53	0.015	0.137
No-AKI (*n* = 271)	196.805 (174.068–221.685)	Ref			Ref			Ref			
AKI 1 (*n* = 87)	191.208 (153.150–235.855)	1.12	0.88–1.42	0.361	1.18	0.93–1.51	0.174	1.20	0.94–1.54	0.140	0.136
AKI 2 (*n* = 44)	207.547 (150.804–278.622)	1.20	0.87–1.65	0.259	1.28	0.93–1.77	0.134	1.25	0.90–1.73	0.190	0.136
AKI 3 (*n* = 69)	184.000 (143.163–232.863)	1.26	0.97–1.64	0.086	1.42	1.08–1.87	0.010	1.37	1.04–1.81	0.023	0.136

Table 2. Impact of AKI and AKI stage on death-censored graft failure. Model 1 was adjusted for various donor-related (age, sex, BMI, diabetes mellitus, hypertension, DCD, death due to CVA cause, Hepatitis C virus), recipient-related (age, sex, diabetes mellitus, previous kidney transplantation, dialysis duration, ischemic heart diseases, CVA, cancer), transplantation-related (HLA mismatch number). Model 2 was adjusted for center effects in addition to model 1 and was further adjusted using IPTW for donor-related (sex, weight, hypertension, DCD, death due to CVA cause) covariates. Numbers in parentheses indicate the number of events in each group. *AKI* acute kidney injury, *CI* confidence interval, *HR* hazard ratio, *n* number, *Ref* reference, *CVA* cerebrovascular accidents, *DCD* donation after circulatory death, *V* Variance of random effect for center.

(Table S5). In the multivariable model 1, AKI kidneys did not have a higher risk of death-censored graft failure compared to no-AKI kidneys in the low K-KDPI groups < 70% (Table 3; Fig. 3A) and < 85% (Table S5, Fig. S3A), whereas AKI kidneys had a higher risk compared to no-AKI kidneys in the high K-KDPI groups ≥ 70% (HR: 1.33, 95% CI: 1.04–1.71, $P = 0.023$, Table 3; Fig. 3B) and ≥ 85% (HR: 1.49, 95% CI: 1.11–1.98, $P = 0.007$, Table S5, Fig. S3B). In addition, when AKI subgroups according to AKI stage were compared with no-AKI, AKI stage 3 was associated with a higher risk of death-censored graft failure in the high K-KDPI group ≥ 70% (AKI stage 3, HR: 1.47, 95% CI: 1.04–2.08, $P = 0.027$, Table 3) and AKI stage 1 and 3 was associated with a higher risk of death-censored graft failure in the high K-KDPI group ≥ 85% (AKI stage 1, HR: 1.45, 95% CI: 1.01–2.08, $P = 0.040$; AKI stage 3, HR: 1.74, 95% CI: 1.17–2.57, $P = 0.005$, Table S5).

As sensitivity analysis, Fine and gray competing risk analysis demonstrated that AKI kidneys were associated with a higher risk of death-censored graft failure in the high K-KDPI group ≥ 85%, whereas AKI kidneys did not have a higher risk compared to no-AKI kidneys in the low K-KDPI group < 85% (Table S2). As another sensitivity analysis, cause-specific Cox analysis after multiple imputation also showed that the risk of death-censored graft failure in the AKI group remained higher than that in the no-AKI group in the high K-KDPI group ≥ 85% (model 1 and 2, Table S4).

Impact of AKI on graft outcomes according to changing trends of serum creatinine

In the multivariable model 1, AKI did not have a higher risk for death-censored graft failure than no-AKI in the decreasing-creatinine subgroup (HR: 1.14, 95% CI: 0.82–1.59, $P = 0.443$, Table 4; Fig. 4A). When each AKI stage was compared with no-AKI, no-AKI stage was associated with a higher risk (AKI 1, HR: 1.30, 95% CI: 0.82–2.07, $P = 0.260$; AKI 2, HR: 1.22, 95% CI: 0.69–2.16 $P = 0.489$; AKI 3, HR: 0.94, 95% CI: 0.57–1.56, $P = 0.826$, Table 4). In the stable- or increasing-creatinine subgroup, AKI showed a higher risk for graft failure than no-AKI (HR: 1.38, 95% CI: 1.09–1.73, $P = 0.006$, Table 4; Fig. 4B) and stage 3 AKI showed a higher risk (HR: 1.77, 95% CI: 1.27–2.45, $P < 0.001$, Table 4). These trends were similar in the model 2 (Table 4).

As sensitivity analysis, Fine and gray competing risk analysis also demonstrated that AKI showed a higher risk for death-censored graft failure than no-AKI in the stable- or increasing-creatinine subgroup (Table S2). In contrast, AKI was not associated with a higher risk for graft failure (Table S2). Cause-specific Cox analysis in the multiple imputation dataset also showed that the risk of death-censored graft failure in the AKI group remained higher than that in the no-AKI group only in the stable- or increasing-creatinine subgroup (Table S4).

Impact of AKI on secondary outcomes

Stage 3 AKI group showed a higher risk for death-uncensored graft failure (model 1, HR: 1.19, 95% CI: 1.02–1.38, $P = 0.025$, Table S6, Fig. S4) in parallel with results for death-censored graft failure, although total AKI group showed only a trend of higher risk than no-AKI group (model 1, HR: 1.10, 95% CI: 0.99–1.22, $P = 0.079$, Table S6). However, neither model 1 nor model 2 showed a significant difference in mortality between the AKI and no-AKI groups (Table S7, Fig. S5).

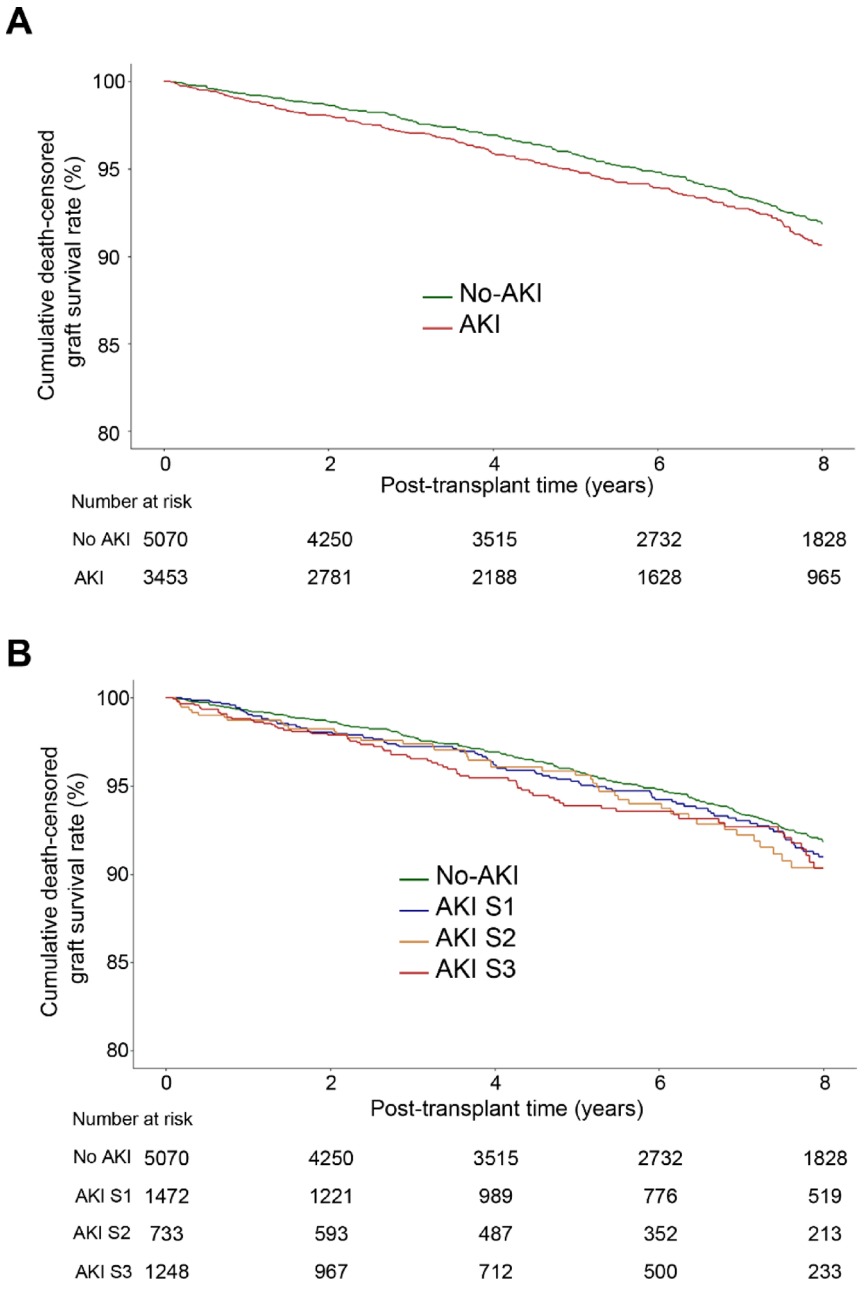

Fig. 2. Death-censored graft failure rates according to AKI and AKI stages. **A** Comparison of death-censored graft failure rates between AKI group and no-AKI group (Log rank test, $P = 0.072$). **B** Comparison of death-censored graft failure rates among AKI stage 1–3 and no-AKI group (Log rank test, $P = 0.086$ for AKI S3 vs. no-AKI). *AKI* acute kidney injury, *S1* stage 1, *S2* stage 2, *S3* stage 3.

Discussion

We hypothesized that DDKT using kidneys with favorable prognostic indicators would have comparable outcomes to DDKT using donor kidneys without AKI. This Korean nationwide study demonstrated that donor AKI was associated with a higher risk of death-censored graft failure; however, AKI kidneys at stage 1 or 2 and those with K-KDPI < 70% or a decreasing creatinine trend, had comparable graft outcomes to donor kidneys without AKI.

Considering the aggravating severe organ shortage in Asian countries, maximal utilization of limited deceased donor kidneys, including kidneys with AKI, is an important issue[26]. A previous Korean study reported that the discard rate of donors' kidneys was approximately 0.68%, which was much lower when compared to that of the USA (17.28%) and Eurotransplant group (12.00%)[27–30]. Generally, donor kidneys with AKI have been used in more than half of DDKT cases (50.55%), as shown in this study, because

K-KDPI (event number)	Event per 1000 patient-years (95% CI)	Unadjusted			Model 1			Model 2			
		HR	95% CI	*P* value	HR	95% CI	*P* value	HR	95% CI	*P* value	V
K-KDPI < 70											
No-AKI (*n* = 144)	230.031 (193.995–270.820)	Ref			Ref			Ref			
AKI (*n* = 59)	193.442 (147.257–249.526)	0.97	0.72–1.32	0.351	1.24	0.91–1.69	0.174	1.18	0.86–1.61	0.310	0.163
AKI 1 (*n* = 27)	200.000 (131.801–290.989)	0.85	0.56–1.28	0.937	1.04	0.69–1.57	0.858	1.03	0.68–1.57	0.890	0.158
AKI 2 (*n* = 14)	259.259 (141.739–434.993)	1.15	0.66–1.99	0.128	1.59	0.91–2.79	0.105	1.38	0.78–2.43	0.260	0.158
AKI 3 (*n* = 18)	155.172 (91.965–245.239)	1.09	0.67–1.78	0.325	1.43	0.86–2.35	0.164	1.32	0.80–2.20	0.280	0.158
K-KDPI ≥ 70											
No-AKI (*n* = 127)	169.107 (140.977–201.206)	Ref			Ref			Ref			
AKI (*n* = 141)	191.316 (161.041–225.628)	1.19	0.94–1.52	0.150	1.33	1.04–1.71	0.023	1.37	1.06–1.76	0.015	0.087
AKI 1 (*n* = 60)	187.500 (143.082–241.349)	1.22	0.90–1.66	0.198	1.30	0.95–1.78	0.096	1.34	0.98–1.84	0.065	0.085
AKI 2 (*n* = 30)	189.873 (128.106–271.056)	1.12	0.75–1.66	0.585	1.22	0.82–1.83	0.324	1.27	0.84–1.91	0.260	0.085
AKI 3 (*n* = 51)	196.911 (146.613–258.901)	1.20	0.87–1.67	0.262	1.47	1.04–2.08	0.027	1.48	1.05–2.09	0.027	0.085

Table 3. Impact of AKI and AKI stage on death-censored graft failure according to K-KDPI. Model 1 was adjusted for various donor-related (age, sex, BMI, diabetes mellitus, hypertension, DCD, death due to CVA cause, Hepatitis C virus), recipient-related (age, sex, diabetes mellitus, previous kidney transplantation, dialysis duration, ischemic heart diseases, CVA, cancer), transplantation-related (HLA mismatch number). Model 2 was adjusted for center effects in addition to model 1 and was further adjusted using IPTW for donor-related (sex, weight, hypertension, DCD, death due to CVA cause) covariates. Numbers in parentheses indicate the number of events in each group. *AKI* acute kidney injury, *CI* confidence interval, *HR* hazard ratio, *n* number, *ref* reference, *CVA* cerebrovascular accidents, *DCD* donation after circulatory death, *V* Variance of random effect for center.

of severe organ shortage and long waiting time for DDKT (> 10 years) in Korea. However, we need to determine the outcomes of patients who underwent DDKT using donor kidneys with AKI and define their acceptance criteria for active and safe utilization.

A US single-center study (*n* = 994) reported that the impact of AKI in donors' kidneys did not decrease graft survival, irrespective of AKI stage[15,20]. A US multicenter study (*n* = 585) with a median follow-up of 4.0 years also reported that the donor's AKI did not increase the risk for graft failure irrespective of AKI stage, thereby suggesting the active utilization of AKI kidneys[16,20]. However, larger European studies reported that donor AKI could significantly impact the graft survival of DDKT. For instance, a French CRISTAL registry study with a median follow-up of 5.7 years, reported that donor kidneys with AKI based on the Kidney disease: improving global outcomes (KDIGO) criteria (ongoing AKI, *n* = 3,373; recovery from AKI, *n* = 2,392; undetermined AKI/Chronic kidney disease [CKD], *n* = 1,745) were associated with decreased graft survival[18,31]. Moreover, three types of AKI were all associated with decreased graft survival (ongoing AKI, HR: 1.23; recovery from AKI, HR: 1.10; undetermined AKI/CKD, HR: 1.28)[18]. Likewise, a UK transplant registry study evaluated the short-term impact of AKI according to AKIN criteria as ours on DDKT[17]. Graft failure in 1 year was higher in all AKI stages (*n* = 1,869, HR: 1.20) compared to no-AKI. Despite the small degree of inferior graft survival in the AKI group, the authors suggested that the 20% increase in 1-year graft failure in the AKI group may be better than a 37% increase in graft failure in DDKT after dialysis for longer than 1 year while waiting for a donor's kidney without AKI[17]. They recommended the utilization of donors' kidneys with AKI stages 1 and 2 but advised that those with AKI stage 3 be used with caution[17]. Therefore, we consider that these conflicting results about the outcomes of DDKT from AKI donors might stem from differences in either cohort size and accompanying statistical power or cohort characteristics, such as the proportion of ethnicity and practice pattern of organ discard. The proportion of black donors and discard rate were 1% and 28%, respectively, in the UK cohort, whereas they were 24% and 21%, respectively, in the US cohort[16,17].

This Korean nationwide study, which included a relatively large number of patients with AKI (*n* = 3,453) and a median follow-up of 6.38 years, demonstrated the impact of AKI severity, as defined by the AKIN stage, on graft failure. AKI, especially stage 3 AKI was associated with a higher risk of graft failure compared to the no-AKI group in parallel with the European studies.

A previous Korean multicenter study (*n* = 386) used the old US ECD criteria and found that AKI in the ECD group was associated with a higher risk of graft failure, whereas AKI in the SCD group was not[32–34]. We also analyzed subgroup analysis according to the K-KDPI score. We found that in K-KDPI < 70% or 85%, the AKI group was not associated with a higher risk of graft failure compared to the no-AKI group. However, the AKI group was associated with a higher risk of graft failure in K-KDPI ≥ 70% subgroup, especially in K-KDPI ≥ 85% subgroup. When AKI stages were analyzed along with K-KDPI, there was no significant difference in the risk of graft failure according to AKI stages in the K-KDPI < 70% or 85% subgroup. Therefore, we could use AKI donors with stage 3 only in case of low K-KDPI subgroup.

The impact of AKI on graft outcomes according to changing patterns of serum creatinine concentration was

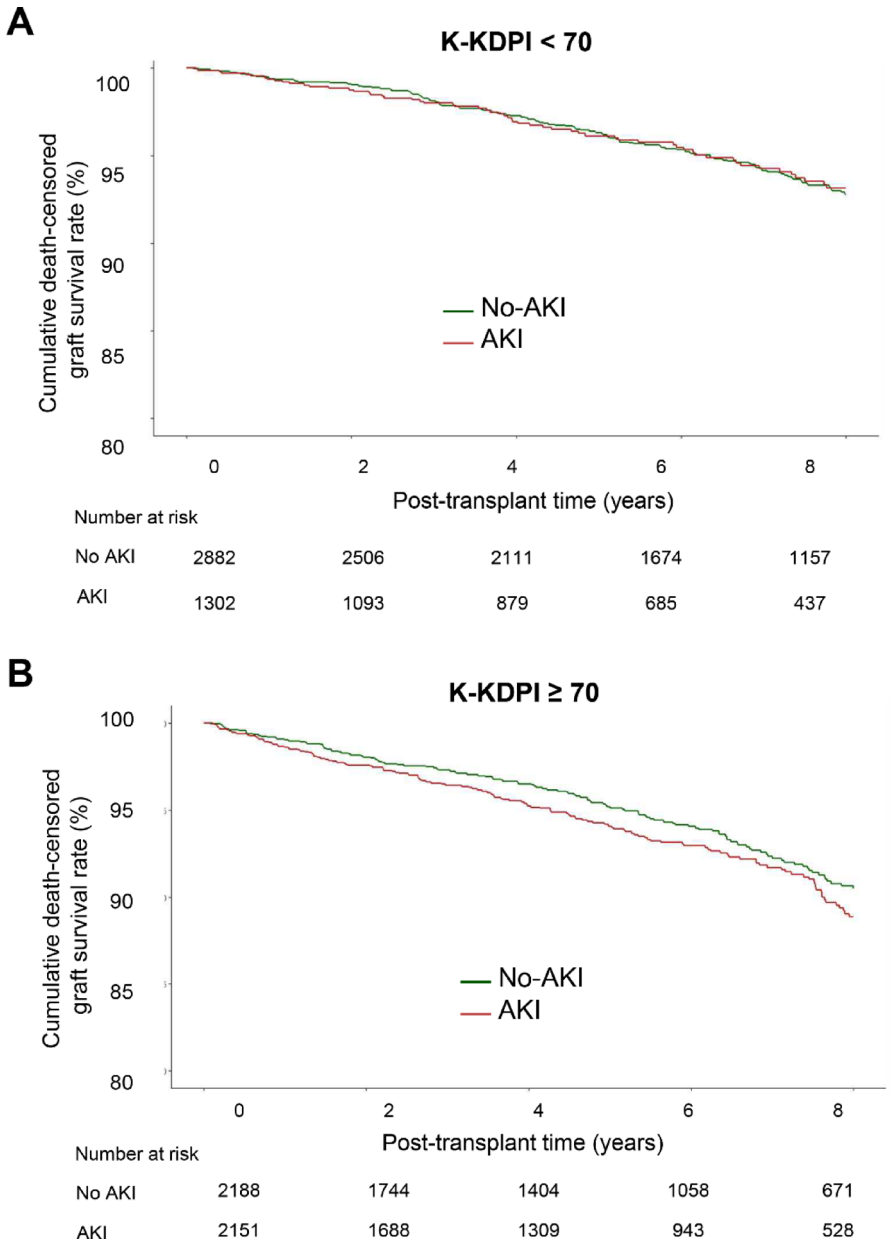

Fig. 3. Death-censored graft failure rates according to AKI according to K-KDPI subgroups in Model A. **A** Comparison of death-censored graft failure rates between AKI group and no-AKI group in the low K-KDPI group (< 70, Log rank test, $P = 0.351$). **B** Comparison of death-censored graft failure rates between AKI group and no-AKI group in the high K-KDPI group (≥ 70, Log rank test, $P = 0.150$). *AKI* acute kidney injury, *K-KDPI* Korean-kidney donor profile index.

also analyzed. AKI or any AKI stage was not associated with a higher risk of graft failure in the decreasing-trend group, which may be referred to as the recovering status. In contrast, AKI was associated with a higher risk of graft failure by 36% in the stable- or increasing-creatinine group. Neither stage 1 nor stage 2 was associated with higher risk, although stage 3 were associated with a higher risk of graft failure. These findings suggest that donor kidneys with AKI can be used when serial serum creatinine levels exhibit a decreasing trend, as such kidneys demonstrate a graft failure rate comparable to that of no-AKI kidneys and donors' kidneys with stage 1 and stage 2 AKI can be used in stable- or increasing-creatinine trend as well.

The present study has several limitations. First, some detailed clinical information, including immunosuppressive agents was missing in this registry-based nationwide study; therefore, we cannot completely adjust potential confounding factors. Second, preexisting CKD could be a risk factor for AKI[35,36]. However, due to the lack of zero-time kidney biopsy information, history of CKD, and pre-admission serum creatinine levels, we could not determine preexisting CKD with or without AKI. Consequently, confounding AKI with undiagnosed CKD may have biased the prognostic estimates toward worse outcomes. Third, the

Creatinine trend (event number)	Event per 1000 patient-years (95% CI)	Unadjusted			Model 1			Model 2			
		HR	95% CI	*P* value	HR	95% CI	*P* value	HR	95% CI	*P* value	V
Decreasing sCr											
No-AKI (*n* = 116)	193.333 (159.755–231.884)	Ref			Ref			Ref			
AKI (*n* = 56)	176.100 (133.024–228.681)	1.02	0.74–1.40	0.907	1.14	0.82–1.59	0.443	1.09	0.79–1.52	0.590	0.132
AKI 1 (*n* = 23)	200.000 (126.782–300.098)	1.12	0.71–1.75	0.622	1.30	0.82–2.07	0.260	1.24	0.79–1.97	0.350	0.126
AKI 2 (*n* = 14)	241.379 (131.964–404.993)	1.14	0.66–1.99	0.638	1.22	0.69–2.16	0.489	1.15	0.65–2.05	0.630	0.126
AKI 3 (*n* = 19)	131.034 (78.891–204.626)	0.86	0.53–1.39	0.536	0.94	0.57–1.56	0.826	0.92	0.56–1.52	0.750	0.126
Stable or increasing sCr											
No-AKI (*n* = 155)	199.485 (169.316–233.478)	Ref			Ref			Ref			
AKI (*n* = 144)	198.895 (167.736–234.162)	1.27	1.01–1.59	0.040	1.38	1.09–1.73	0.006	1.36	1.07–1.72	0.012	0.128
AKI 1 (*n* = 64)	188.235 (144.964–240.372)	1.13	0.84–1.51	0.414	1.18	0.88–1.58	0.270	1.18	0.88–1.60	0.260	0.127
AKI 2 (*n* = 30)	194.805 (131.434–278.096)	1.24	0.84–1.83	0.280	1.38	0.93–2.06	0.110	1.36	0.91–2.04	0.140	0.127
AKI 3 (*n* = 50)	217.391 (161.352–286.603)	1.53	1.11–2.11	0.008	1.77	1.27–2.45	< 0.001	1.70	1.22–2.38	< 0.001	0.127

Table 4. Impact of AKI and AKI stage on death-censored graft failure according to changing trends of serum creatinine levels. Model 1 was adjusted for various donor-related (age, sex, BMI, diabetes mellitus, hypertension, DCD, death due to CVA cause, Hepatitis C virus), recipient-related (age, sex, diabetes mellitus, previous kidney transplantation, dialysis duration, ischemic heart diseases, CVA, cancer), transplantation-related (HLA mismatch number). Model 2 was adjusted for center effects in addition to model 1 and was further adjusted using IPTW for donor-related (sex, weight, hypertension, DCD, death due to CVA cause) covariates. Numbers in parentheses indicate the number of events in each group. *AKI* acute kidney injury, *CI* confidence interval, *HR* hazard ratio, *n* number, *ref* reference, *CVA* cerebrovascular accidents, *DCD* donation after circulatory death, *sCr* serum creatinine, *V* Variance of random effect for center.

baseline serum creatinine concentration is ideally defined as the average of all values measured within the year before admission[37]; however, owing to the absence of this information, we defined creatinine levels at admission as the baseline levels[16,17,22]. In the last, the prognostic impact of AKI etiology could not be analyzed due to insufficient information. Kidneys from donors with AKI due to interstitial nephritis may warrant more active utilization, given their higher potential for recovery compared with AKI associated with acute cortical necrosis or vasculitis[38].

Nevertheless, the present study is of high significance. First, this study was a nationwide study with a large-sized, relatively long-term follow-up duration compared to previous studies. Second, we analyzed the impact of AKI on death-censored graft failure in various subgroups, such as AKI stage, K-KDPI, changing trends of serum creatinine levels. Based on this subgroup analysis, we could recommend accepting donor kidneys with all AKI stages in K-KDPI < 70 subgroup, all AKI stages in the decreasing-creatinine trend, and AKI stage 1 and 2 in stable- or increasing- creatinine trend (Fig. S6). Third, this study is the first nationwide Asian study that investigated different characteristics as against those of Western countries, such as a very long waiting time for DDKT and low discard rate of donor's kidneys. Therefore, we believe our results would contribute to a balanced and better understanding of the impact of AKI on graft outcomes on an international basis.

In conclusion, donor AKI, especially stage 3 was associated with a higher risk for death-censored graft failure; however, subgroups of K-KDPI < 70% or a decreasing-creatinine trend, showed comparable risk of graft failure. Therefore, the active utilization of AKI kidneys with relatively favorable prognostic indicators may help address the organ shortage, particularly in Asian countries with a long waiting time for DDKT.

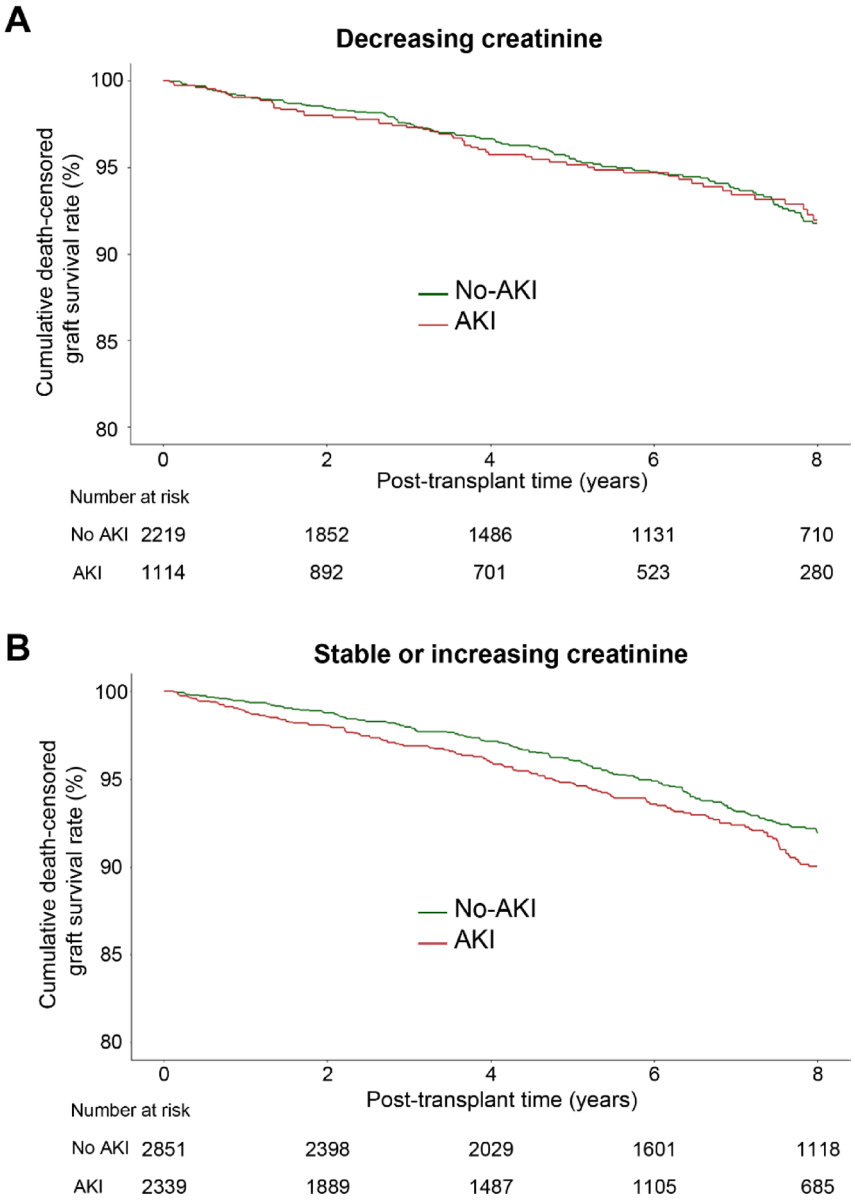

Fig. 4. Death-censored graft failure rates according to changing trends in serum creatinine levels. **A** Comparison of death-censored graft failure rates between AKI group and no-AKI group in the decreasing-creatinine group (Log rank test, $P = 0.907$). **B** Comparison of death-censored graft failure rates between AKI group and no-AKI group in the stable- or increasing-creatinine group (Log rank test, $P = 0.040$). *AKI* acute kidney injury.

Data availability

Data supporting the findings of this study are available from the corresponding author uponreasonable request.

Received: 12 October 2025; Accepted: 20 January 2026
Published online: 23 January 2026

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Acknowledgements

We thank the Korean Network for Organ Sharing (KONOS) and the National Health Insurance Data Sharing Service (NHISS) for sharing their database.

Author contributions

Research idea and study design: O.N., J.Y.; data acquisition: O.N., G.L., T.Y.K.; data analysis/interpretation: O.N., G.L., T.Y.K., B.K., J.Y.; statistical analysis: O.N., G.L., T.Y.K., J.H.L., H.B.K.; wrote the manuscript: O.N., J.Y. All authors read and approved of the final manuscript.

Funding

This study was supported by a grant from the National Institute of Organ, Tissue and Blood. Management (20232400C1B-00), which was not involved in the design or analysis of the study.

Declarations

Competing interests
The authors declare no competing interests.

Consent for publication
All co-authors agreed to publication of the acquired data.

Additional information
Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-026-37147-0.

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