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Self-Reported Sleep Duration, Death-Censored Graft Failure and Health-Related Quality of Life Among Kidney Transplant Recipients: Results From the KNOW-KT Study

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ABSTRACT

Aim: Sleep duration is associated with cardiovascular events, mortality and rapidly declining kidney function in the general population. Less is known about its impact on transplant outcomes and health-related quality of life (HRQOL) among kidney transplant recipients (KTRs).

Methods: This prospective multicenter longitudinal cohort study included 986 incident KTRs and investigated self-reported sleep duration at the time of kidney transplantation. Analyses were performed to examine associations between sleep duration and biopsy-proven acute rejection, death-censored graft failure, major cardiovascular events, all-cause mortality and HRQOL.

Results: During a median follow-up period of 8 years, the death-censored graft failure rate was significantly higher among patients with sleep durations of ≤5 h (adjusted hazard ratio [aHR], 3.69; 95% confidence interval [CI], 1.42–9.57; $p=0.007$), 6 h (aHR, 3.25; 95% CI, 1.34–7.87; $p=0.009$), 8 h (aHR, 2.93; 95% CI, 1.22–7.01; $p=0.016$) and ≥9 h (aHR, 3.58; 95% CI, 1.29–9.93; $p=0.014$), compared to patients with a 7-h sleep duration. Sleep duration was not significantly associated with biopsy-proven acute rejection, major cardiovascular events or all-cause mortality. Multivariable linear regression demonstrated that shorter sleep duration (≤5 h) was associated with lower HRQOL, indicated by decreases in the physical component summary score (β, −4.51; 95% CI, −8.75 to −0.26; $p=0.038$) and mental component summary score (β, −5.47; 95% CI, −10.29 to −0.65; $p=0.026$), relative to patients with a 7-h sleep duration.

Conclusion: Compared to a 7-h sleep duration, both shorter and longer sleep durations were associated with a significantly higher risk of death-censored graft failure among incident KTRs. Additionally, short sleep duration was significantly correlated with impaired HRQOL.

Trial Registration: NCT02042963; clinicaltrials.gov identifier

Abbreviations: ACD, all-cause death; aHR, adjusted hazard ratio; BPAR, biopsy-proven acute rejection; CI, confidence interval; CKD, chronic kidney disease; DCGF, death-censored graft failure; eGFR, estimated glomerular filtration rate; HR, hazard ratio; HRQOL, health-related quality of life; KTR, kidney transplant recipient; MACE, major adverse cardiovascular events; MCS, mental component summary; PCS, physical component summary.

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Summary at a Glance

- Both shorter and longer sleep durations are significantly associated with a higher risk of DCGF in incident KTRs.
- Most notably, a short sleep duration of 5 h or less was significantly associated with impaired HRQOL.
- Larger and longer-term studies are needed to determine whether assessment and intervention regarding sleep duration are associated with improved transplant graft survival in KTRs.

1 | Introduction

Sleep supports the regulation of immune and endocrine functions [1], removal of metabolic wastes [2], enhancement of cognitive function [3, 4] and restoration of kidney functional capacity and homeostasis [5, 6]. In the general population, a sleep duration that is too short or long is associated with greater risks of overall mortality [7, 8], cardiovascular events [9, 10], hypertension [11], diabetes mellitus [12], obesity [13] and rapid decline in kidney function [14].

Previous studies have explored the criteria for optimal sleep duration in the general population. The National Sleep Foundation recommends a sleep duration of 7–9 h for healthy young adults and 7–8 h for older adults [15]. A sleep duration of 7 h is associated with the lowest mortality [7] and the lowest risk of cardiovascular events [9] in the general population. A meta-analysis revealed that a sleep duration of < 7 h or > 9 h is associated with increased cardiovascular mortality [10]. Moreover, a sleep duration of < 5 h, compared to 7–8 h, is associated with a rapid decline in kidney function [14]. In a study of pre-dialysis patients with chronic kidney disease (CKD), a sleep duration of ≤ 5 h or ≥ 9 h, compared to 7 h, was associated with impaired health-related quality of life (HRQOL) [16].

Despite the clinical significance of sleep duration, few studies have examined its impact on transplant outcomes and HRQOL among kidney transplant recipients (KTRs). Notably, the association between sleep duration and graft failure in KTRs has not previously been investigated. This prospective multicenter longitudinal cohort study was performed to determine the association between self-reported sleep duration and comprehensive outcomes among KTRs.

2 | Methods

2.1 | Study Participants

A total of 1080 incident KTRs were enrolled from the Korean Cohort Study for Outcome in Patients With Kidney Transplantation (KNOW-KT) between 2012 and 2016 and were followed up until 2023 (clinicaltrials.gov identifier: NCT02042963). At each annual visit, the subjects underwent scheduled tests and the investigators reviewed their recent medical history and event occurrence. After 94 KTRs were excluded due to a lack of information about their sleep duration, 986 participants were included in the study (Figure 1).

2.2 | Self-Reported Sleep Duration

The KTRs self-reported their sleep duration at the time of transplant, as part of a prospective multicenter longitudinal cohort study. Participants were asked "How many hours do you usually sleep per day?" and were divided into five groups based on their reported sleep duration: ≤ 5, 6, 7, 8 and ≥ 9 h. A sleep duration of 7 h was set as the reference, in accordance with the findings of previous studies on sleep duration [7, 9, 10, 14–16].

2.3 | Outcomes

The primary outcome was death-censored graft failure (DCGF) and the secondary outcomes were biopsy-proven acute rejection (BPAR), major adverse cardiovascular events (MACE), all-cause death (ACD) and longitudinal decline in estimated glomerular filtration rate (eGFR). BPAR was defined as biopsy-proven acute T-cell-mediated rejection, acute antibody-mediated rejection, chronic active T-cell-mediated rejection or chronic active antibody-mediated rejection. DCGF was defined as the need for dialysis or receipt of a new kidney transplant. For this analysis, patients who died with a functioning graft were censored at the time of death. MACE included cardiovascular death, myocardial infarction, unstable angina, percutaneous coronary intervention, coronary artery bypass grafting and stroke. eGFR slopes were assessed according to sleep duration.

2.4 | HRQOL

The Kidney Disease Quality of Life Short Form (KDQOL-SF) is a self-reported questionnaire that comprises the 36-item Short Form Health Survey (SF-36), along with kidney disease-targeted items and is used to comprehensively assess HRQOL in patients with kidney disease [17, 18]. The validated Korean versions of KDQOL-SF [19] and SF-36 [20] were used in this study. The SF-36 includes a physical component summary (PCS) with four subscales (physical function, role limitation due to physical problems, bodily pain and general health) and a mental component summary (MCS) with four subscales (vitality, role limitation due to emotional problems, social function and mental health). Each scale is scored from 0 to 100, with higher scores indicating better HRQOL.

2.5 | Other Variables

Possible confounders for BPAR, DCGF, MACE and all-cause mortality included age; sex; body mass index; diabetes; hypertension; coronary artery disease; cerebrovascular disease; congestive heart failure; deceased-donor kidney transplantation; donor age; retransplantation; desensitisation, for example, direct crossmatch (+) plus donor-specific antibodies (+), direct crossmatch (−) plus donor-specific antibodies (+) or ABO-incompatible kidney transplantation; total number of human leukocyte antigen mismatches; antithymocyte

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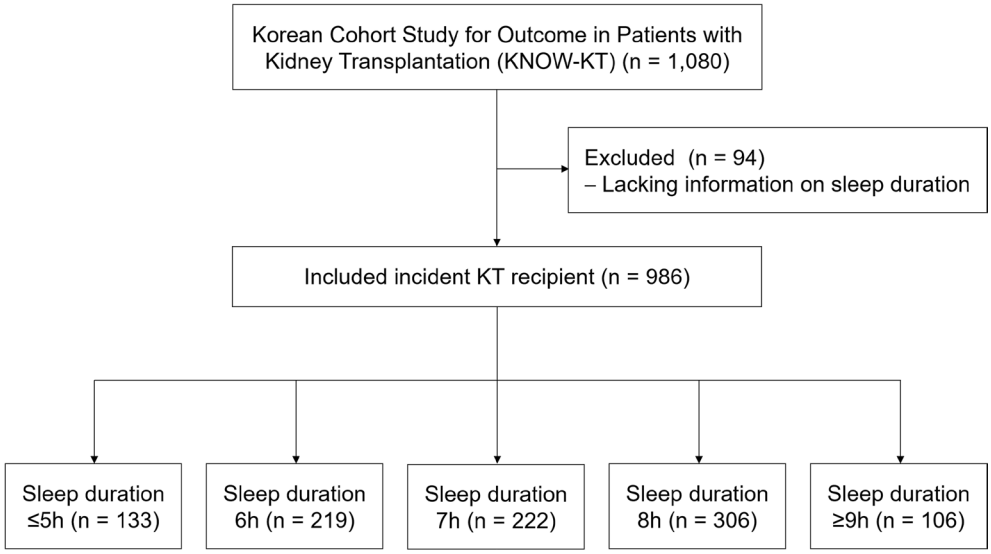


FIGURE 1 | Flow chart of study inclusion. Of the 1080 enrolled KTRs, 94 were excluded due to a lack of information regarding self-reported sleep duration.

globulin induction; use of calcineurin inhibitors (tacrolimus or cyclosporine); steroid use; alcohol consumption; smoking; marital status; employment; and low income. Possible confounders for HRQOL included age, sex, body mass index, diabetes, hypertension, coronary artery disease, cerebrovascular disease, retransplantation, alcohol consumption, smoking, marital status, employment and low income. Low income was defined as a personal monthly income of less than approximately US$1500.

2.6 | Statistical Analysis

Continuous variables are expressed as mean ± standard deviation or median with interquartile range. Trends across the five groups were evaluated using the Jonckheere-Terpstra test for continuous variables and the Cochran-Armitage test or Cochran–Mantel–Haenszel trend test for categorical variables. Multivariate Cox proportional hazards models were used to analyse associations of sleep duration with BPAR, DCGF, MACE and all-cause mortality. The covariate sets were selected based on their clinical relevance to each specific outcome. For example, donor-related and immunological factors were included in the DCGF models but not in the HRQOL models, since these factors are expected to have limited direct impact on patient-reported quality of life. Grey's test was used to compare differences in cumulative outcome incidence according to sleep duration and cumulative incidence functions were estimated using the Aalen-Johansen method, with all-cause death treated as a competing risk for graft failure. The eGFR slope was determined using generalised linear mixed models. Linear regression analyses were used to examine associations between sleep duration and HRQOL. To account for the risk of Type 1 errors arising from multiple statistical comparisons, we applied the Benjamini-Hochberg procedure to calculate false discovery rate-adjusted p values. This adjustment was prioritised for the primary clinical outcome (DCGF) and the key secondary outcome (eGFR slope), to ensure the robustness of the core results. Other secondary assessments, including HRQOL, were considered exploratory in nature and are therefore reported using unadjusted p values to avoid overlooking potentially meaningful clinical trends. Statistical analyses were performed using SAS for Windows, version 9.4 (SAS Institute, Cary, NC, USA) and R, version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria). A p value of < 0.05 was considered statistically significant.

3 | Results

3.1 | Baseline Characteristics

Table 1 presents the patients' baseline characteristics grouped by sleep duration. Among the 986 eligible KTRs, the most common sleep duration was 8 h (31.0%), followed by 7 h (22.5%). Sleeping for ≥ 9 h was the least common (10.8%). The mean age was 45.5 ± 11.5 and 361 patients (36.6%) were female. Comorbidities included diabetes (240 patients; 25.5%), coronary artery disease (60 patients; 6.4%) and cerebrovascular disease (37 patients; 3.9%). Regarding immunosuppressants, 918 patients (93.1%) used tacrolimus and 59 (6.0%) used cyclosporine. A total of 361 patients (36.6%) drank alcohol and 67 (6.8%) were current smokers. The majority of patients were married (719 patients; 72.9%).

3.2 | Self-Reported Sleep Duration and Outcomes

During the median follow-up period of 8 years (interquartile range, 7.0–9.0), the following incidence rates were observed: BPAR, 6.4 per 100 person-years; DCGF, 0.9 per 100 person-years; MACE, 0.7 per 100 person-years; and all-cause mortality, 0.4 per 100 person-years. Figure 2 shows the cumulative incidence curves for each outcome. The incidence of graft failure was significantly higher among patients with a sleep duration of 6 h, compared to 7 h ($p = 0.041$). The incidences of

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TABLE 1 | Baseline characteristics stratified by self-reported sleep duration.

	Total ($n=986$)	≤5 h ($n=133$)	6 h ($n=219$)	7 h ($n=222$)	8 h ($n=306$)	≥9 h ($n=106$)	p for trend
Age, years	45.5 ± 11.5	47.2 ± 10.8	45.1 ± 12.0	44.6 ± 11.1	45.8 ± 11.7	45.3 ± 11.6	0.594
Sex, female	361 (36.6%)	51 (38.3%)	81 (37.0%)	84 (37.8%)	107 (35.0%)	38 (35.8%)	0.514
BMI, kg/m^2	22.9 ± 3.5	22.8 ± 3.3	22.9 ± 3.3	22.9 ± 3.5	23.0 ± 3.5	22.8 ± 4.1	0.883
Diabetes	240 (25.5%)	38 (29.5%)	47 (22.4%)	57 (26.9%)	69 (23.8%)	29 (28.7%)	0.898
Hypertension	901 (95.6%)	122 (94.6%)	199 (94.8%)	204 (96.2%)	280 (96.6%)	96 (95.0%)	0.432
Coronary artery disease	60 (6.4%)	9 (7.0%)	16 (7.6%)	7 (3.3%)	22 (7.6%)	6 (5.9%)	0.868
Cerebrovascular disease	37 (3.9%)	5 (3.9%)	7 (3.3%)	8 (3.8%)	12 (4.1%)	5 (5.0%)	0.579
Congestive heart failure	21 (2.2%)	3 (2.3%)	6 (2.9%)	4 (1.9%)	7 (2.4%)	1 (1.0%)	0.867
Donor type							0.178
Living	805 (81.6%)	112 (84.2%)	166 (75.8%)	179 (80.6%)	259 (84.6%)	89 (84.0%)	
Deceased	181 (18.4%)	21 (15.8%)	53 (24.2%)	43 (19.4%)	47 (15.4%)	17 (16.0%)	
Donor age, years	44.0 ± 11.9	44.0 ± 12.4	43.2 ± 11.9	43.5 ± 12.1	44.9 ± 11.4	44.4 ± 12.6	0.166
Total HLA mismatches, median [IQR]	3 [2–5]	3 [2–5]	3 [2–4]	3 [2–5]	3 [2–5]	3 [2–4]	0.644
Retransplantation	60 (6.1%)	9 (6.8%)	14 (6.4%)	14 (6.3%)	19 (6.2%)	4 (3.8%)	0.446
Desensitisation therapy	251 (25.5%)	34 (25.6%)	61 (27.9%)	60 (27.0%)	77 (25.2%)	19 (17.9%)	0.181
Induction therapy							0.058
IL-2RB	901 (91.4%)	117 (88.0%)	201 (91.8%)	199 (89.6%)	282 (92.2%)	102 (96.2%)	
ATG	84 (8.5%)	16 (12.0%)	18 (8.2%)	23 (10.4%)	23 (7.5%)	4 (3.8%)	
Immunosuppressant doses at discharge							
Tacrolimus, mg/day	6.6 ± 12.5	6.0 ± 3.8	6.2 ± 3.6	7.3 ± 21.5	6.1 ± 3.4	8.3 ± 20.1	0.425
Cyclosporine, mg/day	503.3 ± 391.0	438.9 ± 315.5	393.1 ± 378.2	515.0 ± 364.4	563.2 ± 447.9	550.0 ± 287.2	0.084
Mycophenolate, mg/day	1183.2 ± 382.7	1141.8 ± 386.3	1164.1 ± 357.3	1223.4 ± 415.0	1180.9 ± 376.0	1215.3 ± 394.5	0.389
Myfortic acid, mg/day	697.3 ± 407.8	699.0 ± 411.1	760.1 ± 355.7	683.3 ± 408.1	656.8 ± 447.0	704.6 ± 400.5	0.208
Steroid dose, mg/day	16.4 ± 7.2	16.3 ± 8.0	17.1 ± 7.0	15.9 ± 6.8	16.3 ± 7.3	16.1 ± 7.2	0.503
Immunosuppressants at discharge, n (%)							
Tacrolimus	918 (93.1%)	126 (94.7%)	204 (93.2%)	204 (91.9%)	283 (92.5%)	101 (95.3%)	0.506

(Continues)

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TABLE 1 | (Continued)

	Total ($n=986$)	≤5 h ($n=133$)	6 h ($n=219$)	7 h ($n=222$)	8 h ($n=306$)	≥9 h ($n=106$)	*p* for trend
Cyclosporine	59 (6.0%)	6 (4.5%)	14 (6.4%)	16 (7.2%)	20 (6.5%)	3 (2.8%)	0.656
Mycophenolate	505 (51.2%)	73 (54.9%)	112 (51.1%)	100 (45.0%)	164 (53.6%)	56 (52.8%)	0.820
Myfortic acid	324 (32.9%)	39 (29.3%)	77 (35.2%)	85 (38.3%)	88 (28.8%)	35 (33.0%)	0.981
Steroid	980 (99.4%)	133 (100.0%)	219 (100.0%)	219 (98.6%)	303 (99.0%)	106 (100.0%)	0.175
Alcohol consumption	361 (36.6%)	46 (32.3%)	87 (39.7%)	83 (37.4%)	114 (37.3%)	34 (32.1%)	0.868
Current smoker	67 (6.8%)	11 (8.3%)	13 (5.9%)	11 (5.0%)	25 (8.2%)	7 (6.6%)	0.856
Married	719 (72.9%)	100 (75.2%)	159 (72.6%)	165 (74.3%)	217 (70.9%)	78 (73.6%)	0.556
Low income	717 (72.7%)	91 (68.4%)	163 (74.4%)	159 (71.6%)	220 (71.9%)	84 (79.2%)	0.267

Note: Values are presented as the mean ± standard deviation, number (%) or median (interquartile range).
Abbreviations: ATG, antithymocyte globulin; BMI, body mass index; HLA, human leukocyte antigen; IL-2RB, interleukin-2 receptor blocker.

BPAR, MACE and ACD did not significantly differ between the five groups.

Table 2 shows the results of a multivariate Cox proportional hazards model for the associations of sleep duration with BPAR, DCGF, MACE and all-cause mortality. DCGF risk was significantly higher in patients with a sleep duration of ≤5 h (adjusted hazard ratio [aHR], 3.69; 95% confidence interval [CI], 1.42–9.57; $p=0.007$), 6 h (aHR, 3.25; 95% CI, 1.34–7.87; $p=0.009$), 8 h (aHR, 2.93; 95% CI, 1.22–7.01; $p=0.016$) and ≥9 h (aHR, 3.58; 95% CI, 1.29–9.93; $p=0.014$), compared in patients with a 7-h sleep duration. Sleep duration was not significantly associated with BPAR, MACE or all-cause mortality (Table 2).

Table 3 presents the slopes of eGFR decline, according to sleep duration. Shorter sleep duration was associated with steeper decline of eGFR (p for trend <0.001), with eGFR decline slopes of −1.03, −0.06, −0.37, −0.36 and −0.26 mL/min per 1.73 m^2 per year among patients with sleep durations of ≤5, 6, 7, 8 and ≥9 h, respectively.

After applying the Benjamini-Hochberg adjustment for multiple comparisons, the primary associations remained statistically significant. Specifically, there was still a significantly increased DCGF risk in the non-optimal sleep duration groups (Model 2; adjusted p value of 0.016) and a significant association between sleep duration and the slope of eGFR decline ($p<0.001$).

3.3 | Self-Reported Sleep Duration and HRQOL

Sleep duration showed inverse U-shaped relationships with PCS scores and all subscale scores. Among the five groups, patients who slept 7 h had the highest PCS (59.8 ± 19.6) and MCS (57.0 ± 21.7) scores, whereas those who slept ≤5 h had the lowest PCS (54.2 ± 19.9) and MCS (50.0 ± 22.5) scores (Figure 3). Multivariable linear regression analysis (Table 4) revealed that patients with sleep durations of ≤5 h had significantly lower PCS (β, −4.51; 95% CI, −8.75 to −0.26; $p=0.038$) and MCS (β, −5.47; 95% CI, −10.29 to −0.65; $p=0.026$) scores, compared to patients with 7-h sleep durations.

4 | Discussion

In this prospective cohort study of 986 incident KTRs, a 7-h sleep duration was used as a reference and a shorter or longer sleep duration was significantly associated with a nearly three-fold higher risk of DCGF over a median follow-up of 8 years, even after adjustment for relevant clinical, immunologic and sociodemographic variables. Additionally, a shorter sleep duration was associated with steeper declines in eGFR and a sleep duration of ≤5 h was significantly correlated with impaired HRQOL. On the other hand, sleep duration was not associated with differing risks of BPAR, MACE and all-cause mortality among KTRs.

Although KTRs have universally experienced CKD progression and/or dialysis, they constitute a unique group distinct from CKD patients, end-stage kidney disease patients on dialysis and

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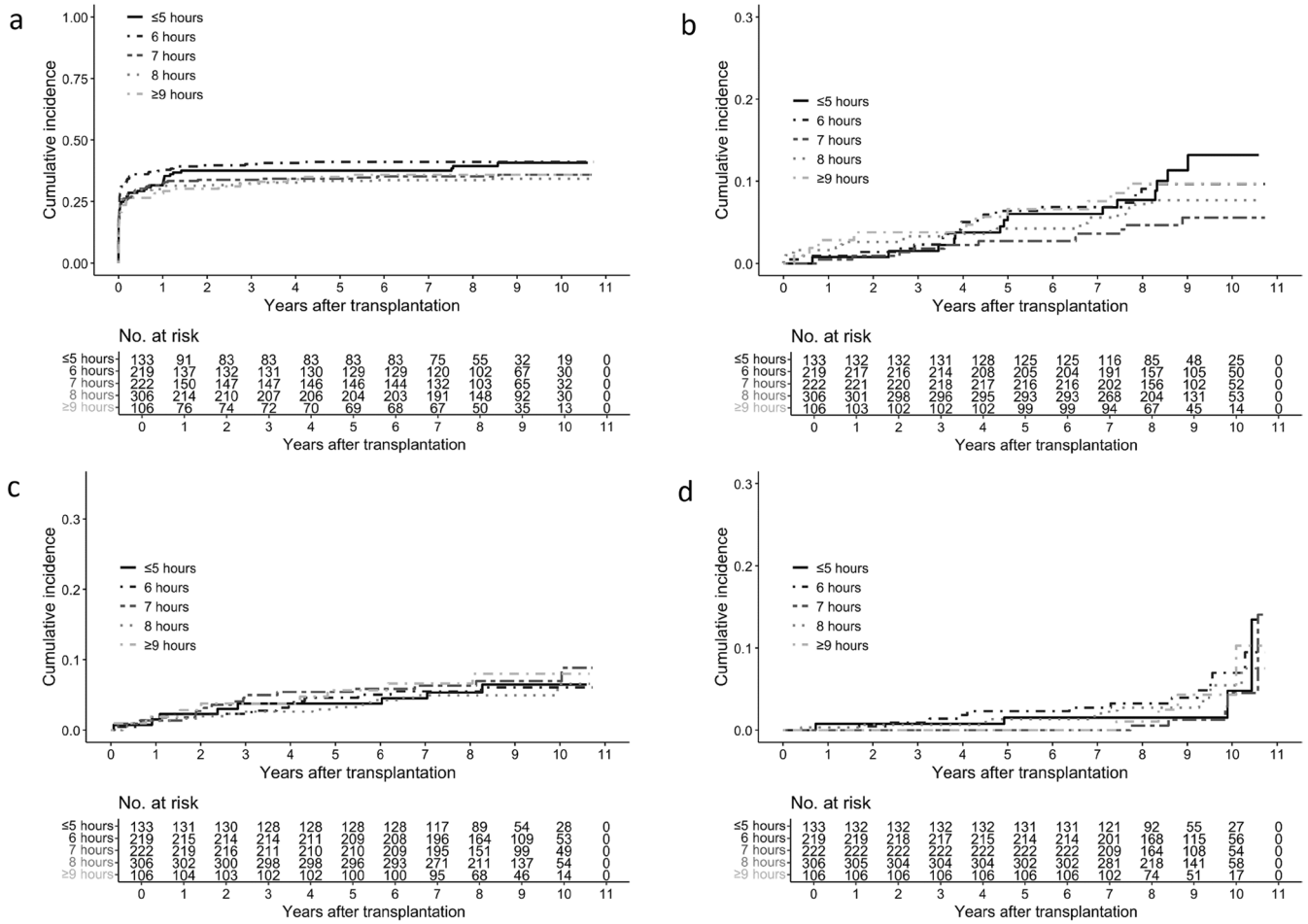


FIGURE 2 | Cumulative incidence curves for biopsy-proven acute rejection (a), graft failure (b), major adverse cardiovascular events (c) and all-cause death (d). The incidence of graft failure was significantly higher among patients with a 6-h sleep duration, compared to those with a 7-h sleep duration ($p = 0.041$). The groups did not significantly differ in the estimated incidence rates of biopsy-proven acute rejection, major adverse cardiovascular events or all-cause death.

the general population. Notably, KTRs have a single kidney; take immunosuppressants, including calcineurin inhibitors and corticosteroids; commonly have various comorbidities, including obesity; and carry higher risks of cardiovascular complications and cancer. Additionally, this population may have concerns about adjusting to life after transplantation, rejection episodes or graft loss [21]. All of these factors can interfere with sleep quality among KTRs. Previous studies show that the prevalence of insomnia among KTRs is similar to that in the general population, but considerably lower than among dialysis patients [22]. In a study of 110 KTRs, nearly half of the patients reported improved sleep quality after kidney transplantation, as evaluated with the widely used self-report questionnaire, the Pittsburgh Sleep Quality Index (PSQI); however, poor sleep quality persisted in this population after transplantation [23]. Other cross-sectional studies using the PSQI have also reported poor sleep quality among KTRs [24, 25].

Previous studies have reported decreased sleep duration or sleep quality in KTRs; however, little information is available regarding how sleep duration may impact transplant outcomes. The present study is the first to identify an association between sleep duration and the risk of DCGF. This analysis confirmed that DCGF risk was significantly increased when sleep duration was shorter or longer than 7 h, even after adjustment for related clinical, immunologic and sociodemographic variables. Previous studies also show that short sleep duration is associated with rapid deterioration of kidney function, suggesting that changes in sleep duration may induce established CKD risk factors [5, 14, 26]. In the general population, it has been demonstrated that sleep durations shorter than 5–7 h are associated with the onset of new hypertension [27] and type 2 diabetes mellitus [28, 29]. Several earlier reports have reported that sleep deprivation can lead to sympathetic nervous system hyperactivity [30], elevated blood pressure and pulse rate [31, 32] and alterations in glucose metabolism [33]. Evidence also indicates that long sleep duration is linked to negative physical indicators—such as poor health status, higher depression scores, older age and increased drinking and smoking rates—all of which contribute to unfavourable outcomes [34–36]. However, the present analyses included adjustment for significant clinical variables, including diabetes mellitus and hypertension, as well as clinical and sociodemographic variables, for example, alcohol and smoking habits. Therefore, the increased risk of graft loss observed in groups with both shorter and longer sleep durations was likely due to mechanisms other than established CKD risk factors. It can be

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TABLE 2 | Crude and adjusted hazard ratios for biopsy-proven acute rejection (BPAR), death-censored graft failure (DCGF), major adverse cardiovascular events (MACEs) and all-cause death (ACD) according to self-reported sleep duration.

	Total (*N*)	Events, *n* (%)	Crude		Model 1		Model 2	
			HR (95% CI)	*p* value	HR (95% CI)	*p* value	HR (95% CI)	*p* value
BPAR								
≤5 h	133	53 (39.8%)	1.12 (0.79–1.59)	0.514	1.10 (0.77–1.59)	0.591	1.09 (0.76–1.57)	0.656
6 h	219	90 (41.1%)	1.19 (0.88–1.61)	0.253	1.12 (0.82–1.53)	0.468	1.14 (0.84–1.56)	0.404
7 h	222	79 (35.6%)	Reference		Reference		Reference	
8 h	306	104 (34.0%)	0.95 (0.71–1.28)	0.744	0.91 (0.67–1.23)	0.529	0.90 (0.67–1.22)	0.505
≥9 h	106	38 (35.8%)	0.98 (0.66–1.44)	0.909	0.92 (0.62–1.38)	0.702	0.92 (0.61–1.37)	0.672
DCGF								
≤5 h	133	14 (10.5%)	2.32 (1.05–5.11)	0.037	3.58 (1.38–9.28)	0.009	3.69 (1.42–9.57)	0.007
6 h	219	20 (9.1%)	1.90 (0.91–3.97)	0.088	3.23 (1.34–7.78)	0.009	3.25 (1.34–7.87)	0.009
7 h	222	11 (5.0%)	Reference		Reference		Reference	
8 h	306	22 (7.2%)	1.60 (0.77–3.30)	0.205	2.98 (1.25–7.13)	0.014	2.93 (1.22–7.01)	0.016
≥9 h	106	10 (9.4%)	1.98 (0.84–4.68)	0.118	3.72 (1.35–10.26)	0.011	3.58 (1.29–9.93)	0.014
MACE								
≤5 h	133	8 (6.0%)	0.83 (0.36–1.94)	0.670	0.59 (0.24–1.46)	0.254	0.59 (0.24–1.46)	0.255
6 h	219	13 (5.9%)	0.81 (0.39–1.68)	0.573	0.60 (0.28–1.27)	0.180	0.61 (0.29–1.31)	0.207
7 h	222	16 (7.2%)	Reference		Reference		Reference	
8 h	306	16 (5.2%)	0.72 (0.36–1.44)	0.355	0.56 (0.27–1.16)	0.117	0.55 (0.26–1.14)	0.105
≥9 h	106	8 (7.5%)	1.07 (0.46–2.49)	0.882	0.83 (0.34–2.02)	0.685	0.82 (0.34–2.01)	0.670
ACD								
≤5 h	133	4 (3.0%)	1.47 (0.39–5.48)	0.566	1.25 (0.28–5.63)	0.776	1.33 (0.29–5.98)	0.713
6 h	219	12 (5.5%)	2.45 (0.86–6.95)	0.093	2.77 (0.93–8.27)	0.068	2.67 (0.89–7.99)	0.113
7 h	222	5 (2.3%)	Reference		Reference		Reference	
8 h	306	12 (3.9%)	1.90 (0.67–5.39)	0.229	2.50 (0.82–7.61)	0.106	2.55 (0.83–7.79)	0.102
≥9 h	106	4 (3.8%)	1.89 (0.51–7.03)	0.345	1.70 (0.43–6.76)	0.452	1.61 (0.40–6.46)	0.506

Note: Model 1: Adjusted for age, sex, body mass index, diabetes, hypertension, coronary artery disease, cerebrovascular disease, congestive heart failure, donor type (deceased), donor age, total number of human leukocyte antigen mismatches, retransplantation, desensitisation, antithymocyte globulin induction, use of calcineurin inhibitors (tacrolimus or cyclosporine) and steroid use. Model 2: Adjusted for all covariates in Model 1 plus alcohol consumption, smoking, marital status and low income.

Abbreviations: CI, confidence interval; HR, hazard ratio.

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assumed that this relationship was based on non-immunological mechanisms, since the results showed no significant association between sleep duration and BPAR.

The association between sleep duration and DCGF among KRTs could potentially be related to increased inflammation and oxidative stress. Previous studies have reported that shorter or longer sleep durations are associated with increased inflammatory molecules, including interleukin-6, C-reactive protein or tumour necrosis factor-α [37, 38]. Sleep deprivation has been linked to elevated proinflammatory cytokines [39]. Considering the important role of chronic inflammation in CKD progression [40], suboptimal sleep duration may adversely affect the kidneys by accelerating the changes caused by the inflammatory response. Chronic sleep deprivation profoundly affects the hypothalamic–pituitary–adrenal axis, resulting in dysregulated secretion of stress hormones, like cortisol, which can lead to chronic high blood pressure, a leading cause of kidney damage [41]. Furthermore, sleep deprivation raises oxidative stress by interfering with cellular processes and increasing reactive oxygen species production, leading to cellular damage and decreased function [42]. Another possible mechanism is that circadian rhythm disruption might predispose to renal impairment, raising the risk of CKD. A previous study demonstrated that mice with genetic deletion of the circadian clock protein are more susceptible to adenine-induced CKD development, compared to wild-type mice [43]. Moreover, in patients with type 1 diabetes, circadian disruption precedes the development of microalbuminuria or clinical evidence of kidney disease [44]. The kidneys themselves have intrinsic circadian clocks and are involved in various physiological processes regulated by the circadian system and circadian disruption can lead to inflammation, fibrosis and oxidative stress, which can contribute to kidney damage [45]. However, this single study could not conclusively confirm that sleep duration had a causal effect on metabolic and vascular outcomes. Further investigation is warranted to elucidate the mechanisms mediating the association between sleep duration and DCGF.

TABLE 3 | Slopes of eGFR decline according to self-reported sleep duration.

	Slope[a] of eGFR decline (95% CI) (mL/min per 1.73 m^2 per year)	*p* for trend
≤5 h	−1.03 (−1.36 to −0.70)	<0.001
6 h	−0.60 (−0.84 to −0.35)	
7 h	−0.37 (−0.62 to −0.12)	
8 h	−0.36 (−0.57 to −0.15)	
≥9 h	−0.26 (−0.60 to 0.08)	

Abbreviations: CI, confidence interval; eGFR, estimated glomerular filtration rate.
[a]Adjusted for age, sex, body mass index, diabetes, hypertension, coronary artery disease, cerebrovascular disease, donor type (deceased), donor age, total number of human leukocyte antigen mismatches, retransplantation, desensitisation, antithymocyte globulin induction, use of calcineurin inhibitors (tacrolimus or cyclosporine), steroid use and biopsy-proven acute rejection.

Regarding the long-term follow-up data, the cumulative incidence curves exhibited a more pronounced divergence during the later time-points. To address concerns regarding the declining number at risk toward the end of the study, we conducted a sensitivity analysis restricted to an 8-year follow-up period. The sensitivity analysis results were consistent with our primary findings, demonstrating a robust association between non-optimal sleep duration and DCGF, even when excluding the final years of follow-up, during which the sample size was smaller (Table S1). This suggests that the observed prognostic value of baseline sleep duration is not disproportionately influenced by the reduced number of remaining participants at the end of the 10-year period.

Although sleep duration is reportedly associated with cardiovascular events and mortality in the general population [7–10], the current study did not show that sleep duration was significantly associated with MACE or all-cause mortality. This may be related to the relatively low incidence rates of MACE and overall mortality in this study group, which were 0.7 per 100 person-years and 0.4 per 100 person-years, respectively. These low incidence rates may be due to characteristics of the study population. Overall, the study cohort was younger than the general population and only 6.4% of patients had prior coronary artery disease, 3.9% had cerebrovascular disease and 25.5% had diabetes. To derive more accurate and confirmatory findings in this

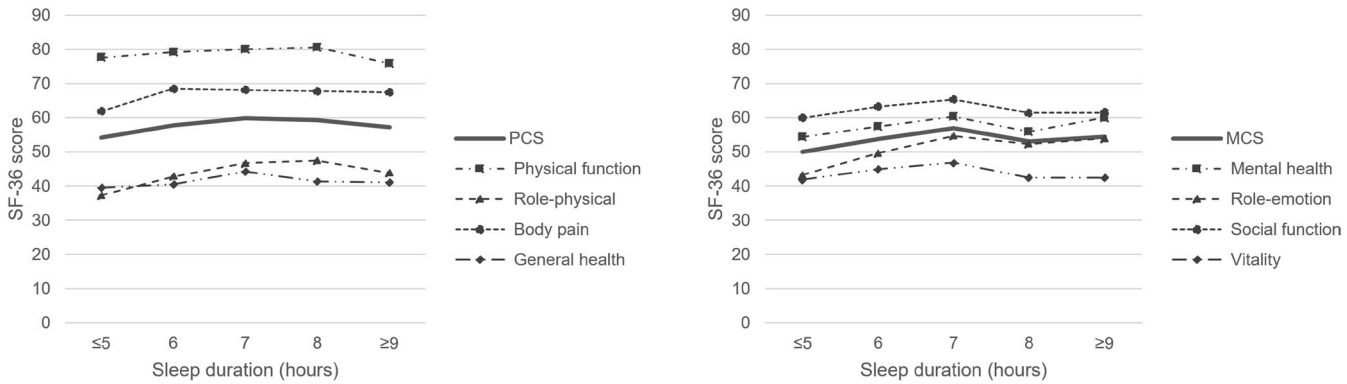


FIGURE 3 | Distribution of scores for the physical component summary (PCS), mental component summary (MCS) and the four subscales of the Short Form-36 (SF-36), according to self-reported sleep duration. Sleep duration showed inverse U-shaped correlations with PCS scores and all subscale scores. Among the five patient groups, individuals with a 7-h sleep duration exhibited the highest PCS (59.8 ± 19.6) and MCS (57.0 ± 21.7) scores, while individuals with a sleep duration of ≤5 h had the lowest PCS (54.2 ± 19.9) and MCS (50.0 ± 22.5) scores.

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TABLE 4 | Linear regression model examining health-related quality of life according to self-reported sleep duration.

	Crude		Model 1		Model 2	
	β (95% CI)	*p* value	*β* (95% CI)	*p* value	*β* (95% CI)	*p* value
Physical component summary						
≤5 h	−5.69 (−9.87 to −1.51)	0.008	−4.83 (−9.06 to −0.61)	0.025	−4.51 (−8.75 to −0.26)	0.038
6 h	−2.07 (−5.70 to −1.57)	0.265	−1.28 (−4.97 to 2.41)	0.495	−1.51 (−5.20 to 2.19)	0.423
7 h	0.00	Reference	0.00	Reference	0.00	Reference
8 h	−0.52 (−3.89 to 2.85)	0.762	0.003 (−3.44 to 3.44)	0.999	0.05 (−3.89 to 3.49)	0.978
≥9 h	−2.69 (−7.22 to −1.84)	0.244	−2.69 (−7.31 to 1.93)	0.254	−2.50 (−7.13 to 2.12)	0.288
R^2	0.009		0.047		0.053	
Mental component summary						
≤5 h	−7.02 (−11.73 to −2.31)	0.004	−6.17 (−10.99 to −1.36)	0.012	−5.47 (−10.29 to −0.65)	0.026
6 h	−3.20 (−7.29 to 0.90)	0.126	−1.90 (−6.10 to 2.31)	0.376	−2.12 (−6.31 to 2.08)	0.322
7 h	0.00	Reference	0.00	Reference	0.00	Reference
8 h	−4.00 (−7.80 to −0.20)	0.039	−3.66 (−7.58 to 0.26)	0.067	−3.68 (−7.58 to 0.23)	0.065
≥9 h	−2.52 (−7.62 to 2.58)	0.333	−2.00 (−7.26 to 3.27)	0.457	−1.93 (−7.18 to 3.33)	0.472
R^2	0.009		0.027		0.037	

Note: Model 1: Adjusted for age, sex, body mass index, diabetes, hypertension, coronary artery disease, cerebrovascular disease, congestive heart failure and retransplantation. Model 2: Adjusted for Model 1 variables plus alcohol consumption, smoking, marriage and low income.
Abbreviation: CI, confidence interval.

regard, there remains a need for further studies evaluating sleep quality in a larger patient population and with a longer follow-up period.

With improving patient survival rates after kidney transplantation, quality of life is emerging as an increasingly important consideration. HRQOL is a patient-reported outcome encompassing physical, psychological and social health domains. Although previous studies have reported significantly lower HRQOL among KTRs compared with the general population [46], post-transplantation improvements of HRQOL have also been documented [47]. The present finding that shorter sleep duration was associated with reduced HRQOL corroborates a previously reported association between poor sleep and lower HRQOL in an investigation of 872 KTRs [48]. Moreover, another previous study among 347 KTRs reported that impaired physical HRQOL was related to higher patient mortality and graft failure risk [49]. Collectively, the available data indicate that shorter sleep duration, lower quality of life and increased risk of transplant kidney loss appear to be interconnected outcomes.

This study had several limitations. First, sleep duration was self-reported and sleep quality was not assessed. More valuable information would have been provided by the use of objective measurements (e.g., actigraphy or polysomnography) or validated questionnaires (e.g., PSQI or Epworth Sleepiness Scale) to evaluate sleep quality, insomnia or other sleep disorders at baseline. Second, this study only assessed sleep duration at the time of kidney transplantation and derived results under the assumption that sleep duration would remain static even after kidney transplantation. Limitations of the data made it impossible to integrate sleep duration at various points after kidney transplantation and to evaluate how changes in sleep duration might impact transplant outcomes. Kidney transplantation can profoundly influence various physiological and psychological parameters—including sleep patterns—due to improved renal function, reduced uremic burden and changes in medication regimens; therefore, we cannot completely exclude the possibility that post-transplantation changes in sleep duration might also affect the outcomes regarding DCGF or HRQOL. Future studies should include a more in-depth investigation of how sleep duration changes over time may influence transplant outcomes and HRQOL. Third, it is a limitation that sleep duration was assessed at the time of transplantation. The peri-transplant period is often characterised by substantial emotional and physical stress, such that assessment at this time may not fully represent a patient's stable sleep patterns before or after the surgery. The lack of longitudinal sleep data prevents us from accounting for changes in sleep habits over the 8-year follow-up period and thus our findings should be interpreted with the understanding that sleep is a dynamic behaviour. Fourth, this study did not include information about the regular use of hypnotics or sleep aids, which could influence sleep duration and outcomes. Fifth, this study included only Koreans, which may limit the generalizability of the findings to other ethnic groups. Sixth, there is uncertainty regarding the causality of sleep disturbances and adverse outcomes in KTRs—that is, it remains unclear whether sleep duration is a cause or consequence of adverse outcomes in KTRs. Seventh, despite our comprehensive adjustment for various clinical confounders, we cannot entirely exclude the possibility of residual confounding. Several critical donor and transplant-related variables—including donor acute kidney

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injury, donor comorbidities and cold ischemia time—were unavailable for analysis. We also did not have access to data on obstructive sleep apnea or other primary sleep disorders, which are prevalent in the kidney transplant population and could independently affect both sleep duration and clinical outcomes. Eighth, in the DCGF models, the events-per-variable ratio was below the conventional threshold of 10, which may have influenced the stability and precision of the adjusted estimates. Nevertheless, the robustness of our findings is reinforced by the consistency of the hazard ratios across multiple models and by the strong correlation between sleep duration and the objective slope of eGFR decline. Finally, given the multicenter nature of this cohort, our findings may have been influenced by centre-level effects, such as differences in institutional protocols or surgical practices.

This prospective study also has multiple strengths. Notably, it is the first study to demonstrate that a short or long sleep duration is independently associated with a higher risk of DCGF among incident KTRs. In real-world clinical practice, transplant clinicians monitor numerous clinical and immunological indicators associated with graft survival rates; however, sleep duration is often overlooked and rarely discussed during evaluations. This study is important because it highlights sleep duration as a potentially modifiable factor that may affect transplant outcomes and HRQOL among KTRs. During transplant clinic visits, in addition to ensuring adherence to immunosuppressant medications, it may also be beneficial to assess sleep duration. Second, this study involved a considerable number of consecutive incident KTRs and included a sufficiently long median follow-up duration. Third, all variables and transplant outcomes were robustly collected due to the prospective nature of this study.

In conclusion, the present study revealed that both shorter and longer sleep durations—relative to a sleep duration of 7 h—are significantly associated with a higher risk of DCGF among incident KTRs, even after controlling for important clinical, immunologic and sociodemographic variables. In particular, short sleep duration was significantly correlated with impaired HRQOL. Our findings support that sleep duration is a potent prognostic indicator. This suggests that screening for sleep disturbances and potential referral to sleep specialists, could be considered as part of an exploratory holistic approach to post-transplant management. There remains a need for larger and longer-term studies to determine whether assessing and intervening in sleep duration may contribute to increased transplant graft survival rates among KTRs.

Author Contributions

Conceptualisation: Hee-Yeon Jung. Methodology: Hee-Yeon Jung and Yu Jin Seo. Formal analysis: Hee-Yeon Jung and Yu Jin Seo. Investigation: Hee-Yeon Jung, Kyu Ha Huh, Jae Berm Park, Myung-Gyu Kim, Kyung Pyo Kang, Seungyeup Han, Jang-Hee Cho, Sun-Hee Park, Chan-Duck Kim, Yong-Lim Kim, Jung-Hwa Ryu and Jaeseok Yang. Data curation: Hee-Yeon Jung, Kyu Ha Huh, Jae Berm Park, Myung-Gyu Kim, Kyung Pyo Kang, Seungyeup Han, Jung-Hwa Ryu and Jaeseok Yang. Funding acquisition: Jaeseok Yang. Writing – original draft: Hee-Yeon Jung. Writing – review and editing: Hee-Yeon Jung. Approval of final manuscript: All authors.

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Ethics Statement

The KNOW-KT study protocol was approved by the institutional review committees of each participating centre (Chonbuk National University Hospital, Gachon University Gil Hospital, Keimyung University Dongsan Medical Center, Korea University Anam Hospital, Kyungpook National University Hospital, Samsung Medical Center, Seoul National University Hospital and Severance Hospital) [50]. All patients provided written informed consent to participate in the study. All clinical investigations were conducted in accordance with the guidelines of the 2008 Declaration of Helsinki and Good Clinical Practice.

Consent

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** Sensitivity analysis for death-censored graft failure (DCGF), according to self-reported sleep duration, restricted to an 8-year follow-up period.

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