

RESEARCH

Open Access



Effects of the pretransplant obesity on kidney transplant outcomes in Korea: a nationwide cohort study

Eun-Young Cho^{1†}, You Hyun Jeon^{1†}, Kyu Ha Huh², Seun Deuk Hwang³, Sangil Min⁴, Jaeseok Yang⁵, Myoung Soo Kim⁶, Yu Jin Seo⁷, Jeong-Hoon Lim¹, Hee-Yeon Jung¹, Ji-Young Choi¹, Sun-Hee Park¹, Yong-Lim Kim¹, Jang-Hee Cho^{1*}, Chan-Duck Kim^{1*} and The Korean Organ Transplantation Registry Study Group

Abstract

Background The prevalence of obesity is steadily increasing in patients with end-stage kidney disease. However, it is still debatable whether obesity affects outcomes after kidney transplantation. This study evaluated the relationship between pretransplant obesity, defined as a body mass index (BMI) ≥ 25 kg/m², and posttransplant outcomes in Korean kidney transplant recipients (KTRs).

Methods We investigated prospective nationwide cohort data from the Korean Organ Transplantation Registry (KOTRY) from 2014 to 2021. KTRs were categorized into 4 groups based on pretransplant BMI: underweight (< 18.5), normal weight (18.5–22.9), overweight (23–24.9), and obesity (≥ 25). Posttransplant outcomes, including death-censored allograft loss, cardiovascular events, and all-cause mortality, were compared using Kaplan-Meier curves with the log-rank test. Cox proportional hazard regression analysis was employed to assess associations between BMI and posttransplant outcomes.

Results A total of 9,130 KTRs were finally analyzed. The mean age was 49.9 ± 11.6 and 60.2% of KTRs were male. The prevalence of obesity in KTRs was 28.6% and continued to increase (24.8% in 2014 to 32.5% in 2021). Obese KTRs were characterized by male predominance, shorter dialysis vintage, and more diabetes as primary kidney disease. Kaplan-Meier curve showed a significant difference in death-censored allograft loss among BMI groups ($P = 0.007$). Obesity (BMI ≥ 25 kg/m²) was as an independent risk factor for death-censored allograft loss (adjusted hazard ratio 1.511, 95% confidence interval 1.063–2.148, $P = 0.021$), but not for cardiovascular events or mortality.

[†]Eun-Young Cho and You Hyun Jeon contributed equally to this work.

The authors of The Korean Organ Transplantation Registry Study Group and their affiliations are provided in the Acknowledgments section.

*Correspondence:

Jang-Hee Cho
jh-cho@knu.ac.kr
Chan-Duck Kim
drckim@knu.ac.kr

Full list of author information is available at the end of the article



© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

Conclusions Our study evaluated BMI across a spectrum of categories, suggesting that obesity is an independent risk factor for graft survival in KTRs. Risk stratification using BMI and strategies to prevent obesity should be considered in the preparation for kidney transplantation.

Clinical trial number Not applicable.

Keywords Body mass index, Graft loss, Kidney transplantation, Obesity

Background

Over the decades, kidney transplantation has demonstrated gradual improvement in graft and patient survival [1]. Nevertheless, some kidney transplant recipients (KTRs) may eventually return to dialysis or face death. Graft loss can result from various causes and risk factors, such as acute or chronic rejection, vascular complications, delayed graft function (DGF), or recipient age [2]. Additionally, the leading cause of recipient mortality was cardiovascular disease, with infection and cancer following behind [1, 3, 4].

Obesity has diverse impacts on kidney transplant outcomes through increased adiposity and adipocytokines-related inflammation [5–7]. Several studies have mentioned associations between obesity and adverse short-term outcomes, especially DGF [8–13]. Furthermore, obesity independently elevates the risk of cardiovascular disease and plays a role in other cardiovascular risk factors such as hypertension and diabetes mellitus [14]. These metabolic diseases can worsen upon post-transplant immunosuppressive therapy and potentially lead to graft damage [8, 15, 16].

The prevalence of obesity has consistently risen in both the general population and those with end-stage kidney disease [17, 18]. Although obesity has its disadvantages, its effect on post-transplant outcomes remains a subject of debate. Some previous meta-analyses have indicated that obesity raises the chance of graft failure and recipient death [7, 9, 19–21]. Other research has suggested a limited significant association between obesity and both graft loss and mortality [10, 22]. These conflicting results suggest the necessity for further investigation into the impact of obesity on kidney transplants.

Ethnic diversity should be considered in evaluating the effect of obesity in KTRs. While the World Health Organization (WHO)'s international classification defines obesity as a body mass index (BMI) ≥ 30 kg/m², guidelines specific to the Asian-Pacific region propose a lower threshold for general Asians, at a BMI ≥ 25 kg/m², based on ethnic variations in body fat ratio and associated health risks [23, 24]. Accordingly, some studies have suggested a BMI ≥ 25 kg/m² as a risk factor for graft loss in Asian KTRs [25–27]. However, research on clinical outcomes in obese Asian KTRs is limited, which may impede appropriate management strategies for obese KTRs.

This study aims to analyze the relationship between the pretransplant obesity (defined as BMI $25 \geq$ kg/m² per Asia-Pacific criteria) and kidney transplant outcomes including death-censored allograft loss, all-cause mortality, and cardiovascular events in Korean KTRs. Secondary analyses were performed across BMI categories to explore broader trends in transplant outcomes by analyzing data from a Korean nationwide study.

Methods

Patient population

This study utilized data from the Korean Organ Transplantation Registry (KOTRY). KOTRY has been collecting prospective transplant data from 59 centers (including 30 centers for kidney) nationwide in Korea since 2014 [28]. All KTRs in the KOTRY database from 2014 to 2021 were included in this study. Recipients under the age of 19 or those having simultaneous multiorgan transplants, other than pancreas-kidney cotransplantation, were not eligible. The detailed design and methodologies of KOTRY have been outlined in a previous article [29].

Data collection

The 9,130 KTRs were accounted for in the analysis. The BMI was assessed using the records available at the time of the kidney transplantation, and if no records were present, the measurement taken closest to the date before the transplantation was utilized. We categorized the subjects into four groups based on their pre-transplant BMI: underweight (< 18.5), normal weight (18.5 – 22.9), overweight (23 – 24.9), and obesity (≥ 25), following the WHO's Asia-Pacific guidelines [24]. BMI was calculated using pre-transplant weight divided by height squared (kg/m²). Baseline demographic characteristics of the recipients, dialysis vintage before transplantation, laboratory data, primary renal disease, comorbidities, transplantation type, immunological variables including pretransplant desensitization, types of immunosuppressants, and the occurrence of clinical outcomes were collected. Data on donor demographics, comorbidities, and donor types were also obtained.

Outcomes

The primary clinical outcomes included death-censored allograft loss, all-cause mortality, and cardiovascular

events. The secondary outcomes were DGF, acute rejection, and de novo donor-specific antibody (DSA). De novo DSA was detected using a Luminex single-antigen bead assay. The definition of death-censored allograft loss included either the onset of dialysis lasting over three months or re-transplantation, with censoring at the time of patient death with a functioning graft. Cardiovascular events included post-transplant cardiovascular death, ischemic heart disease with pertinent evidence, myocardial infarction, newly diagnosed congestive heart failure requiring inpatient care, and arrhythmia. All-cause mortality was defined as death caused by any issue. For patients who passed away with a functioning graft, graft survival was censored at the moment of death. DGF was characterized as the requirement for at least one dialysis in the first seven days after transplants. Acute rejection included clinical rejection and biopsy-confirmed rejection based on the Banff classification. De novo DSA was defined as the detection of antibodies in the recipient's panel reactive antibody (PRA) I and PRA II that matched the donor's human leukocyte antigen (HLA) typing following the transplant.

Statistical analysis

Continuous variables were summarized with mean and standard deviation, while categorical variables were outlined using frequency and percentage. To assess statistical differences, one-way analysis of variance was used for continuous variables, and Pearson's chi-squared test or Fisher's exact test was applied for categorical variables. To evaluate the trend of obesity prevalence among KTRs, linear regression analysis was employed. Differences among sub-BMI groups in death-censored allograft loss, cardiovascular events, and all-cause mortality were assessed using Kaplan-Meier curves and the log-rank test. The associations between BMI and these primary outcomes were assessed through Cox proportional hazard regression analysis, with adjustments for clinically relevant covariates and baseline variables that significantly differed across BMI groups. In the multivariable

Cox proportional hazard regression models, we controlled for confounding variables associated with KTRs and donor baseline characteristics in the following manner: Model 1 was without adjustments; Model 2 was adjusted for age, sex, and BMI in KTRs; Model 3 added donor type into the variables from Model 2; and Model 4 included additional adjustments for the donor's gender, BMI, and cardiovascular disease, recipient's diabetes, hypertension, smoking, dialysis vintage, and acute rejection along with the variables established in Model 3. Subgroup analyses for death-censored allograft loss were carried out based on age, sex, comorbidities, donor type, and desensitization status. To identify factors associated with pretransplant obesity, we performed logistic regression analyses. Variables were selected based on clinical relevance and statistically significant differences between BMI groups in the baseline characteristics. Factors that showed significant associations in univariate analyses ($P < 0.05$) were included in the multivariate model. Statistical analyses were conducted using R (R Foundation for Statistical Computing, Vienna, Austria; www.r-project.org) and SPSS version 22.0 (IBM Corp., Armonk, NY). A P -value < 0.05 was deemed statistically significant.

Results

Figure 1a shows the overall distribution of BMI in 9,130 patients. The prevalence of overweight and obesity was 21.0% and 28.6%, respectively (Fig. 1b). The proportion of obesity gradually increased from 24.8% in 2014 to 32.5% in 2021, showing significant changes over the years (Fig. 1c).

The demographics, categorized by BMI groups, are provided in Table 1. The mean age of all KTRs was 49.9 ± 11.6 years, with a male ratio of 60.2% ($n = 5,499$). Statistically significant differences were identified across BMI groups in recipient characteristics, including age, systolic and diastolic blood pressure, and the number of HLA mismatches. Notably, the obesity group was characterized by a higher prevalence of male sex, more frequent smoking, and shorter duration of dialysis, as well

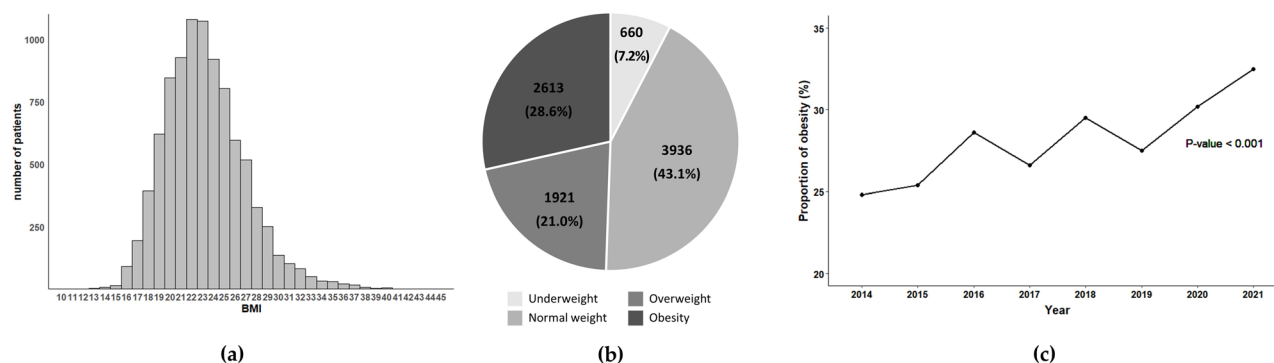


Fig. 1 Distribution and trends of BMI among KTRs. (a) Distribution of BMI; (b) Proportion of BMI categories; (c) annual changes in the proportion of KTRs with obesity. Abbreviations: BMI, body mass index; KTRs, kidney transplant recipients

Table 1 Baseline characteristics

	Underweight (n = 660)	Normal weight (n = 3936)	Overweight (n = 1921)	Obesity (n = 2613)	P-value
Recipient Demographics					
Age (y)	43.0 ± 12.9	49.5 ± 11.6	51.9 ± 10.8	50.6 ± 11.0	< 0.001
Gender (male n, %)	192 (29.1)	2133 (54.2)	1335 (69.5)	1839 (70.4)	< 0.001
Body mass index (kg/m ²)	17.4 ± 1.0	21.0 ± 1.2	23.9 ± 0.6	27.8 ± 2.6	< 0.001
Dialysis vintage before KT (months)	54.3 ± 70.7	57.4 ± 67.9	48.7 ± 57.2	42.9 ± 52.7	< 0.001
Smoking, n (%)	64 (9.8)	743 (19.1)	528 (27.9)	812 (31.8)	< 0.001
Systolic BP (mmHg)	135.2 ± 21.8	139.4 ± 21.0	140.8 ± 20.4	140.0 ± 21.0	< 0.001
Diastolic BP (mmHg)	84.3 ± 13.7	83.6 ± 12.6	83.2 ± 12.4	82.8 ± 12.6	0.026
Fasting glucose (mg/dL)	109.7 ± 39.0	117.9 ± 53.7	125.6 ± 56.2	129.5 ± 60.1	< 0.001
Total cholesterol (mg/dL)	162.3 ± 41.0	153.2 ± 40.9	148.8 ± 41.5	147.8 ± 43.6	< 0.001
LDL (mg/dL)	85.3 ± 29.0	84.3 ± 33.5	83.2 ± 34.2	81.8 ± 35.7	0.056
HDL (mg/dL)	60.1 ± 19.9	52.0 ± 16.5	46.0 ± 15.3	40.5 ± 13.6	< 0.001
Triglyceride (mg/dL)	100.9 ± 54.0	107.7 ± 67.5	126.3 ± 76.7	158.3 ± 103.7	< 0.001
Primary renal disease, n (%)					
Diabetes mellitus	60 (11.8)	749 (23.8)	579 (37.2)	959 (43.8)	< 0.001
Hypertensive nephrosclerosis	80 (15.8)	581 (18.5)	293 (18.8)	424 (19.4)	< 0.001
Glomerulonephritis	288 (56.8)	1407 (44.7)	538 (34.6)	618 (28.2)	< 0.001
Others	79 (15.6)	409 (13.0)	145 (9.3)	190 (8.7)	< 0.001
Comorbid conditions, n (%)					
Hypertension	530 (80.3)	3446 (87.6)	1766 (92.0)	2423 (92.7)	< 0.001
Diabetes mellitus	86 (13.1)	959 (24.4)	718 (37.4)	1187 (45.4)	< 0.001
Cardiovascular disease	43 (6.5)	410 (10.4)	243 (12.7)	402 (15.4)	< 0.001
Transplant-related Variables					
Living donor transplantation, n (%)	484 (73.3)	2566 (65.2)	1240 (64.5)	1830 (70.0)	< 0.001
Deceased donor transplantation, n (%)	176 (26.7)	1370 (34.8)	681 (35.5)	783 (30.0)	< 0.001
Desensitization, n (%)	177 (26.8)	1004 (25.5)	472 (24.6)	628 (24.0)	0.362
ABOi KT, n (%)	121 (18.3)	723 (18.4)	361 (18.8)	478 (18.3)	0.975
HLA mismatch number	3.0 ± 1.7	3.3 ± 1.8	3.3 ± 1.8	3.4 ± 1.8	< 0.001
Basiliximab induction, n (%)	527 (79.8)	3054 (77.6)	1513 (78.8)	2070 (79.3)	0.305
ATG induction, n (%)	143 (21.7)	955 (24.3)	441 (23.0)	564 (21.6)	0.069
Tacrolimus, n (%)	636 (96.4)	3832 (97.4)	1858 (96.8)	2528 (96.9)	0.317
Mycophenolate, n (%)	599 (90.8)	3691 (93.8)	1800 (93.8)	2459 (94.2)	0.012
Corticosteroid, n (%)	649 (98.3)	3878 (98.6)	1886 (98.3)	2542 (97.4)	0.006
Donor Characteristics					
Age (y)	47.4 ± 12.3	48.1 ± 12.6	48.0 ± 13.1	47.9 ± 12.7	0.572
Gender (male n, %)	272 (41.2)	1810 (46.0)	949 (49.4)	1392 (53.3)	< 0.001
Body mass index (kg/m ²)	23.4 ± 3.4	23.8 ± 3.3	24.2 ± 3.5	24.5 ± 3.5	< 0.001
Hypertension, n (%)	105 (16.2)	617 (16.0)	288 (15.3)	447 (17.4)	0.264
Diabetes mellitus, n (%)	18 (2.8)	182 (4.7)	86 (4.6)	129 (5.0)	0.109
Cardiovascular disease, n (%)	9 (1.5)	62 (1.7)	50 (2.8)	37 (1.5)	0.010

Data are presented as mean ± standard deviation or number (%)

Abbreviations: KT, kidney transplantation; BP, blood pressure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; ABOi, ABO incompatible; HLA, human leukocyte antigen; ATG, anti-thymocyte globulin

as higher fasting glucose levels, lower total and high-density lipoprotein cholesterol, and higher triglyceride levels. In this group, diabetes mellitus was the leading cause of primary kidney disease, while hypertension was the most common comorbidity. Most transplants were from living donors, and donor gender and BMI varied significantly across recipient BMI groups.

Patients were followed for a mean duration of 2.5 years with a maximum of 7.4 years. Table 2 summarizes the

occurrence of clinical outcomes in KTRs. Acute rejection rates revealed a significant difference among the BMI groups, with a higher incidence observed in the obesity group (20.3%, $P = 0.031$). The obesity group also exhibited the highest rates of DGF and death-censored allograft loss, though the differences between groups were not statistically meaningful. Cardiovascular events and all-cause mortality rates were similar in the different BMI classifications.

Table 2 Clinical outcomes

	Under-weight (n = 660)	Normal weight (n = 3936)	Over-weight (n = 1921)	Obesity (n = 2613)	P-value
DGF	21 (3.2%)	170 (4.3%)	63 (3.3%)	120 (4.6%)	0.079
Acute rejection	132 (20.0%)	706 (17.9%)	333 (17.3%)	530 (20.3%)	0.031
de novo DSA	61 (9.2%)	375 (9.5%)	217 (11.3%)	239 (9.1%)	0.082
Death-censored allograft loss	18 (2.7%)	101 (2.6%)	55 (2.9%)	96 (3.7%)	0.073
Cardiovascular events	48 (7.3%)	228 (5.8%)	128 (6.7%)	163 (6.2%)	0.371
All-cause mortality	15 (2.3%)	83 (2.1%)	41 (2.1%)	61 (2.3%)	0.935

Data are presented as number (%)

Abbreviations: DGF, delayed graft function; DSA, donor specific antigen

Figure 2 depicts the survival curves of the three clinical outcomes: death-censored allograft loss, cardiovascular events, and all-cause mortality. A significant difference in death-censored allograft loss was found between the groups according to the Kaplan-Meier curve ($P = 0.007$), showing the lowest allograft survival in the obesity KTRs (Fig. 2a, Log-rank $P = 0.007$). No differences were

observed in the cumulative rates of cardiovascular events or all-cause mortality across BMI groups (Fig. 2b and c).

In univariate Cox regression analysis, the obesity group demonstrated a higher risk of death-censored allograft loss relative to the normal weight group (hazard ratio [HR] 1.529, confidence interval [CI] 1.097–2.130, $P = 0.012$). Multivariate analysis confirmed obesity as an independent risk factor of death-censored allograft loss (adjusted hazard ratio [aHR] 1.511, 95% CI 1.063–2.148, $P = 0.021$) (Table 3). The underweight and overweight groups did not show significant changes in the incidence of death-censored allograft loss. However, the obesity group was not linked to the risk of cardiovascular events or all-cause mortality (Tables 4 and 5).

Subgroup analysis verified the influence of demographic factors on death-censored allograft loss in each BMI group (Fig. 3). In the obesity group, death-censored allograft loss was increased in KTRs with age older than 51 (aHR 2.42, CI 1.10–5.34, $P = 0.028$), living donor (aHR 3.38, CI 1.35–8.43, $P = 0.009$), and desensitization (aHR 3.80, CI 1.28–11.33, $P = 0.017$). Obesity in KTRs was linked to a higher hazard of death-censored allograft loss regardless of the presence of hypertension.

Multivariate logistic regression identified male gender, smoking, lower high-density lipoprotein (HDL) levels,

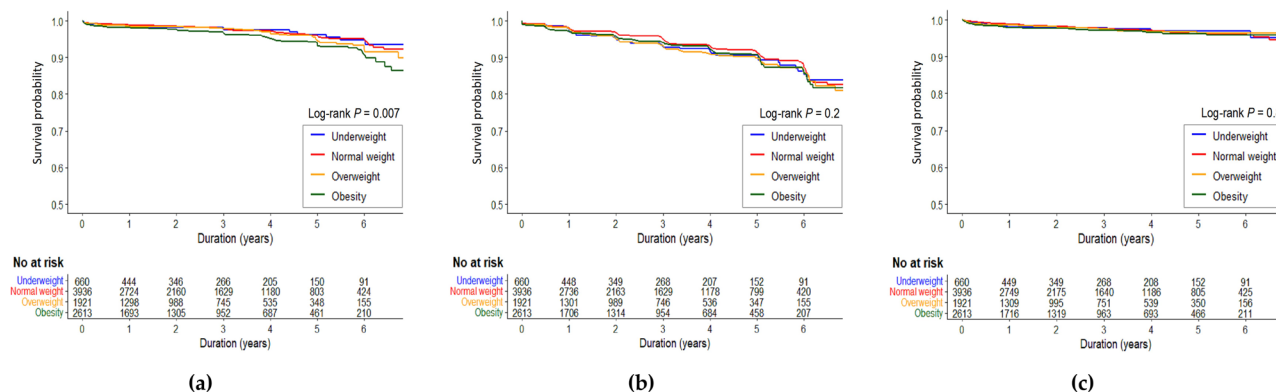


Fig. 2 Kaplan-Meier curves for posttransplant outcomes according to BMI categories. (a) Death-censored allograft loss; (b) Cardiovascular events; (c) All-cause mortality. Abbreviations: BMI, body mass index

Table 3 Cox regression analysis for death-censored allograft loss

	Model 1 ⁽¹⁾		Model 2 ⁽²⁾		Model 3 ⁽³⁾		Model 4 ⁽⁴⁾	
	HR (95% CI)	P-value	aHR (95% CI)	P-value	aHR (95% CI)	P-value	aHR (95% CI)	P-value
Underweight	0.802 (0.401–1.606)	0.534	0.900 (0.446–1.818)	0.769	0.956 (0.473–1.934)	0.901	0.996 (0.490–2.023)	0.990
Normal weight	Reference		Reference		Reference		Reference	
Overweight	1.118 (0.748–1.669)	0.587	1.053 (0.702–1.580)	0.803	1.043 (0.691–1.574)	0.841	1.086 (0.712–1.656)	0.701
Obesity	1.529 (1.097–2.130)	0.012	1.457 (1.041–2.039)	0.028	1.407 (0.996–1.988)	0.053	1.484 (1.039–2.119)	0.030

⁽¹⁾ unadjusted. ⁽²⁾ adjusted for age and sex. ⁽³⁾ adjusted for age, sex, primary renal disease, number of HLA mismatch (ref 0–3), and transplantation type. ⁽⁴⁾ adjusted for age, sex, primary renal disease, diabetes, hypertension, smoking, acute rejection, number of HLA mismatch (ref 0–3), transplantation type, donor gender, donor body mass index and donor cardiovascular disease

Table 4 Cox regression analysis for cardiovascular events

	Model 1 ¹⁾		Model 2 ²⁾		Model 3 ³⁾		Model 4 ⁴⁾	
	HR (95% CI)	P-value	aHR (95% CI)	P-value	aHR (95% CI)	P-value	aHR (95% CI)	P-value
Underweight	1.062 (0.716–1.575)	0.765	1.175 (0.787–1.755)	0.431	1.205 (0.806–1.803)	0.364	1.261 (0.541–1.890)	0.261
Normal weight	Reference		Reference		Reference		Reference	
Overweight	1.212 (0.940–1.561)	0.138	1.148 (0.888–1.485)	0.291	1.141 (0.880–1.480)	0.319	1.166 (0.896–1.516)	0.254
Obesity	1.094 (0.865–1.383)	0.455	1.049 (0.827–1.331)	0.693	1.012 (0.793–1.291)	0.925	0.989 (0.769–1.271)	0.931

⁽¹⁾ unadjusted. ⁽²⁾ adjusted for age and sex. ⁽³⁾ adjusted for age, sex, primary renal disease, number of HLA mismatch (ref 0–3), and transplantation type. ⁽⁴⁾ adjusted for age, sex, primary renal disease, diabetes, hypertension, smoking, acute rejection, number of HLA mismatch (ref 0–3), transplantation type, donor gender, donor body mass index and donor cardiovascular disease

Table 5 Cox regression analysis for all-cause mortality

	Model 1 ¹⁾		Model 2 ²⁾		Model 3 ³⁾		Model 4 ⁴⁾	
	HR (95% CI)	P-value	aHR (95% CI)	P-value	aHR (95% CI)	P-value	aHR (95% CI)	P-value
Underweight	0.934 (0.464–1.881)	0.849	1.450 (0.716–2.937)	0.302	1.540 (0.761–3.119)	0.230	1.355 (0.643–2.853)	0.424
Normal weight	Reference		Reference		Reference		Reference	
Overweight	0.968 (0.619–1.515)	0.888	0.788 (0.502–1.238)	0.302	0.798 (0.508–1.253)	0.327	0.834 (0.530–1.314)	0.434
Obesity	1.246 (0.857–1.811)	0.249	1.130 (0.775–1.647)	0.525	1.115 (0.760–1.636)	0.577	1.142 (0.776–1.683)	0.500

⁽¹⁾ unadjusted. ⁽²⁾ adjusted for age and sex. ⁽³⁾ adjusted for age, sex, primary renal disease, number of HLA mismatch (ref 0–3), and transplantation type. ⁽⁴⁾ adjusted for age, sex, primary renal disease, diabetes, hypertension, smoking, acute rejection, number of HLA mismatch (ref 0–3), transplantation type, donor gender, donor body mass index and donor cardiovascular disease

higher triglycerides, diabetes and cardiovascular disease (all $P < 0.05$) as significant factors associated with pre-transplant obesity (Table 6).

Discussion

This study investigated the relationship between the pre-transplant BMI and post-transplant outcomes in Korean KTRs. Clinical outcomes across different BMI groups were compared based on the rate of death-censored allograft loss, cardiovascular events, and all-cause mortality. Obesity, defined as BMI over 25 kg/m², was related to a higher hazard of allograft failure. The pretransplant BMI did not affect cardiovascular events and mortality.

Obesity is a widely acknowledged independent risk factor with a positive linear relationship to overall mortality and cardiovascular issues in the general population [14, 30]. However, in our study, obesity was not significantly related to death or cardiovascular events in KTRs. The meta-analysis examining BMI in KTRs revealed varying results for these outcomes. Regarding mortality, while some studies have indicated a higher risk of mortality associated with obesity [7, 9, 20], others have found no significant link between BMI and patient survival [10, 11]. Additionally, some studies have reported inconsistencies in mortality results depending on the statistical methods used [9, 19]. Research on the relationship between BMI and cardiovascular events in KTRs is relatively limited

and shows similarly inconsistent results [31, 32]. These inconsistencies in patient mortality and cardiovascular events may be attributed to patient variables such as age, underlying comorbidities, and immune status, which can have a more substantial and complex impact on survival rates and cardiovascular issues, despite the established risk of obesity for cardiovascular disease and general mortality [15].

Regarding allograft outcomes, most meta-analyses have identified obesity as a risk factor for allograft loss [7, 9, 11, 19, 20], which aligns with our results. However, one meta-analysis reported no link between obesity and allograft loss [10]. This study focused on KTRs who underwent kidney transplantation after the 2000s and attributed the lack of association to advancements in transplant medical practices. Our study, based on a nationwide multicenter cohort of over 9,000 KTRs, offers robust statistical power and improved generalizability within an ethnically homogeneous population. In addition, our cohort reflects contemporary transplant practices, including updated immunosuppressive regimens and metabolic management strategies. These contextual improvements may clarify prior conflicting results by controlling for evolving clinical factors.

When considering BMI, it is essential to account for ethnic differences. Compared to Western populations, Asians are inclined to have a greater body fat percentage

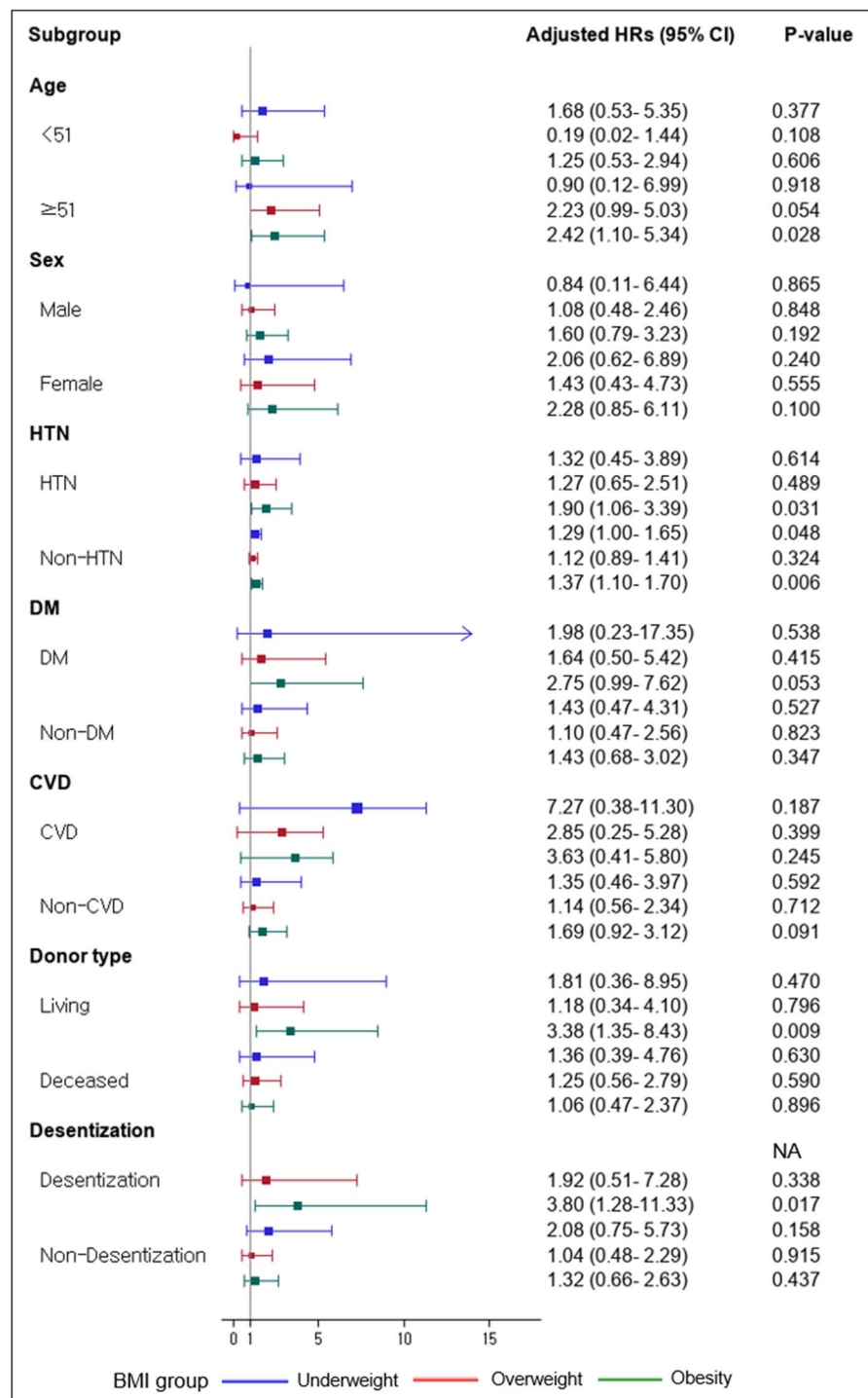


Fig. 3 Subgroup analysis for death-censored allograft loss. Abbreviations: HTN, hypertension; DM, diabetes mellitus; CVD, cardiovascular disease

at the same BMI and are more susceptible to obesity-related health issues even at lower BMI thresholds [23]. Most previous studies on obese KTRs have been conducted in Western countries. Our study reflects ethnic-specific risk stratification by using the Asia-Pacific BMI criterion (≥ 25 kg/m²), which better accounts for differences in body composition and metabolic risk among

Asian populations. Research in China [25], Hong Kong [26], and Japan [27] has consistently shown that obese KTRs with an Asian-specific BMI cutoff of ≥ 25 kg/m² revealed reduced graft survival rates. Some of these studies have reported no statistically significant association with patient survival [25, 27]. The present study also confirmed obesity as an independent factor associated

Table 6 Factors related to pretransplant obesity in logistic regression model

	Univariate			Multivariate		
	B (SE)	β	P-value	B (SE)	β	P-value
Age	−0.91 (0.02)	0.40	< 0.001	−0.003 (0.00)	1.00	0.316
Male gender	0.01 (0.00)	1.01	< 0.001	0.20 (0.08)	1.22	0.017
Dialysis vintage before KT	−0.00 (0.00)	0.99	< 0.001	−0.001 (0.00)	1.00	0.090
Smoking	0.58 (0.05)	1.78	< 0.001	0.31 (0.08)	1.36	< 0.001
Systolic BP	0.001 (0.00)	1.00	0.181			
Diastolic BP	−0.01 (0.00)	1.00	0.015	−0.001 (0.00)	1.00	0.803
Fasting glucose	0.003 (0.00)	1.00	< 0.001	−0.001 (0.00)	1.00	0.052
Total cholesterol	−0.003 (0.00)	1.00	< 0.001	0.001 (0.00)	1.00	0.268
HDL	−0.05 (0.00)	0.95	< 0.001	−0.04 (0.00)	0.97	< 0.001
Triglyceride	0.01 (0.00)	1.01	< 0.001	0.004 (0.00)	1.00	< 0.001
Hypertension	0.54 (0.09)	1.72	< 0.001	0.21 (0.13)	1.23	0.103
Diabetes mellitus	0.81 (0.05)	2.24	< 0.001	0.54 (0.08)	1.72	< 0.001
Cardiovascular disease	0.42 (0.07)	1.52	< 0.001	0.31 (0.10)	1.36	0.002

Abbreviations: B, unstandardized regression coefficient; SE, standard error; β , standardized coefficient; KT, kidney transplantation; BP, blood pressure; HDL, high-density lipoprotein

with death-censored allograft loss, consistent with several prior studies, including a meta-analysis [11] and an analysis of Asian populations [25].

The pathogenic impact of obesity on transplanted organs can be explained through multiple mechanisms. The most extensively researched mechanism is the metabolic disturbances associated with obesity. Obesity-related metabolic issues, such as insulin resistance, dyslipidemia, hypertension, and chronic inflammation, are recognized risk factors for allograft damage due to their hemodynamic and proinflammatory changes [33–35]. Our data indicate that the obese group, which showed a higher risk of death-censored allograft loss, has elevated fasting glucose levels and abnormal lipid profiles, in addition to a greater prevalence of hypertension and diabetes, in contrast to the other BMI groups. These findings support the association between abnormal metabolic conditions and allograft failure in the KTRs.

Several studies have linked obesity to acute rejection [1, 9, 27] and DGF [1, 7, 9–12, 21], both of which heighten the risk of allograft failure [1, 7]. One possibility is that obesity-related inflammation may exacerbate graft rejection pathways. Obesity is reported to be associated with alloimmune dysregulation through elevated leptin and lowered adiponectin levels, activating both innate and adaptive immunity [5, 6]. This association can be supported by experimental studies in obese mice with heart transplants [36, 37]. Additionally, recent clinical research has reported a link between hyperleptinemia and the production of de novo DSA [38], which can contribute to allograft rejection. However, the risk of death-censored allograft loss remained higher in obese recipients, even after adjusting for acute rejection, highlighting a potential independent effect of obesity on graft failure that warrants further investigation. This suggests that obesity may affect graft survival not only through immunologic

pathways but also via non-immunologic mechanisms. These findings underscore the multifactorial impact of obesity on graft outcomes and support the need for comprehensive perioperative management in obese KTRs. Given this association between pretransplant obesity and adverse graft outcomes, efforts to optimize body weight prior to kidney transplantation may be beneficial.

In this context, it is important to recognize that obesity is often accompanied by a cluster of modifiable cardiometabolic risk factors that may collectively influence transplant outcomes. Our multivariate logistic regression analysis suggests that pretransplant obesity is not an isolated phenomenon, but is closely linked to components of metabolic syndrome. These include modifiable risk factors such as smoking, dyslipidemia, diabetes, and cardiovascular disease, all of which warrant comprehensive clinical assessment. Clinicians should consider metabolic risk screening and targeted interventions to optimize pretransplant health and improve posttransplant outcomes in obese transplant candidates.

Another finding from our study was that the impact of obesity on death-censored allograft loss was modified by old age over 51 years, desensitization, and living donors. The elevated risk persisted regardless of hypertension, which may suggest obesity's independent role, possibly through immunometabolic mechanisms. Aging can contribute to allograft loss through altered metabolic and immunologic factors [39, 40], which may be compounded by obesity. The altered inflammatory state in obesity may also explain the poor allograft outcome observed in the high BMI, HLA-presensitized recipients [41]. Additionally, lower graft survival rates have been reported in obese KTRs who received living donor transplants in previous studies [42, 43]. These findings suggest that obese recipients of living donor kidneys may benefit from intensified immunologic monitoring or tailored immunosuppressive

strategies, even in settings traditionally considered lower-risk. However, further research may be warranted to explore the significant impact of obesity on allograft loss in living donor groups, given the similar immunologic barriers between living and deceased donor transplants [44]. In summary, our subgroup analysis highlights the need for tailored management strategies for obese KTRs, especially those with specific risk factors.

There are several limitations to this study. First, the number of patients with extreme obesity is relatively small, and the average follow-up period was short. Post-hoc power analyses indicated that these comparisons were underpowered (power < 40%), likely due to low event rates and small subgroup sizes. Thus, the absence of significant associations should be interpreted with caution. The limited follow-up duration may have restricted our ability to assess long-term outcomes, and the impacts based on the obesity grade could not be compared to the studies of Western patients. Second, discrepancies in our findings may arise from ethnic differences, as Asians have different body fat profiles and obese-related disease rates than Western populations. Accordingly, we used the Asia-Pacific BMI criterion (≥ 25 kg/m²), which more accurately reflects obesity-related risk in Asian populations [23]. However, this ethnic-specific definition may limit the generalizability of our findings to non-Asian populations, where obesity is typically defined using the WHO cutoff of ≥ 30 kg/m². Third, the explanation supporting our results remains ambiguous, as the mechanisms through which obesity impacts kidney transplant outcomes are not yet clearly defined. The lack of data on immunosuppressive drug dosages and trough levels prevented direct assessment of whether obesity-related graft loss was mediated by suboptimal immunosuppression. Sustained data collection and further clinical and experimental research are required to gain a clearer understanding of how obesity affects kidney transplants. Next, BMI is a highly confounded variable that may reflect various underlying clinical and behavioral factors, including nutritional status, inflammation, and comorbidities. Although we adjusted for multiple covariates in our Cox regression models, the possibility of residual or unmeasured confounding cannot be excluded. In addition, post-transplant BMI data were not available in the KOTRY dataset, which precluded us from assessing the duration or persistence of obesity after transplantation. This limits our ability to distinguish between transient and sustained obesity, which may have differing impacts on long-term transplant outcomes.

Conclusion

Our research with the nationwide data from Korea identifies obesity as an independent risk factor for death-censored allograft loss using Asian BMI cut-offs.

Pretransplant BMI showed no association with cardiovascular events or mortality rates. The risk of death-censored allograft loss is influenced by specific patient characteristics including age, desensitization, and donor type. These findings highlight the importance of assessing BMI-related risks and implement personalized management strategies for patients considering kidney transplantation.

Abbreviations

ABOi	ABO incompatible
aHR	Adjusted hazard ratio
BMI	Body mass index
BP	Blood pressure
CI	Confidence interval
CVD	Cardiovascular disease
DGF	Delayed graft function
DM	Diabetes mellitus
DSA	Donor specific antigen
HDL	High-density lipoprotein
HLA	Human leukocyte antigen
HR	Hazard ratio
HTN	Hypertension
KOTRY	Korean Organ Transplantation Registry
KT	Kidney transplantation
KTRs	Kidney transplant recipients
LDL	Low-density lipoprotein
PRA	Panel reactive antibody
WHO	World Health Organization

Acknowledgements

The Korean Organ Transplantation Registry Study Group: Jae Berm Park¹, Jung Hwan Park², Su Hyung Lee³, Ji Yoon Choi⁴, Cheol Woong Jung⁵, Jun Young Lee⁶, Yeong Hoon Kim⁷, Joong Kyung Kim⁸, Eun Jeoung Ko⁹, Sik Lee¹⁰, Yeon Ho Park¹¹, Seok Hui Kang¹², Tae Hyun Ban¹³, Sang Heon Song¹⁴, Seung Hwan Song¹⁵, Ho Sik Shin¹⁶, Byung Ha Chung¹⁷, Hye Eun Yoon¹⁸, Ki-Ryang Na¹⁹, Dong Ryeol Lee²⁰, Dong Won Lee²¹, Jieun Oh²², Su Woong Jung²³, Yu Ho Lee²⁴, Hyejin Mo²⁵, Jeong-Hoon Lee²⁶, Jin Seok Jeon²⁷, Sang Youb Han²⁸, Jin Sug Kim²⁹, Jong Soo Lee³⁰, Man Ki Ju³¹, Jong Cheol Jeong³², Soo Jin Na Choi³³, Sung Shin³⁴, Seungyeup Han³⁵, Young Soo Chung³⁶, Young Joo Kwon³⁷. ¹Department of Surgery, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea; ²Department of Nephrology, Konkuk University School of Medicine, Seoul, Republic of Korea; ³Department of Surgery, Ajou University School of Medicine, Suwon, Republic of Korea; ⁴Department of Surgery, College of Medicine, Han Yang University, Seoul, Republic of Korea; ⁵Department of Surgery, Korea University Anam Hospital, Seoul, Republic of Korea; ⁶Department of Nephrology, Yonsei University Wonju College of Medicine, Wonju Severance Christian Hospital, Wonju, Republic of Korea; ⁷Department of Internal Medicine, Inje University Busan Paik Hospital, Busan, Republic of Korea; ⁸Department of Internal Medicine, Bongseng Memorial Hospital, Busan, Republic of Korea; ⁹Division of Nephrology, Department of Internal Medicine, Bucheon St. Mary's Hospital, Seoul, Republic of Korea; ¹⁰Department of Internal Medicine, Jeonbuk National University Hospital, Jeonju, Republic of Korea; ¹¹Department of Surgery, Gil Medical Center, Gachon University College of Medicine, Incheon, Republic of Korea; ¹²Department of Nephrology, Yeungnam University Hospital, Daegu, Republic of Korea; ¹³Division of Nephrology, Department of Internal Medicine, Eunpyeong St. Mary's hospital, Seoul, Republic of Korea; ¹⁴Department of Internal Medicine, Pusan National University Hospital, Busan, Republic of Korea; ¹⁵Department of Surgery, Ewha Womans University Seoul Hospital, Seoul, Republic of Korea; ¹⁶Department of Internal Medicine, Division of Nephrology, Kosin University College of Medicine, Busan, Republic of Korea; ¹⁷Division of Nephrology, Department of Internal Medicine, Seoul St. Mary's hospital, Seoul, Republic of Korea; ¹⁸Department of Internal Medicine, Incheon St. Mary's Hospital, College of Medicine, The Catholic University of Korea College of Medicine, Incheon, Republic of Korea; ¹⁹Department of Nephrology, Chungnam National University Hospital, Daejeon, Republic of Korea; ²⁰Division of Nephrology, Department of Internal Medicine, Maryknoll Medical Center, Busan, Republic of Korea; ²¹Division of Nephrology, Department of Internal

Medicine, Pusan National University School of Medicine, Busan, Republic of Korea; ²²Department of Internal Medicine, Kangdong Sacred Heart Hospital, Hallym University College of Medicine, Seoul, Republic of Korea; ²³Division of Nephrology, Department of Internal Medicine, College of Medicine, Kyung Hee University, Seoul, Republic of Korea; ²⁴Division of Nephrology, Department of Internal Medicine, CHA Bundang Medical Center, CHA University, Seongnam, Republic of Korea; ²⁵Department of Surgery, SMG-SNU Boramae Medical Center, Seoul, Republic of Korea; ²⁶Department of Surgery, Myongji Hospital, Goyang, Republic of Korea; ²⁷Department of Internal Medicine, Soonchunhyang University Seoul Hospital, Seoul, Republic of Korea; ²⁸Division of Nephrology, Inje University Ilsan-Paik Hospital, Goyang, Republic of Korea; ²⁹Division of Nephrology, Department of Internal Medicine, College of Medicine, Kyung Hee University Hospital, Kyung Hee University, Seoul, Republic of Korea; ³⁰Department of Internal Medicine, Ulsan University Hospital, Ulsan, Republic of Korea; ³¹Department of Surgery, Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea; ³²Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul, Republic of Korea; ³³Department of Surgery, Chonnam National University Medical School, Gwangju, Republic of Korea; ³⁴Department of Surgery, Asan Medical Center, Seoul, Republic of Korea; ³⁵Department of Internal Medicine, Keimyung University School of Medicine, Daegu, Republic of Korea; ³⁶Department of Surgery, Dong-A University College of Medicine, Busan, Republic of Korea; ³⁷Division of Nephrology, Korea University, College of Medicine, Guro Hospital, Seoul, Republic of Korea.

Author contributions

Conceptualization, J.-H.C. and C.-D.K.; methodology, Y.H.J. and J.-H.L.; formal analysis, Y.J.S.; investigation, Y.H.J.; data curation, Y.J.S.; writing—original draft preparation, E.-Y.C.; writing—review and editing, Y.H.J., J.-H.C. and C.-D.K.; supervision, K.H.H., S.D.H., S.M., H.-Y.J., J.-Y.C., S.-H.P., and Y.-L.K.; funding acquisition, J.Y. and M.S.K. All authors have read and approved the final manuscript.

Funding

This research was supported by the "National Institute of Health"(NIH) research project (2014-ER6301-00, 2014-ER6301-01, 2014-ER6301-02, 2017-ER6301-00, 2017-ER6301-01, 2017-ER6301-02, 2020-ER7201-00, 2020-ER7201-01, 2020-ER7201-02) and by a grant of the Korea Health Technology R&D Project through the Korea Health Industry Development Institute(KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (grant number: RS-2022-KH130593 and RS-2025-02303987).

Data availability

The data of this study are available from the corresponding author on reasonable request. The data are not publicly available as they contain information that could compromise the privacy of research participants, and data sharing is subject to ethical and registry constraints in accordance with KOTRY policies.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of the Kyungpook National University Hospital (2020-11-056). Informed consent was obtained from all subjects involved in the study.

Ethics Statement

All procedures involving human participants were conducted in accordance with the 2008 Declaration of Istanbul on Organ Trafficking and Transplant Tourism.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

¹Department of Internal Medicine, School of Medicine, Kyungpook National University, Daegu, Korea

²Department of Transplantation Surgery, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea

³Department of Internal Medicine of Nephrology, Inha University Hospital, The Inha University of Korea, College of Medicine, Incheon, Korea

⁴Department of Surgery, Seoul National University Hospital, Seoul, Korea

⁵Division of Nephrology, Department of Internal Medicine, Yonsei University College of Medicine, Severance Hospital, Seoul, Korea

⁶Department of Surgery, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea

⁷Department of Statistics, Kyungpook National University, Daegu, Korea

Received: 7 February 2025 / Accepted: 22 July 2025

Published online: 29 July 2025

References

1. Hariharan S, Israni AK, Danovitch G. Long-Term survival after kidney transplantation. *N Engl J Med*. 2021;385(8):729–43.
2. Foroutan F, Friesen EL, Clark KE, Motaghi S, Zyla R, Lee Y, Kamran R, Ali E, De Snoo M, Orchanian-Cheff A, et al. Risk factors for 1-Year graft loss after kidney transplantation: systematic review and Meta-Analysis. *Clin J Am Soc Nephrol*. 2019;14(11):1642–50.
3. Kim C-D. Kidney transplantation. *Korean J Med*. 2014;86(2):142–51.
4. Awan AA, Niu J, Pan JS, Erickson KF, Mandayam S, Winkelmayer WC, Navaneethan SD, Ramanathan V. Trends in the causes of death among kidney transplant recipients in the united States (1996–2014). *Am J Nephrol*. 2018;48(6):472–81.
5. Heinbokel T, Floerchinger B, Schmiderer A, Edtinger K, Liu G, Elkhali A, Tullius SG. Obesity and its impact on transplantation and alloimmunity. *Transplantation*. 2013;96(1):10–6.
6. Wu D, Dawson NAJ, Levings MK. Obesity-Associated adipose tissue inflammation and transplantation. *Am J Transpl*. 2016;16(3):743–50.
7. Chan WM, Bosch JAP, Jones DP, McTernan PGP, Phillips ACP, Borrow RMA. Obesity in kidney transplantation. *J Ren Nutr*. 2014;24(1):1–12.
8. Jindal RM, Zawada ET. Obesity and kidney transplantation. *Am J Kidney Dis*. 2004;43(6):943–52.
9. Lafranca JA, Ijermans JNM, Betjes MGH, Dor FJMF. Body mass index and outcome in renal transplant recipients: a systematic review and meta-analysis. *BMC Med*. 2015;13(1):111–111.
10. Nicoletto BB, Fonseca NKO, Manfro RC, Gonçalves LFS, Leitão CB, Souza GC. Effects of obesity on kidney transplantation outcomes: A systematic review and Meta-Analysis. *Transplantation*. 2014;98(2):167–76.
11. Hill CJ, Courtney AE, Cardwell CR, Maxwell AP, Lucarelli G, Veroux M, Furriel F, Cannon RM, Hoogeveen EK, Doshi M, et al. Recipient obesity and outcomes after kidney transplantation: a systematic review and meta-analysis. *Nephrol Dial Transpl*. 2015;30(8):1403–11.
12. Fellmann M, Balssa L, Clément E, Frey P, Frontczak A, Bernardini S, Chabannes E, Guichard G, Bittard H, Kleinclauss F. Effects of obesity on postoperative complications and graft survival after kidney transplantation. *Transpl Proc*. 2020;52(10):3153–9.
13. Kim J-M, Kwon HE, Ko Y, Jung JH, Kwon H, Kim YH, Kim EK, Shin S. Robot-assisted kidney transplantation in a morbidly obese patient with incisional hernia reconstruction and abdominoplasty: a case report. *In*. 2023;37:210–5.
14. Powell-Wiley TM, Poirier P, Burke LE, Després J-P, Gordon-Larsen P, Lavie CJ, Lear SA, Ndumele CE, Neeland IJ, Sanders P. Obesity and cardiovascular disease: a scientific statement from the American heart association. *Circulation*. 2021;143(21):e984–1010.
15. Gill JS, Abichandani R, Kausz AT, Pereira BJG. Mortality after kidney transplant failure: the impact of non-immunologic factors. *Kidney Int*. 2002;62(5):1875–83.
16. Pischon T, Sharma AM. Obesity as a risk factor in renal transplant patients. *Nephrol Dial Transpl*. 2001;16(1):14–7.
17. Shelton BA, Reed RD, MacLennan PA, McWilliams D, Mustian MN, Sawinski D, Kumar V, Ong S, Locke JE. Increasing obesity prevalence in the united States End-Stage renal disease population. *J Health Sci Educ* 2018, 2(5).
18. Yang YS, Han B-D, Han K, Jung J-H, Son JW. Taskforce team of the obesity fact sheet of the Korean society for the study of O: obesity fact sheet in Korea, 2021: trends in obesity prevalence and obesity-Related comorbidity incidence stratified by age from 2009 to 2019. *J Obes Metabolic Syndrome*. 2022;31(2):169–77.

19. Yin SF, Wu LY, Huang ZL, Fan Y, Lin T, Song TR. Nonlinear relationship between body mass index and clinical outcomes after kidney transplantation: A dose-response meta-analysis of 50 observational studies. *Surgery*. 2022;171(5):1396–405.
20. Ahmadi SF, Zahmatkesh G, Streja E, Molnar MZ, Rhee CM, Kovesdy CP, Gillen DL, Steiner S, Kalantar-Zadeh K. Body mass index and mortality in kidney transplant recipients: A systematic review and Meta-Analysis. *Am J Nephrol*. 2014;40(4):315–24.
21. Prudhomme T, Bento L, Frontczak A, Timsit M-O, Boissier R. Effect of recipient body mass index on kidney transplantation outcomes: A systematic review and Meta-analysis by the transplant committee from the French association of urology. *European urology focus*; 2023.
22. Järvi L, Pechter Ü, Kuudeberg A, Lember M, Ots-Rosenberg M. Effect of pretransplant body mass index on kidney transplant recipient and graft Long-term survival. *Transpl Proc*. 2021;53(10):2879–87.
23. Barba C, Cavalli-Sforza T, Cutter J, Darnton-Hill I, consultation WHOE, Consultation WHOE. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet (British edition)*. 2004;363(9403):157–63.
24. Organization WH. The Asia-Pacific perspective: redefining obesity and its treatment. 2000.
25. Chow KM, Szeto CC, Leung CB, Lui SF, Tong YF, Li PK-T. Body mass index as a predictive factor for long-term renal transplant outcomes in Asians. *Clin Transplant*. 2006;20(5):582–9.
26. Cheung CY, Chan YH, Chan HW, Chau KF, Li CS. Optimal body mass index that can predict long-term graft outcome in Asian renal transplant recipients. *Nephrology*. 2010;15(2):259–65.
27. Ishikawa S, Tasaki M, Ikeda M, Nakagawa Y, Saito K, Tomita Y. Pretransplant BMI should be < 25 in Japanese kidney transplant recipients: A Single-Center experience. *Transpl Proc*. 2023;55(1):72–9.
28. Ahn C, Koo TY, Jeong JC, Kim M, Yang J, Lee J, Min SI, Lee JE, Kim MS, Kwon OJ et al. Initial Report of the Korean Organ Transplant Registry: The First Report of National Kidney Transplantation Data. *Transplantation proceedings* 2014, 46(2):425–430.
29. Yang J, Jeong JC, Lee J, Kim YH, Paik HC, Kim J-J, Park H-Y, Kim MS, Ahn C. Design and methods of the Korean organ transplantation registry. *Transplantation Direct*. 2017;3(8):e191–191.
30. Di Angelantonio E, Bhupathiraju SN, Wormser D, Gao P, Kaptoge S, de Gonzalez AB, Cairns BJ, Huxley R, Jackson CL, Joshy G, et al. Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. *Lancet (British edition)*. 2016;388(10046):776–86.
31. Lentine KL, Rocca-Rey LA, Bacchi G, Wasi N, Schmitz L, Salvalaggio PR, Abbott KC, Schnitzler MA, Neri L, Brennan DC. Obesity and cardiac risk after kidney transplantation: experience at one center and comprehensive literature review. *Transplantation*. 2008;86(2):303–12.
32. Ladhani M, Craig JC, Irving M, Clayton PA, Wong G. Obesity and the risk of cardiovascular and all-cause mortality in chronic kidney disease: a systematic review and meta-analysis. *Nephrol Dial Transplant*. 2017;32(3):439–49.
33. Czaja-Stolc S, Potrykus M, Stankiewicz M, Kaska Ł, Małgorzewicz S. Pro-Inflammatory profile of adipokines in obesity contributes to pathogenesis, nutritional disorders, and cardiovascular risk in chronic kidney disease. *Nutrients*. 2022;14(7):1457.
34. Wolf G, Chen S, Han DC, Ziyadeh FN. Leptin and renal disease. *Am J Kidney Dis*. 2002;39(1):1–11.
35. Martínez-Montoro JI, Morales E, Cornejo-Pareja I, Tinahones FJ, Fernández-García JC. Obesity-related glomerulopathy: current approaches and future perspectives. *Obes Rev*. 2022;23(7):e13450. -n/a.
36. Molinero LL, Yin D, Lei YM, Chen L, Wang Y, Chong AS, Alegre M-L. High-Fat Diet-Induced obesity enhances allograft rejection. *Transplantation*. 2016;100(5):1015–21.
37. Okamoto Y, Christen T, Shimizu K, Asano K, Kihara S, Mitchell RN, Libby P. Adiponectin inhibits allograft rejection in murine cardiac transplantation. *Transplantation*. 2009;88(7):879–83.
38. Dedinská I, Kleinová P, Machálek K, Graňák K, Vnučák M, Beliančinová M. The role of hyperleptinaemia and low values of Interleukin 10 in de Novo DSA production after kidney transplantation. *Transpl Immunol*. 2024;83:101982–101982.
39. Meier-Kriesche HU, Ojo AO, Cibrik DM, Hanson JA, Leichtman AB, Magee JC, Port FK, Kaplan B. Relationship of recipient age and development of chronic allograft failure. *Transplantation*. 2000;70(2):306–10.
40. Peeters LEJ, Andrews LM, Hesselink DA, de Winter BCM, van Gelder T. Personalized immunosuppression in elderly renal transplant recipients. *Pharmacol Res*. 2018;130:303–7.
41. Park Y, Lee H, Ko EJ, Lee S, Ban TH, Min J-w, Yoon H-e, Oh E-j, Yang CW, Chung BH. Impact of high body mass index on allograft outcomes in kidney transplant recipients with presensitization to human leukocyte antigen. *Kidney Res Clin Pract*. 2021;40(2):304–16.
42. Schwarznau A, Matevosian E, Novotny A, Stangl M. Outcome of living donor renal transplantation in obese recipients. *Transpl Proc*. 2008;40(4):921–2.
43. Yemini R, Rahamimov R, Neshet E, Anteby R, Ghinea R, Hod T, Mor E. The impact of obesity and associated comorbidities on the outcomes after renal transplantation with a living donor vs. Deceased donor grafts. *J Clin Med*. 2022;11(11):3069.
44. Najarian JS, Gillingham KJ, Sutherland DER, Reinsmoen NL, Payne WD, Matas AJ. The impact of the quality of initial graft function on cadaver kidney transplants. In: 1994; *Hagerstown, MD*: Lippincott: 812–6.

Publisher's note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.