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Kidney Transplant Outcomes in Recipients With Congenital Anomalies of the Kidney and Urinary Tract: A Single-Center Propensity Score-Matched Study

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Research Article

Keywords: congenital anomalies of the kidney and urinary tract, kidney transplantation, graft survival, urinary tract infection, lower urinary tract dysfunction

Posted Date: November 1st, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10607260/v1

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Additional Declarations: No competing interests reported.

Abstract

Objective

Congenital anomalies of the kidney and urinary tract (CAKUT) are a major cause of kidney failure in children and young adults, but the extent to which persistent urinary tract abnormalities influence long-term kidney transplant outcomes remains uncertain. We aimed to compare graft, patient, infectious, and urological outcomes between kidney transplant recipients with CAKUT and propensity score-matched recipients with non-CAKUT causes of kidney failure.

Materials & Methods

We retrospectively evaluated kidney transplantations performed at a single center between January 2010 and August 2025. The primary outcome was death-censored graft failure; secondary outcomes included overall graft and patient survival, delayed graft function, biopsy-proven acute rejection, major surgical complications, symptomatic and recurrent urinary tract infection (UTI), graft pyelonephritis, and allograft function.

Results

Delayed graft function, acute rejection, and major surgical complication rates were similar between groups. Symptomatic UTI (27.5% vs 13.8%; p = 0.005) and recurrent UTI (12.3% vs 5.1%; p = 0.033) were more frequent in CAKUT recipients, whereas the difference in graft pyelonephritis did not reach statistical significance (10.9% vs 5.1%; p = 0.075). Ten-year death-censored graft survival was higher in the CAKUT group (88.3% vs 76.9%; log-rank p = 0.018), while overall graft survival (82.4% vs 73.8%; p = 0.071) and patient survival (93.5% vs 92.7%; p = 0.740) were not significantly different. CAKUT remained associated with a lower adjusted risk of death-censored graft failure (HR 0.55; 95% CI 0.29–0.99; p = 0.048).

Conclusion

Recipients with CAKUT experienced a greater burden of posttransplant UTI but achieved similar patient and overall graft survival and superior death-censored graft survival compared with matched non-CAKUT recipients, supporting kidney transplantation after individualized urological assessment and sustained lower urinary tract surveillance.

INTRODUCTION

Congenital anomalies of the kidney and urinary tract (CAKUT) comprise a heterogeneous spectrum of developmental disorders affecting the renal parenchyma, collecting system, ureters, bladder, and urethra,

and they remain the leading cause of chronic kidney disease and kidney failure in children worldwide [1–3]. Because the natural history of CAKUT is highly variable, many patients progress to kidney failure in childhood, whereas a substantial proportion reach end-stage kidney disease only in adolescence or adulthood, making CAKUT a life-course disorder rather than an exclusively pediatric condition [1–3]. Kidney transplantation (KT) is the preferred renal replacement modality for these patients, and CAKUT accounts for a large share of pediatric transplant recipients and a clinically important subset of young adult recipients with kidney failure [2, 4, 5].

KT in CAKUT is not a uniform clinical entity, because the post-transplant course may be shaped by the underlying phenotype, the presence of lower urinary tract dysfunction (LUTD), prior reconstructive surgery, residual native urinary tract abnormalities, and the need for multidisciplinary pretransplant urologic optimization [4, 5]. Persistent bladder dysfunction, impaired compliance, incomplete emptying, urinary diversion, prior augmentation cystoplasty, vesicoureteral reflux, and recurrent urinary tract infection (UTI) may expose the allograft to elevated storage pressures, obstruction, pyelonephritis, repeated urological interventions, and late graft injury even when short-term outcomes appear acceptable [4–10]. Consequently, the principal clinical controversy is whether CAKUT itself, or selected high-risk lower urinary tract phenotypes within the CAKUT spectrum, adversely affects graft function and long-term graft survival despite contemporary pretransplant evaluation and postoperative management [6, 8–15].

Available evidence indicates that overall graft outcomes after transplantation for CAKUT are at least comparable with those observed in recipients with non-CAKUT kidney disease, although UTIs are consistently more frequent [8, 11–15]. In an Australian and New Zealand registry cohort of 1,099 recipients with posterior urethral valves, renal hypoplasia/dysplasia, or reflux nephropathy, 10-year death-censored graft survival ranged from 70% to 76%, but posterior urethral valves were associated with increased graft loss beyond 10 years [8]. A United States Renal Data System analysis of more than 10,000 first kidney transplants reported better death-censored graft survival in CAKUT recipients than in matched non-CAKUT controls, with the benefit persisting in both pediatric and adult strata [11]. Similarly, a Turkish single-center propensity score-matched study found superior 10-year graft survival in CAKUT recipients, despite a significantly higher frequency of posttransplant UTI [12]. Pediatric cohorts have also reported similar patient survival and generally superior or equivalent graft survival in CAKUT compared with non-CAKUT disease, although differences in disease recurrence among control groups may partly explain the apparent graft-survival advantage [13, 14].

The effect of specific urological phenotypes remains less certain because many studies are small, pediatric-only, diagnosis-specific, or lack adequately matched controls [6, 8–10, 14, 15]. Recent data suggest that reflux into the transplanted kidney does not necessarily reduce medium-term graft or patient survival, whereas posterior urethral valve cohorts and adult congenital lower urinary tract malformation series show that recurrent infection and bladder-management complexity may increase morbidity without uniformly causing graft failure [6, 9, 10, 15]. These findings support multidisciplinary

assessment but do not identify which CAKUT recipients remain at clinically meaningful excess risk after transplantation [4, 6, 9, 10, 15].

Evidence is particularly limited for contemporary single-center cohorts that include predominantly adult recipients together with a smaller pediatric population, provide detailed urological phenotyping, and compare outcomes with non-CAKUT recipients while addressing baseline treatment-selection differences [8, 11–13, 16]. Therefore, we aimed to evaluate graft survival, infectious and urological outcomes among KT recipients with CAKUT treated at a single center between January 2010 and August 2025 and to compare them with propensity score-matched recipients transplanted for non-CAKUT causes of kidney failure.

MATERIALS AND METHODS

After institutional review board approval, data of patients who underwent KT between January 2010 and August 2025 were evaluated. Clinical data were obtained from hospital electronic medical records and patient charts. KT recipients were excluded if they underwent multiorgan transplantation, lost their graft within 30 days post-transplant, patients with incomplete data. Demographic characteristics, primary cause of kidney failure, dialysis modality and duration, preemptive transplantation, previous kidney transplantation, panel-reactive antibody (PRA) status, HLA mismatch number, living or deceased donor status and post-transplant follow-up were reviewed.

CAKUT phenotypes included renal agenesis or hypoplasia/dysplasia, multicystic dysplastic kidney, vesicoureteral reflux or reflux nephropathy, ureteropelvic or ureterovesical junction obstruction, posterior urethral valves, prune belly syndrome, neurogenic or non-neurogenic LUTD associated with a congenital disorder and bladder exstrophy. Non-CAKUT causes of kidney failure included primary glomerular disease, tubulointerstitial disease, hereditary non-CAKUT disorders, diabetic kidney disease, hypertensive kidney disease, and kidney failure of other established acquired etiologies. CAKUT-specific variables included the dominant anatomical diagnosis, upper versus lower urinary tract involvement, history of recurrent UTI, clean intermittent catheterization, and other pretransplant reconstructive procedures.

The primary outcome was death-censored graft failure, defined as return to permanent dialysis, retransplantation, or transplant nephrectomy for irreversible graft dysfunction. Secondary outcomes were overall graft survival, patient survival, delayed graft function (DGF), biopsy-proven acute rejection, postoperative surgical or urological complications, symptomatic UTI, recurrent UTI, graft pyelonephritis, and allograft function.

Immunosuppressive therapy for both groups consisted of the same induction therapy and triple maintenance agents. Induction therapy included anti-thymocyte globulin. Routine maintenance therapy consisted of tacrolimus, mycophenolate mofetil, and steroids. DGF was defined as the requirement for dialysis within the first 7 days after KT. Surgical and urological complications were graded using the Clavien-Dindo classification, and complications of grade 3 or higher were considered major [17]. A symptomatic UTI was defined as compatible urinary or systemic symptoms with pyuria and a positive

urine culture. Recurrent UTI was defined as at least two episodes within 6 months or at least three episodes within 12 months. Graft pyelonephritis was defined as a febrile UTI with graft pain, systemic inflammatory findings, bacteremia, or hospitalization. Adult glomerular filtration rate (GFR) was estimated from serum creatinine level using the Modification of Diet in Renal Disease, and pediatric values were calculated using the age- and sex-dependent CKiD U25 equation [18, 19]. Allograft function was evaluated at discharge, 1, 3, 6, and 12 months, annually thereafter, and at the last available follow-up.

Propensity score matching was used to reduce confounding arising from systematic baseline differences between recipients with and without CAKUT. For propensity score analysis, multivariable logistic regression analysis was done with CAKUT and non-CAKUT as binary dependent variables, and the following variables as independent variables: recipient age, sex, previous transplantation, preemptive transplantation, dialysis duration, donor type, donor age, HLA mismatch number, panel-reactive antibody positivity. Subsequently, recipients with CAKUT were matched 1:1 to non-CAKUT recipients using nearest-neighbor matching without replacement and a caliper width of 0.20. Matching for the propensity score was performed using the R version 4.6.1 (R Foundation for Statistical Computing, Vienna, Austria).

Continuous variables were summarized with mean±standard deviation, and categorical variables with number and percentage. Baseline characteristics were presented for the full unmatched groups and the propensity score-matched groups. In matched analyses, continuous outcomes were compared using paired t tests or Wilcoxon signed-rank tests, and categorical outcomes were compared using McNemar tests or regression models accounting for matched pairs. Kaplan-Meier curves were used to describe graft survival, overall graft survival, and patient survival. Survival curves were compared using the log-rank test. Associations between CAKUT status and time-to-event outcomes were estimated using Cox proportional hazards models. Statistical analysis was performed using SPSS version 22.0 (IBM, NY, USA). A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 1124 KT were performed between January 2010 and August 2025. Among these recipients, 146 had kidney failure attributed to CAKUT. Eight patients were excluded because of multiorgan transplantation (n = 2), lost their graft within 30 days post-transplant (n = 1) and missing data (n = 5) in CAKUT group, and 36 patients were excluded because of lost their graft within 30 days post-transplant (n = 11) and missing data (n = 25) in non-CAKUT group. The final unmatched cohort therefore included 138 recipients with CAKUT and 942 potentially eligible recipients with non-CAKUT causes of kidney failure.

The CAKUT group comprised 66 recipients with vesicoureteral reflux or reflux nephropathy (47.8%), 27 with renal hypoplasia or dysplasia (19.6%), 15 with a complex congenital bladder disorder (10.9%), 14 with posterior urethral valves (10.1%), 12 with congenital obstructive uropathy (8.7%), and 4 with other CAKUT phenotypes (2.9%). Lower urinary tract involvement was documented in 42 recipients (30.4%), 20

patients (14.5%) had undergone previous urinary tract reconstruction, and 15 (10.9%) were using clean intermittent catheterization at the time of transplantation.

Before matching, CAKUT recipients were younger, more frequently male, more likely to undergo preemptive transplantation, and less likely to undergo living-donor transplantation than non-CAKUT recipients (all $p < 0.05$), while dialysis duration and donor age also differed significantly between the groups. After matching, none of the baseline covariates differed significantly between the groups (all $p > 0.05$) (Table 1). In total, 276 KT recipients who were followed up for a median of 126 (IQR 72–156) months were included in the study. DGF occurred in 12 CAKUT recipients (8.7%) and 15 matched controls (10.9%; $p = 0.59$). Biopsy-proven acute rejection during the first posttransplant year was diagnosed in 12 CAKUT recipients (8.7%) and 14 non-CAKUT recipients (10.1%; $p = 0.62$). Major surgical complications classified as Clavien-Dindo grade 3 or higher occurred in 11 CAKUT recipients (8.0%) and 7 non-CAKUT recipients (5.1%; $p = 0.33$).

Table 1
Baseline characteristics before and after propensity score matching.

	Unmatched			Matched		
	CAKUT (n = 138)	Non-CAKUT (n = 942)	p value	CAKUT (n = 138)	Non-CAKUT (n = 138)	p value
Recipient age, years	34 (23–46)	48 (34–58)	< 0.05	34 (23–46)	35 (24–47)	0.680
Male Sex	86 (62.3%)	499 (52.9%)	0.043	86 (62.3%)	87 (63.0%)	0.992
Living donor transplantation	104 (75.3%)	788 (83.6%)	< 0.05	104 (75.3%)	109 (78.9%)	0.796
Preemptive transplantation	29 (21.0%)	108 (11.4%)	< 0.05	29 (21.0%)	25 (18.1%)	0.770
Dialysis duration, months	18 (0–44)	30 (12–58)	< 0.05	18 (0–44)	20 (0–46)	0.710
Previous kidney transplantation	8 (5.8%)	89 (9.4%)	0.208	8 (5.8%)	9 (6.5%)	0.989
PRA positivity	17 (12.3%)	169 (17.9%)	0.133	17 (12.3%)	18 (13.0%)	0.932
HLA mismatch	3 (2–4)	3 (2–4)	0.720	3 (2–4)	3 (2–4)	0.917
Donor age, years	45 ± 12.3	48 ± 13.9	< 0.05	45 ± 12.3	46 ± 13.1	0.521

Values are number (%), mean ± standard deviation, or median (interquartile range). CAKUT, congenital anomalies of the kidney and urinary tract; PRA, panel-reactive antibody; HLA, human leukocyte antigen

At least one symptomatic UTI occurred in 38 CAKUT recipients (27.5%) compared with 19 matched non-CAKUT recipients (13.8%; p = 0.005). Recurrent UTI was observed in 17 patients in the CAKUT group (12.3%) and 7 patients in the non-CAKUT group (5.1%; p = 0.033). Graft pyelonephritis occurred in 15 CAKUT recipients (10.9%) and 7 non-CAKUT recipients (5.1%), although this difference did not reach statistical significance (p = 0.075) (Table 2). Within the CAKUT group, symptomatic UTI (n = 19/42 (45.2%) vs n = 19/96 (19.8%); p = 0.003) and recurrent UTI (n = 9/42 (21.4%) vs n = 8/96 (8.3%); p = 0.031) were more common among recipients with LUTD than among those without LUTD. Recipients who had

undergone urinary tract reconstruction before transplantation also had a higher incidence of recurrent UTI than recipients without previous reconstruction (n = 5/20 (25.0%) vs n = 12/118 (10.2%); p = 0.047).

Table 2
Early posttransplant and infectious outcomes in the matched cohort.

Delayed graft function	**CAKUT (n = 138)**	**Non-CAKUT (n = 138)**	**p value**
	12 (8.7%)	**15 (10.9%)**	**0.543**
Biopsy-proven acute rejection within 1 year	12 (8.7%)	14 (10.1%)	0.680
Clavien-Dindo grade 3 or higher complication	11 (8.0%)	7 (5.1%)	0.329
Symptomatic UTI	38 (27.5%)	19 (13.8%)	0.004
Recurrent UTI	17 (12.3%)	7 (5.1%)	0.032
Graft pyelonephritis	15 (10.9%)	7 (5.1%)	0.075
Values are number (%), UTI, urinary tract infection			

Median estimated glomerular filtration rate (eGFR) at 1 month was 58.7 mL/min/1.73 m2 in CAKUT recipients and 56.1 mL/min/1.73 m2 in matched controls (p = 0.28). At 12 months, median eGFR was 63.1 mL/min/1.73 m2 in the CAKUT group and 59.2 mL/min/1.73 m2 in the non-CAKUT group (p = 0.09). At 5 years, the eGFR values were 58.4 and 53.6 mL/min/1.73 m2, respectively (p = 0.06). At the final follow-up, median eGFR was 55.8 mL/min/1.73 m2 in CAKUT recipients and 50.9 mL/min/1.73 m2 in matched controls (p = 0.08).

During follow-up, death-censored graft failure occurred in 14 CAKUT recipients (10.1%) and 25 non-CAKUT recipients (18.1%). The estimated 1-, 5-, and 10-year death-censored graft survival rates were 98.5%, 94.2%, and 88.3% in the CAKUT group and 97.8%, 89.0%, and 76.9% in the non-CAKUT group, respectively (log-rank p = 0.018) (Fig. 1.A). Overall graft failure, including death with a functioning graft, occurred in 21 CAKUT recipients (15.2%) and 33 matched controls (23.9%). The estimated 1-, 5-, and 10-year overall graft survival rates were 97.8%, 91.3%, and 82.4% in the CAKUT group and 97.1%, 85.5%, and 73.8% in the non-CAKUT group, respectively (log-rank P = 0.071) (Fig. 1.B). Seven CAKUT recipients (5.1%) and eight non-CAKUT recipients (5.8%) died during follow-up. The estimated 1-, 5-, and 10-year

patient survival rates were 99.2%, 97.0%, and 93.5% in CAKUT recipients and 99.1%, 96.1%, and 92.7% in matched controls, respectively (log-rank P = 0.740) (Fig. 1.C).

In the multivariable Cox proportional hazards model, CAKUT was associated with a lower risk of death-censored graft failure compared with non-CAKUT kidney disease (HR 0.55; 95% CI, 0.29–0.99; p = 0.048). DGF, biopsy-proven acute rejection, and pretransplant panel-reactive antibody positivity were independently associated with graft failure. Symptomatic UTI was not independently associated with graft failure after adjustment for rejection, DGF, and immunological risk (Table 3).

Table 3
Cox proportional hazards models for death-censored graft failure.

	Univariable			Multivariable		
	HR	**95% CI**	**p value**	**HR**	**95% CI**	**p value**
Recipient age per 10 years	1.08	0.89–1.31	0.44			
CAKUT status	0.52	0.28–0.96	0.037	0.55	0.29–0.99	0.048
Delayed graft function	2.37	1.28–4.39	0.006	2.09	1.09–4.01	0.027
Biopsy-proven acute rejection	4.01	2.24–7.18	< 0.001	3.44	1.88–6.31	< 0.001
PRA positivity	2.68	1.39–5.18	0.003	2.41	1.21–4.79	0.012
Symptomatic UTI	2.05	1.03–4.09	0.041	1.47	0.70–3.08	0.31
Living donor transplantation	0.74	0.39–1.41	0.36			

HR, hazard ratio; CI, confidence interval; CAKUT, congenital anomalies of the kidney and urinary tract; PRA, panel-reactive antibody; UTI, urinary tract infection

DISCUSSION

This propensity score-matched study showed that kidney transplant recipients with CAKUT experienced significantly higher rates of symptomatic and recurrent UTI but had similar rates of DGF, biopsy-proven acute rejection, major surgical complications, allograft function, and patient survival compared with non-CAKUT recipients, while death-censored graft survival was superior in the CAKUT group. After adjustment, CAKUT was associated with a lower risk of death-censored graft failure, and the association with UTI was weakened after adjustment for immunological and early posttransplant factors, consistent with previous matched cohort studies and large registry analyses [11–14]. Our findings indicate that, with appropriate evaluation and management of the urinary tract, CAKUT is associated with increased urinary morbidity but not with inferior transplant survival.

We found the 10-year death-censored graft survival of 88.3% and 76.9% in the CAKUT and non-CAKUT groups, respectively (p = 0.018). In the United States Renal Data System analysis of more than 10,000

CAKUT recipients, Cornwell et al. similarly reported superior death-censored graft survival in CAKUT compared with matched non-CAKUT controls, and this advantage persisted in both pediatric and adult strata [11]. Oto et al. reported 87.6% 10-year graft survival in the Turkish propensity score-matched study, in which CAKUT was also independently associated with a lower risk of graft loss [12]. Pediatric data from Monteverde et al. also showed better graft survival in CAKUT than in recurrent and nonrecurrent glomerular disease groups, despite a greater frequency of UTI during the first posttransplant year [13]. By contrast, McKay et al. reported 10-year graft survival between 70% and 76% across major CAKUT phenotypes, illustrating the influence of diagnostic composition, transplant era, donor source, recipient age, and duration of follow-up on survival estimates [8]. A possible explanation for the favorable death-censored graft survival in CAKUT is the absence of recurrence of the congenital primary disease in the allograft, whereas non-CAKUT control recipients often include focal segmental glomerulosclerosis, immune-complex glomerulonephritis, and other disorders with recurrence-related graft injury [8, 11–13]. Younger recipient age, a greater likelihood of preemptive transplantation, and living donation may also contribute to the apparent CAKUT advantage, although propensity score matching reduces but cannot eliminate confounding from these factors [2, 4, 11, 12]. The persistence of a protective association after matching may therefore reflect a combination of true biological differences, differential recurrence risk, residual confounding, and selection of CAKUT candidates whose lower urinary tracts were considered sufficiently safe for transplantation [4, 11–13].

The higher rates of symptomatic UTI and recurrent UTI in the present CAKUT cohort are consistent with the 20.7% versus 10.7% UTI difference reported by Oto et al. and with pediatric studies showing greater infectious morbidity after KT for CAKUT [12, 13]. The particularly high UTI burden among recipients with LUTD is biologically plausible because impaired compliance, incomplete emptying, residual urine, catheterization, reflux, and reconstructed urinary reservoirs may facilitate bacterial persistence and ascending infection [4–7, 9, 10, 15, 20]. Marchal et al. found that enterocystoplasty and continent urinary diversion were associated with more frequent acute graft pyelonephritis, although long-term graft survival remained similar to transplantation into native bladders when bladder management was adequate [10]. Comparable findings have been reported in cohorts of recipients with posterior urethral valves, vesicoureteral reflux nephropathy, and other forms of lower urinary tract dysfunction, in whom infection rates were elevated but acceptable graft outcomes were achievable after appropriate bladder optimization and catheterization strategies [6, 7, 9, 15]. In our study, symptomatic UTI was associated with death-censored graft failure in the univariable model but lost statistical significance after adjustment for CAKUT status, DGF, rejection, and pretransplant PRA status, suggesting that UTI may partly function as a marker of recipient complexity rather than an independent cause of graft loss in this cohort. The published literature on this association is heterogeneous. Jahromi et al. reported that posttransplant UTI was associated with graft loss in a pediatric cohort, whereas Oto et al. did not identify UTI as an independent predictor after multivariable adjustment [12, 14]. In addition, recurrent or febrile infections may have different prognostic implications from isolated UTI episodes, and analyses based only on the occurrence of any UTI may obscure important differences in frequency, severity, microbial resistance, and graft involvement [3, 10, 14, 15].

The comparable eGFR trajectories, rejection rates, and DGF rates between the matched groups support the view that appropriately selected CAKUT recipients do not have an inherent disadvantage in early or intermediate allograft function. In the current analysis, we found that DGF, acute rejection, and PRA positivity were independently associated with graft failure. Monteverde et al. reported lower DGF and higher eGFR in pediatric CAKUT recipients, while DGF and acute rejection remained major predictors of graft loss across etiological groups [13]. Oto et al. identified pretransplant PRA positivity and kidney failure due to non-CAKUT etiologies as independent predictors of graft failure [12]. These observations emphasize that conventional immunological and perioperative determinants remain central to graft prognosis even in recipients with complex congenital urinary tract disease [4, 12, 13].

The heterogeneity of CAKUT remains important because aggregate comparisons may conceal clinically relevant differences among renal hypodysplasia, reflux nephropathy, posterior urethral valves, neurogenic bladder, and complex reconstructed lower urinary tracts [4, 6–10, 15]. McKay et al. found that recipients with posterior urethral valves had an increased risk of graft loss beyond 10 years compared with recipients with renal hypoplasia or dysplasia, suggesting that adverse effects of bladder dysfunction may emerge only during prolonged follow-up [8]. Conversely, studies focused on posterior urethral valves and LUTD have demonstrated acceptable graft survival when bladder pressures, emptying, and catheterization are actively managed [6, 7, 9]. Recent pediatric data also indicate that vesicoureteral reflux into the transplanted kidney does not reduce medium-term graft or patient survival, although the selected nature of patients undergoing posttransplant cystography limits generalizability [15].

This study has several limitations. Its retrospective single-center design is susceptible to incomplete documentation, indication bias, temporal changes in transplant practice, and residual confounding despite propensity score matching. The number of graft-failure events and the sizes of individual CAKUT subgroups limit the precision of phenotype-specific and multivariable estimates. Infection outcomes were based on documented clinical episodes rather than standardized screening, and the available data did not fully characterize antimicrobial resistance, recurrent-event timing or bladder pressure. Despite these limitations, the study adds adult-dominant, single-center propensity score-matched evidence to a literature that remains heavily weighted toward pediatric cohorts, registry analyses, and small diagnosis-specific series [4, 8, 10–14]. The findings support a multidisciplinary pathway in which transplantation is not withheld solely because of CAKUT, bladder augmentation, diversion, reflux, or the requirement for clean intermittent catheterization [4–7, 9, 10, 15]. Pretransplant evaluation should identify unsafe storage pressure, impaired compliance, obstruction, residual urine, and catheterization barriers, while posttransplant care should prioritize adherence to bladder-emptying regimens and prompt evaluation of febrile or recurrent infection [4–7, 9, 10, 15]. Future multicenter studies should use standardized CAKUT phenotyping, urodynamic definitions, infection-event rates, and sufficiently long follow-up to determine whether specific lower urinary tract phenotypes modify late graft survival.

CONCLUSION

In this propensity score-matched, predominantly adult cohort, kidney transplant recipients with CAKUT achieved comparable allograft function and patient survival and superior death-censored graft survival despite a significantly greater burden of symptomatic and recurrent UTI. Kidney transplantation should therefore remain the preferred kidney replacement therapy for suitable patients with CAKUT. Prospective multicenter studies are required to validate these findings and to define which CAKUT phenotypes require intensified long-term urological and transplant follow-up.

Abbreviations

& Acronyms
CAKUT
Congenital anomalies of the kidney and urinary tract
DGF
Delayed graft function
GFR
Glomerular filtration rate
KT
Kidney transplantation
LUTD
Lower urinary tract dysfunction
PRA
Panel-reactive antibody
UTI
Urinary tract infection

Declarations

Ethics approval and consent to participate

The study was approved by the local ethics committee of Acibadem University (approval number ATADEK 2025-14, Clinical trial number: not applicable) and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent prior to enrolment.

Consent for publication:

Publication consent for research findings was obtained from each participant

Conflicts of Interest:

The author(s) declare that there is no conflict of interest regarding the publication of this study.

Authorship: • Participated in research design:

AE, IO **Participated in the writing of the paper**: RGE, **Participated in the performance of the research**: HT; ESO, **Contributed new reagents or analytic tools**: AE; RGE; ESO, **Participated in data analysis**: AE; HT; RGE

Funding:

The authors received no financial support for the research, authorship, and/or publication of this article.

Author Contribution

Authorship: • Participated in research design: AE, IO Participated in the writing of the paper: RGE, Participated in the performance of the research: HT; ESO, Contributed new reagents or analytic tools: AE; RGE; ESO, Participated in data analysis: AE; HT; RGE

Data Availability

Data will be made available on request.

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Figures

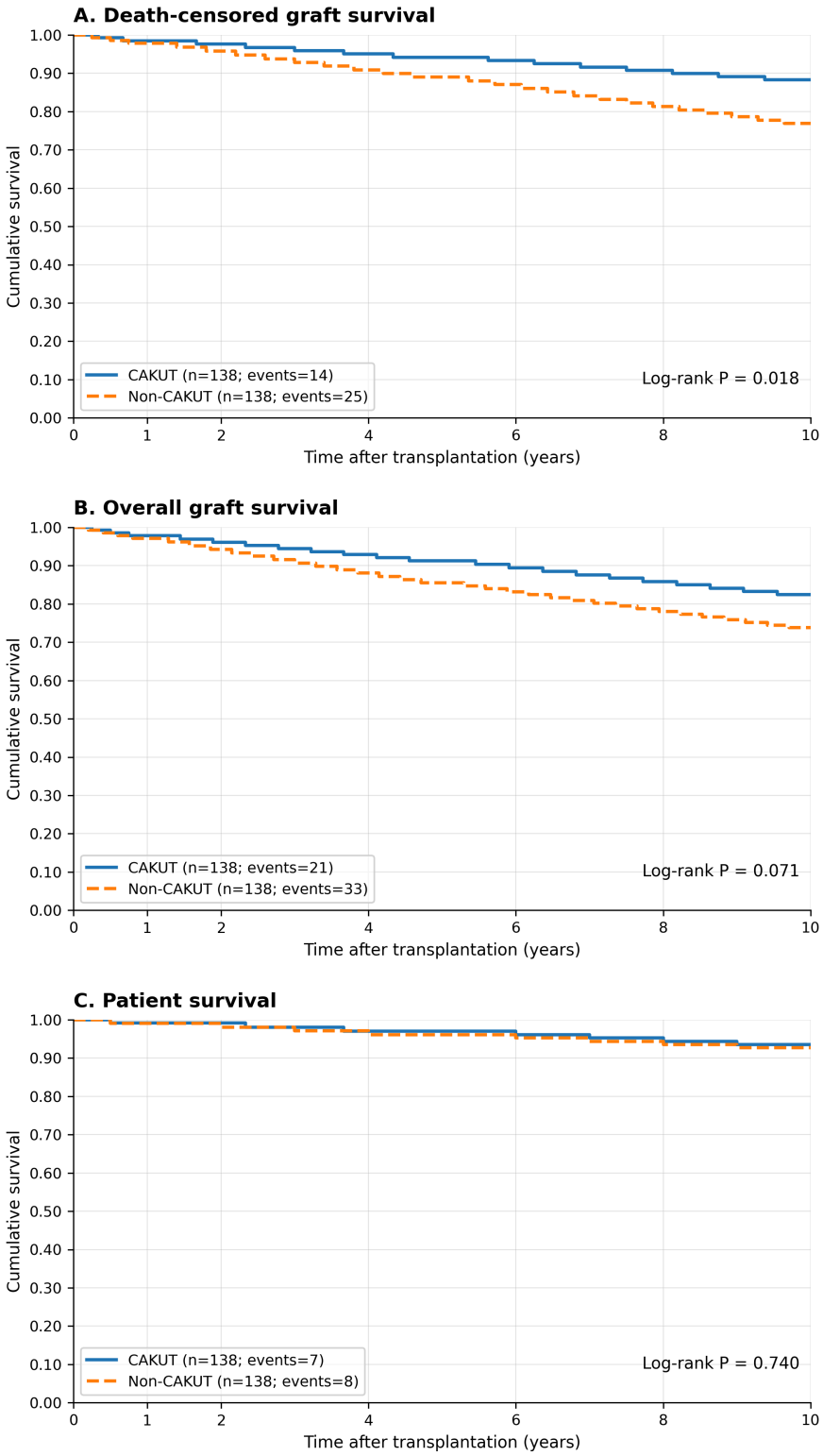


Figure 1

A. Kaplan-Meier analysis of death-censored graft survival. **B.** Kaplan-Meier analysis of overall graft survival. **C.** Kaplan-Meier analysis of patient survival.