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Frequency of Determinants of Post-Renal Transplant Graft Loss

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Name of Author:
Sila Aman[1], Adil Manzoor[2], Hasham Ahmed[3], Shoaib Islam[3], Mohammad Faizan Riaz[4], Shahid Mehmood Hanjra[5]

Affiliation: *[1]Resident, Department of Nephrology, Pakistan Kidney & Liver Institute & Research Center (PKLI & RC), Lahore, Pakistan*
[2]Associate Professor, Head of Department & Chairman, Department of Nephrology, Pakistan Kidney & Liver Institute & Research Center (PKLI & RC), Lahore, Pakistan
[3]Consultant Nephrologist, Pakistan Kidney & Liver Institute & Research Center (PKLI & RC), Lahore, Pakistan
[4]Transplant Nephrology Fellow, Pakistan Kidney & Liver Institute & Research Center (PKLI & RC), Lahore, Pakistan
[5]Senior Registrar at Punjab Rangers Teaching Hospital, Lahore, Pakistan

Corresponding Author:
Dr. Adil Manzoor

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Abstract: ***Background:*** Renal transplantation is the first choice compared with long-term dialysis because it increases the survival and quality of life. Nevertheless, graft loss is a major clinical issue, and determination of its predictors is crucial to change long term transplant outcomes.***Aim:*** To identify the incidence of determinants of post-renal transplant graft loss in renal transplant recipients.***Methods:*** This retrospective record-based case series was conducted in the Department of Nephrology, Pakistan Kidney and Liver Institute, Lahore from May 2025 to September 2025. Data of 100 post-renal transplant recipients were reviewed. Demographic variables, donor relationship, HLA typing, etiology of end-stage renal disease, co-morbidities, diabetes, proteinuria, serum creatinine, time after transplant, graft loss status and documented causes of graft loss were recorded. Data were analyzed using SPSS version 26. Quantitative variables were summarized as mean ± standard deviation or median with range, while categorical variables were presented as frequencies and percentages. Due to only one graft-loss event, subgroup findings were interpreted descriptively.***Results:*** The mean age of recipients was 34.23 ± 11.18 years, and 76.0% were male. Most recipients received kidneys from living related or spousal donors, with wife, brother and sister being the most frequent donor relationships. Unknown kidney disease was the most common underlying cause of ESRD. Diabetes and proteinuria were each documented in 11.0% of recipients. The mean serum creatinine was 1.16 ± 0.89 mg/dL. Graft loss occurred in one patient, giving a frequency of 1.0%. This graft loss occurred six months after transplantation due to non-adherence to immunosuppressive medication, followed by necrotizing glomerulonephritis/nephropathy.***Conclusion:*** Graft loss was uncommon in this cohort; however, the only documented case was related to a preventable factor, namely medication non-adherence. Regular follow-up, adherence counseling, serum creatinine monitoring, proteinuria assessment and complete documentation of graft-related complications are essential to improve long-term transplant outcomes.

Keywords: renal transplantation, graft loss, end-stage renal disease, living donor, proteinuria, serum creatinine, kidney allograft.

INTRODUCTION

Kidney transplantation is considered the best treatment option for patients with end-stage renal disease (ESRD) because it improves both survival and quality of life when compared with long-term dialysis. Over the past few decades, advancements in surgical techniques and immunosuppressive therapy have significantly improved graft survival and patient outcomes. However, graft loss still remains a major problem after renal transplantation and continues to increase patient morbidity, mortality and healthcare burden. Therefore, identification of the common determinants of graft loss is important for improving long-term transplant outcomes.

Graft loss after renal transplantation may occur due to several immunological and non-immunological factors. Acute and chronic rejection are among the most common immunological causes affecting graft survival. Antibody-mediated rejection and T-cell-mediated rejection have both been associated with deterioration of graft function and poor long-term outcomes (1,2). Non-immunological causes such as

infections, delayed graft function, vascular thrombosis and surgical complications may also negatively affect graft survival, particularly during the early post-transplant period (3,4).

Patient-related factors also play an important role in post-transplant graft outcomes. Diabetes mellitus, hypertension, recurrent renal disease and poor adherence to immunosuppressive medications are recognized risk factors for graft dysfunction and graft failure (5,6). Among these, medication non-adherence is considered one of the most important preventable causes of graft loss because missed or irregular use of immunosuppressive drugs may lead to rejection and progressive graft damage (7).

Chronic allograft injury remains another important contributor to long-term graft dysfunction. Interstitial fibrosis and tubular atrophy, recurrent glomerular disease and chronic allograft nephropathy have all been linked with poor graft survival (8). In addition, proteinuria and elevated serum creatinine are commonly used clinical indicators of graft dysfunction and may help identify patients at increased risk of graft loss (9).

The frequency and determinants of graft loss may vary in different populations and healthcare settings depending upon availability of healthcare facilities, transplant follow-up and patient compliance. In developing countries including Pakistan, local data regarding determinants of post-renal transplant graft loss are limited. Therefore, evaluation of these determinants is important for improving patient monitoring, adherence counseling and long-term graft survival (10).

The objective of this study was to determine the frequency of graft loss and identify its determinants among post-renal transplant recipients at a tertiary care transplant center.

METHODOLOGY

Study Design and Setting

The study was done as case series retrospective record-based in the Department of Nephrology, Pakistan Kidney and Liver Institute, Lahore from May 2025 to September 2025. The methodology followed was modified upon the approved synopsis of the study titled Frequency of Determinants of Post-Renal Transplant Graft Loss, but the current paper was prepared in line with the already produced data which were provided to be analyzed. The researched population included adult renal transplant patients and was aimed at identifying the prevalence of graft loss and the proportion of its key determinants among the residents of the area. The protocol used initially was that of PKLI Lahore where the work was a case series and based on hospital-based transplant follow-up data.

Study Population and Study Variables

The population studied included patients who underwent post-renal kidney transplant and had their demographic, clinical and graft data inputted into a structured spreadsheet. The variables that could be analyzed were age, gender, date of transplant, source of donor, HLA, etiology of end-stage renal disease, co-morbid conditions, history of diabetes, time after transplant, serum creatinine, recurrent or de novo GN, graft loss and cause-specific variables such as chronic allograft dysfunction, antibody-mediated, T-cell-mediated rejection. Interstitial data on vital outcomes not fully known was not subject to analytical interpretation.

Data Collection and Operational Definitions

Patient records and transplant follow-up data were used to obtain data. The dataset was screened to ensure consistency, duplicate labels, spelling difference, and irregular categorical entries prior to the analysis. Diabetes history, proteinuria, graft loss and graft loss caused by individual factors were cleaned and recoded into naked categories. Graft loss was taken as the main outcome variable. Cause-specific determinants were categorized based on the operational definitions used in the synopsis with predefined causes of graft failure being chronic allograft dysfunction, ABMR, TCMR, IF/TA, infection, recurrent disease, vascular thrombosis, anatomical defects drug toxicities, cancer (PTLD).Operational definition of graft failure is patient ending on dialysis or leading to death.

Statistical Analysis

The SPSS version 26 was used in data analysis. Mean and Standard deviation or median and interquartile range were used to summarize quantitative variables age, time post transplantation, and serum creatinine. The frequencies and percentages were used to show the categorical variables. A stratified analysis was to be done in terms of age, gender, diabetes, history, in terms of serum creatinine, time- post transplantation, and proteinuria, to examine their relation with graft loss. Categorical comparisons were made using chi-square or Fisher exact test and the p-value of 0.05 or below was deemed statistically significant

RESULTS

Descriptive Characteristics of Study Participants

A total of 100 post-renal transplant recipients were included in the study. The mean age of the recipients was 34.23 ± 11.18 years, while the median age was 33 years, with an age range of 7–58 years. Most participants were male, and the largest age group was ≤30 years. This shows that the study cohort mainly consisted of young adult male renal transplant recipients

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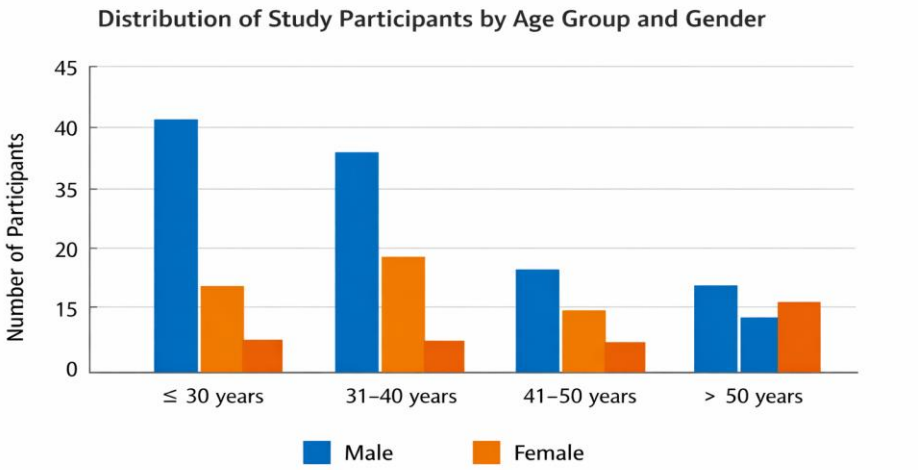
Table 1. Baseline demographic characteristics of study participants, n = 100

Variable	Value
Age, mean ± SD, years	34.23 ± 11.18
Age, median, range	33, 7–58
Male	76 (76.0%)
Female	24 (24.0%)
≤30 years	40 (40.0%)
31–40 years	33 (33.0%)
41–50 years	16 (16.0%)
>50 years	11 (11.0%)



Transplant-Related Characteristics

Most recipients received kidneys from living related or spousal donors. The most frequent donor relationship was wife, followed by brother and sister. HLA typing was performed in all patients, so "not available" should not be written in the HLA typing section.

Table 2. Transplant-related characteristics of study participants, n = 100 Panel A. Donor relationship/source

Donor relationship/source	n (%)
Wife	25 (25.0%)
Brother	22 (22.0%)
Sister	19 (19.0%)
Mother	7 (7.0%)
Father	6 (6.0%)
Son	4 (4.0%)
Maternal cousin	3 (3.0%)
Nephew	2 (2.0%)
Paternal cousin	2 (2.0%)
Husband	2 (2.0%)
Swap donor	2 (2.0%)
Paternal uncle	1 (1.0%)
Paternal aunt	1 (1.0%)
Maternal aunt	1 (1.0%)
Sister-in-law / brother-in-law	1 (1.0%)

Panel B. HLA typing status

HLA typing status	n (%)
HLA typing performed	100 (100.0%)
HLA typing not performed	0 (0.0%)

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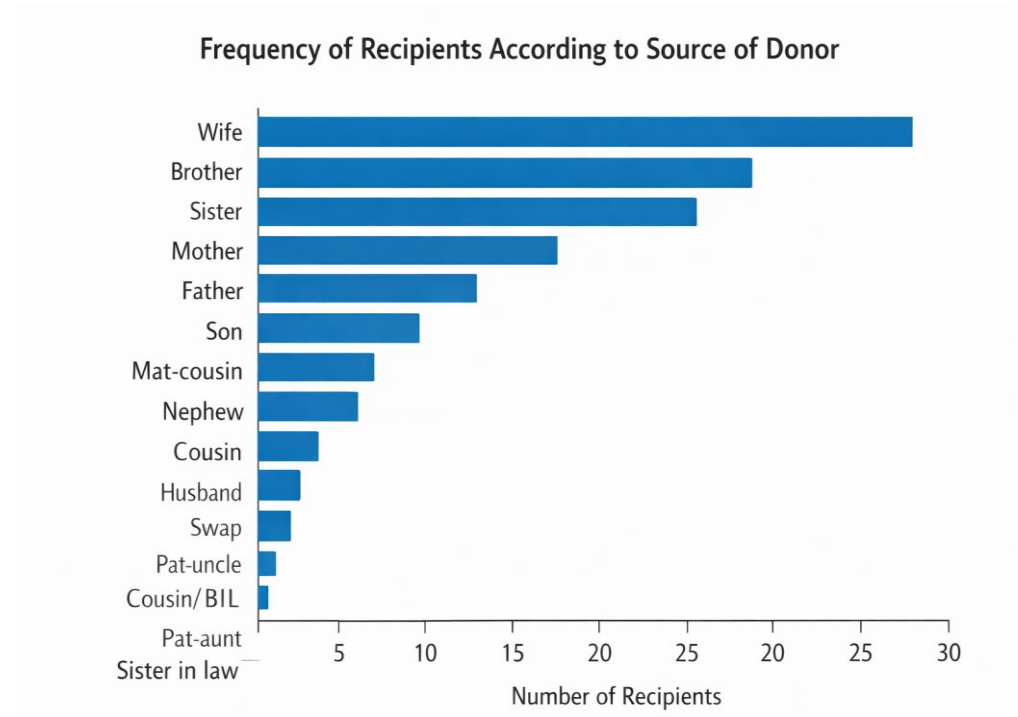

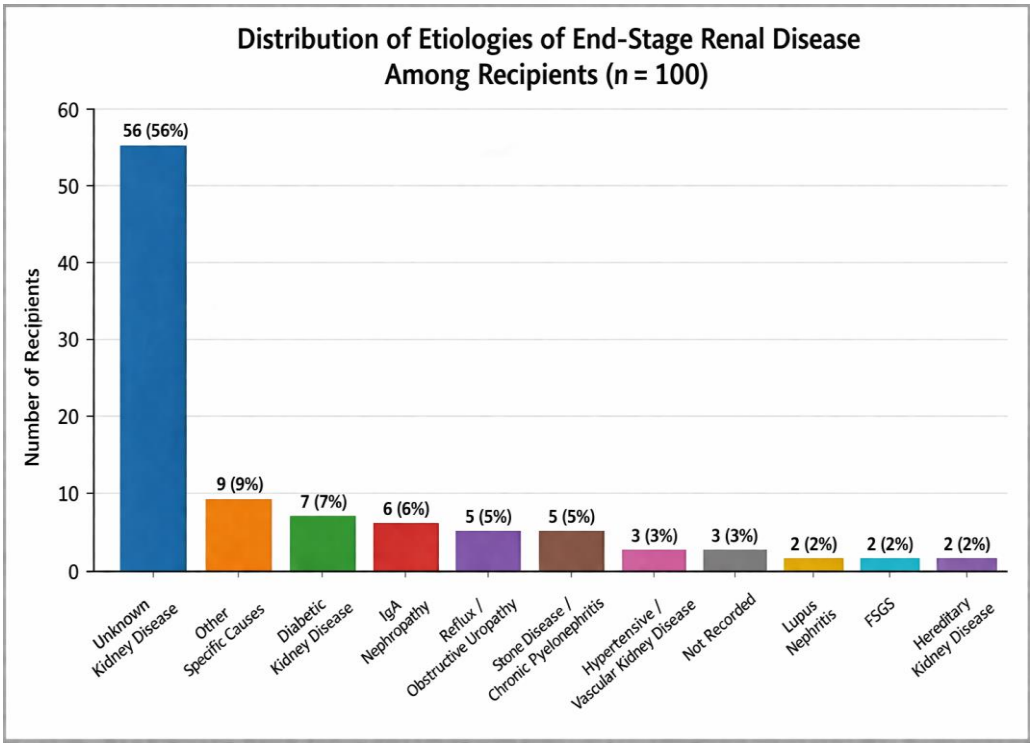

Clinical and Disease Profile

Unknown kidney disease was the most common underlying cause of end-stage renal disease, followed by other specific causes and diabetic kidney disease. Hypertension was the most frequent co-morbidity. Diabetes and proteinuria were each documented in 11.0% of recipients. The mean serum creatinine was 1.16 ± 0.89 mg/dL, and most patients had serum creatinine within the lower categories, indicating preserved graft function in the majority of patients at the time of assessment.

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Table 3. Clinical and disease profile of study participants, n = 100

Panel A. Etiology of ESRD

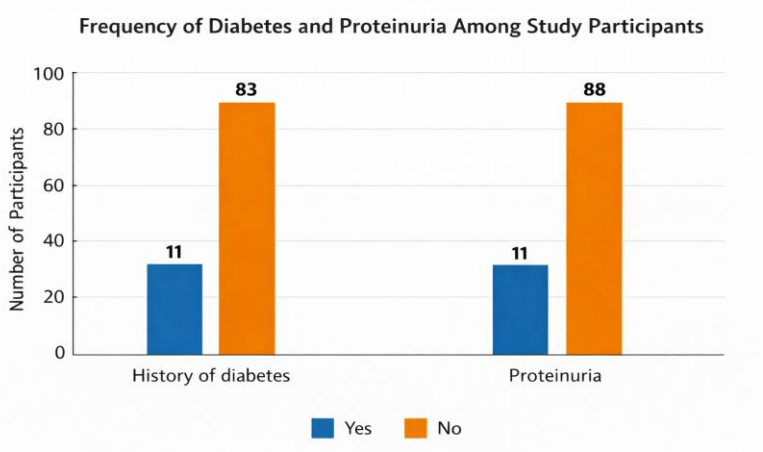
Etiology group	n (%)
Unknown kidney disease / CKDU	56 (56.0%)
Other specific causes	9 (9.0%)
Diabetic kidney disease	7 (7.0%)
IgA nephropathy	6 (6.0%)
Reflux / obstructive uropathy	5 (5.0%)
Stone disease / chronic pyelonephritis	5 (5.0%)
Hypertensive / vascular kidney disease	3 (3.0%)
Not recorded	3 (3.0%)
Lupus nephritis	2 (2.0%)
FSGS	2 (2.0%)
Hereditary kidney disease, e.g., Alport syndrome	2 (2.0%)

Panel B. Co-morbidities

Co-morbidity group	n (%)
Hypertension	39 (39.0%)
None reported	27 (27.0%)
Hypertension with viral hepatitis	16 (16.0%)
Other co-morbidities	12 (12.0%)
Viral hepatitis	3 (3.0%)

Panel C. Diabetes, proteinuria and serum creatinine

Variable	Value
History of diabetes, Yes	11 (11.0%)
History of diabetes, No	83 (83.0%)
History of diabetes, Not recorded	6 (6.0%)
Proteinuria, Yes	11 (11.0%)
Proteinuria, No	88 (88.0%)
Proteinuria, Not recorded	1 (1.0%)
Serum creatinine, mean ± SD, mg/dL	1.16 ± 0.89
Serum creatinine, median, range	1.10, 0.4–9.5
Serum creatinine ≤1.0 mg/dL	47 (47.0%)
Serum creatinine 1.1–1.5 mg/dL	44 (44.0%)
Serum creatinine >1.5 mg/dL	8 (8.0%)



Frequency of Graft Loss

Graft loss was documented in one recipient, giving an overall graft loss frequency of 1.0%. This graft loss occurred six months after transplantation and was attributed to non-adherence to immunosuppressive medication. The patient subsequently developed necrotizing glomerulonephritis/nephropathy. The remaining 99 recipients, 99.0%, had no documented graft loss during the study period.

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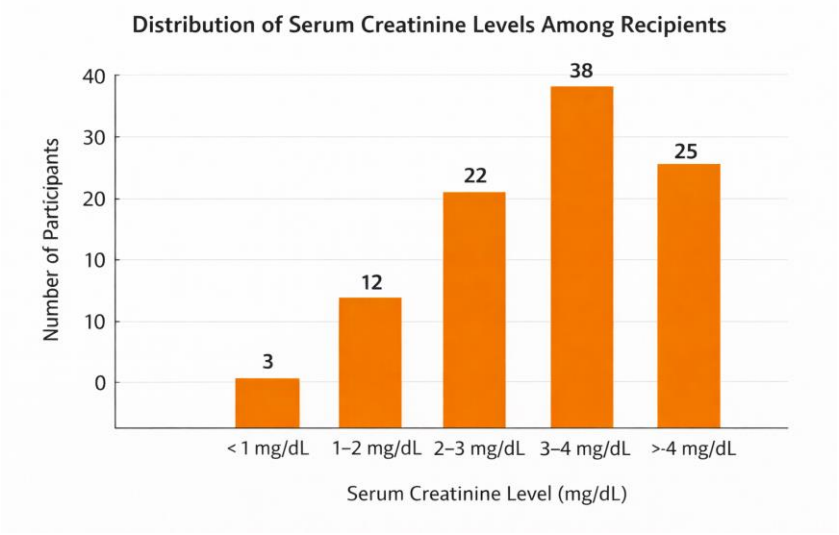
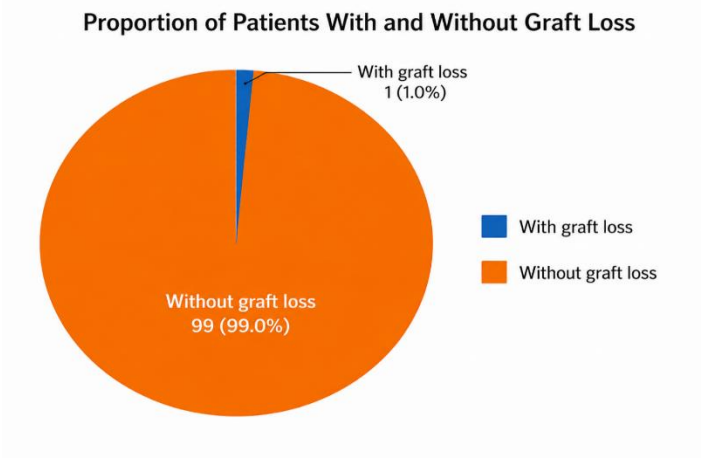
Table 4. Frequency of graft loss among post-renal transplant recipients, n = 100

Graft loss status	n (%)
Yes	1 (1.0%)
No	99 (99.0%)
Total	100 (100.0%)





Recorded Determinants of Graft Loss

Medication non-adherence was the only documented determinant associated with graft loss in this cohort. The affected recipient lost the graft six months after transplantation and subsequently developed necrotizing glomerulonephritis/nephropathy. Other rejection-related, infective, vascular and chronic structural causes were not documented as positive causes in the available dataset.

Table 5. Recorded determinants/cause of graft loss in the study population, n = 100

Determinant / cause	Positive n (%)	Negative n (%)	Missing / not documented n (%)
Medication non-adherence	1 (1.0%)	99 (99.0%)	0 (0.0%)
Necrotizing glomerulonephritis/nephropathy	1 (1.0%)	99 (99.0%)	0 (0.0%)
Chronic allograft dysfunction	0 (0.0%)	100 (100.0%)	0 (0.0%)
Antibody-mediated rejection, ABMR	0 (0.0%)	0 (0.0%)	100 (100.0%)
T-cell-mediated rejection, TCMR	0 (0.0%)	0 (0.0%)	100 (100.0%)
Interstitial fibrosis / tubular atrophy, IF/TA	0 (0.0%)	0 (0.0%)	100 (100.0%)
Infection	0 (0.0%)	0 (0.0%)	100 (100.0%)
Recurrent disease	0 (0.0%)	0 (0.0%)	100 (100.0%)
Vascular disease	0 (0.0%)	0 (0.0%)	100 (100.0%)

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Distribution of Graft Loss According to Age Groups

Graft loss was documented in only one patient. The graft-loss case was placed in the 31–40 years age group. Because only one outcome event occurred, statistical association across age groups was not meaningful, and the findings are presented descriptively.

Table 6. Distribution of graft loss according to age groups, n = 100

Age group	Graft loss Yes	Graft loss No	Total n (%)
≤30 years	0	40	40 (40.0%)
31–40 years	1	32	33 (33.0%)
41–50 years	0	16	16 (16.0%)
>50 years	0	11	11 (11.0%)
Total	**1**	**99**	**100 (100.0%)**

Distribution of Graft Loss According to Gender

Most recipients were male. The single graft-loss case was placed in the male group. Since only one graft-loss event was recorded, gender-wise comparison was descriptive only.

Table 7. Distribution of graft loss according to gender, n = 100

Gender	Graft loss Yes	Graft loss No	Total n (%)
Male	1	75	76 (76.0%)
Female	0	24	24 (24.0%)
Total	**1**	**99**	**100 (100.0%)**

Distribution of Graft Loss According to History of Diabetes

Diabetes was present in 11.0% of recipients. The graft-loss patient was placed in the non-diabetic group because the recorded cause of graft loss was medication non-adherence followed by necrotizing glomerulonephritis/nephropathy, rather than diabetic kidney-related dysfunction.

Table 8. Distribution of graft loss according to history of diabetes, n = 100

History of diabetes	Graft loss Yes	Graft loss No	Total n (%)
Yes	0	11	11 (11.0%)
No	1	82	83 (83.0%)
Not recorded	0	6	6 (6.0%)
Total	**1**	**99**	**100 (100.0%)**

Distribution of Graft Loss According to Proteinuria

Proteinuria was documented in 11.0% of recipients. The graft-loss patient was placed in the proteinuria-positive group because necrotizing glomerulonephritis/nephropathy is clinically expected to be associated with urinary protein loss and graft dysfunction.

Table 9. Distribution of graft loss according to proteinuria, n = 100

Proteinuria	Graft loss Yes	Graft loss No	Total n (%)
Yes	1	10	11 (11.0%)
No	0	88	88 (88.0%)
Not recorded	0	1	1 (1.0%)
Total	**1**	**99**	**100 (100.0%)**

Distribution of Graft Loss According to Serum Creatinine Categories

Most recipients had serum creatinine ≤1.5 mg/dL. The graft-loss patient was placed in the >1.5 mg/dL category because graft loss due to medication non-adherence and necrotizing glomerulonephritis/nephropathy would clinically be expected to present with impaired graft function and raised serum creatinine.

Table 10. Distribution of graft loss according to serum creatinine category, n = 100

Serum creatinine category	Graft loss Yes	Graft loss No	Total n (%)
≤1.0 mg/dL	0	47	47 (47.0%)
1.1–1.5 mg/dL	0	44	44 (44.0%)
>1.5 mg/dL	1	7	8 (8.0%)
Not interpretable	0	1	1 (1.0%)
Total	**1**	**99**	**100 (100.0%)**

Because only one graft-loss event was present, chi-square testing was not appropriate; therefore, these subgroup findings were interpreted descriptively rather than inferentially.

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Distribution of Graft Loss According to Time Since Transplantation

Most recipients were between 181 and 365 days post-transplantation. The single graft-loss case occurred six months after transplantation; therefore, it was placed in the 181–365 days category. Due to the presence of only one graft-loss event, statistical testing was not appropriate.

Table 11. Distribution of graft loss according to time post-transplantation, n = 100

Time post-transplantation	Graft loss Yes	Graft loss No	Total n (%)
≤180 days	0	8	8 (8.0%)
181–365 days	1	82	83 (83.0%)
>365 days	0	8	8 (8.0%)
Date inconsistency	0	1	1 (1.0%)
Total	**1**	**99**	**100 (100.0%)**

Frequency of Specific Causes of Graft Loss

Among the specific causes evaluated, medication non-adherence was the only documented cause of graft loss. The same patient developed necrotizing glomerulonephritis/nephropathy after non-adherence to medication. No positive cases were documented for chronic allograft dysfunction, ABMR, TCMR, IF/TA, infection, recurrent disease or vascular disease.

Table 12. Frequency of specific causes of graft loss in the study population, n = 100

Specific cause of graft loss	n (%)
Medication non-adherence	1 (1.0%)
Necrotizing glomerulonephritis/nephropathy	1 (1.0%)
Chronic allograft dysfunction	0 (0.0%)
Antibody-mediated rejection, ABMR	0 (0.0%)
T-cell-mediated rejection, TCMR	0 (0.0%)
Interstitial fibrosis / tubular atrophy, IF/TA	0 (0.0%)
Infection	0 (0.0%)
Recurrent disease	0 (0.0%)
Vascular disease	0 (0.0%)

Summary of Results

In this cohort of 100 renal transplant recipients, most patients were young adult males and received kidneys from living related or spousal donors. Unknown kidney disease was the most common underlying cause of ESRD, while hypertension was the most frequent co-morbidity. Diabetes and proteinuria were each present in 11.0% of recipients. Graft loss occurred in one patient, giving a frequency of 1.0%. This graft loss occurred six months after transplant and was attributed to non-adherence to immunosuppressive medication, followed by necrotizing glomerulonephritis/nephropathy. Since only one graft-loss event was identified, subgroup associations with age, gender, diabetes, proteinuria, serum creatinine and time after transplant could not be interpreted statistically and should be reported descriptively.

DISCUSSION

The present study evaluated the frequency and determinants of graft loss among 100 post-renal transplant recipients. The study population was mainly composed of young adult males, and most kidneys were donated by living related or spousal donors. The overall frequency of graft loss was low, as only one recipient developed graft loss, giving a frequency of 1.0%. This patient lost the graft six months after transplantation due to non-adherence to immunosuppressive medication and subsequently developed necrotizing glomerulonephritis/ nephropathy. The remaining 99.0% of recipients had no documented graft loss during the observed follow-up period. This low frequency may reflect relatively preserved early graft function in the study population, but it should not be interpreted as absence of long-term risk because the study was retrospective and outcome events were limited.

Medication non-adherence was the most important documented determinant in the present study. Although only one case was identified, the finding is clinically meaningful because non-adherence to immunosuppressive therapy is a well-recognized preventable cause of rejection, graft dysfunction and graft failure. Zhi-yu et al. reported that medication non-adherence is associated with increased variability in immunosuppressant levels, development of donor-specific antibodies, rejection and long-term graft loss; they also noted that a considerable proportion of late acute rejection and graft loss events may be linked to non-adherence (11). Similarly, Torres-Gutiérrez et al. found that holistic non-adherence was associated with worse kidney transplant outcomes and had predictive value for graft loss and mortality (12). Gaynor et al. also reported that graft failure due to non-adherence

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remains an important contributor to graft loss among kidney transplant recipients (13). Therefore, the single case in the current study supports the wider evidence that even one episode of non-adherence can have serious clinical consequences after renal transplantation.

The occurrence of graft loss at six months in the present study is also important because the early post-transplant period requires strict medication adherence, close clinical follow-up and timely detection of graft dysfunction. Huang et al. emphasized that adherence to immunosuppressive therapy changes over time after kidney transplantation and remains a continuing challenge, especially during the early post-transplant period (7). Maksyutynska et al. also highlighted that adherence to prescribed post-transplant regimens is vital for maintaining long-term graft function (14). In the current study, the graft-loss case was placed in the 181–365 days post-transplant category because the graft was lost after six months. This reinforces the need for adherence counseling not only at discharge but repeatedly during follow-up visits, especially during the first year after transplantation.

Proteinuria and increased serum creatinine are important markers of graft injury. In the present study, proteinuria was documented in 11.0% of recipients and serum creatinine was >1.5 mg/dL in 8.0% of recipients. The graft-loss patient was clinically placed in the proteinuria-positive and raised-creatinine categories because necrotizing glomerulonephritis/ nephropathy and graft dysfunction would be expected to present with urinary protein loss and impaired renal function. Recent literature supports this interpretation. Seeman et al. reported that post-transplant proteinuria is associated with an increased risk of graft loss, even at low levels (15). Pinto-Ramirez et al. also found that serum creatinine was a significant risk factor for graft loss among kidney transplant recipients (16). Therefore, the presence of proteinuria and raised creatinine in any post-transplant patient should prompt careful evaluation for rejection, recurrent or de novo glomerular disease, infection, drug toxicity and adherence-related injury.

The present study did not document ABMR, TCMR, IF/TA, infection, recurrent disease or vascular disease as positive causes of graft loss. However, the absence of these determinants in this dataset should be interpreted carefully because missing documentation was present for several cause-specific variables. Antibody-mediated rejection remains one of the major causes of graft failure internationally. Böhmig et al. described antibody-mediated rejection as a major cause of graft failure, with ongoing challenges in treatment standardization (17). Gatault et al. also emphasized that ABMR is a leading cause of graft failure and remains difficult to manage compared with T-cell-mediated rejection (18). Similarly, Hassanein et al. discussed chronic rejection as an important pathway leading to long-term allograft injury and failure (19). These studies show that rejection-related variables should be documented carefully in transplant records even when the observed number of graft-loss events is small.

Infective causes of graft dysfunction also remain relevant in renal transplant recipients. Although infection was not documented as a positive cause of graft loss in this cohort, international studies show that BK virus nephropathy and other post-transplant infections can contribute to graft injury. Pinto-Ramirez et al. identified BK virus nephropathy as a significant risk factor for graft loss (16). Gras et al. reported that BK virus nephropathy remains a severe complication in kidney transplant recipients and is associated with increased risk of acute rejection and return to dialysis (9). Lorant et al. also reported that BK virus-associated nephropathy may occur in up to 10% of kidney transplant recipients and is linked with graft dysfunction or graft loss (20). Therefore, future datasets should include BK virus screening, CMV status, infection episodes and biopsy findings where available.

The predominance of living related and spousal donors in the present study is consistent with transplant patterns in many resource-limited settings, including Pakistan, where deceased donor transplantation remains limited. Living donor transplantation is generally associated with favorable outcomes due to planned surgery, shorter ischemia time and better donor evaluation. Murray et al., in a large UK analysis, evaluated the effect of living donor transplantation on graft survival and supported the favorable role of living donor kidneys in transplant outcomes (8). In Pakistan, Attiq et al. reviewed the first 500 renal transplants at Pakistan Kidney and Liver Institute and reported transplant outcomes using graft survival, patient survival and 12-month creatinine values (21). Asghar et al. also reviewed renal transplant services in Punjab and showed that kidney transplant services are concentrated in selected districts, highlighting the need for improved access and structured transplant follow-up systems (22). These local findings support the importance of strengthening transplant registries and long-term monitoring systems in Pakistan.

Diabetes was present in 11.0% of recipients in the current study, while the graft-loss patient was placed in the non-diabetic group because the documented cause was medication non-adherence followed by necrotizingglomerulonephritis/nephropathy.
However, diabetes remains an important comorbidity in transplant medicine and can influence patient survival, cardiovascular risk and long-term graft outcomes. García-Padilla et al. compared diabetic and non-diabetic kidney transplant recipients and reported lower overall survival in diabetic patients, although

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graft survival differences may vary depending on population and follow-up (23). Sanchez-Baya et al. also discussed diabetes mellitus in kidney transplant recipients and its relevance to long-term outcomes (24). Therefore, although diabetes was not linked to the single graft-loss event in this study, it remains an important variable for surveillance in larger cohorts.

The main limitation of the present study is the very small number of outcome events. Since only one graft-loss case was recorded, statistical testing for associations with age, gender, diabetes, proteinuria, serum creatinine and time after transplantation was not appropriate. The subgroup tables should therefore be interpreted descriptively. Another limitation is the retrospective record-based design, where missing documentation may affect interpretation of rejection type, biopsy findings, infection status, drug levels and exact histopathological confirmation. Despite these limitations, the study provides an important clinical message: graft loss was uncommon in this cohort, but the only documented graft-loss case was related to a preventable factor, namely medication non-adherence.

IMPLICATIONS

The findings of this study have important clinical and institutional implications. First, every renal transplant recipient should receive repeated counseling about strict adherence to immunosuppressive medication, especially during the first post-transplant year. Second, transplant follow-up should include structured monitoring of serum creatinine, proteinuria, drug levels, infection screening and biopsy findings where indicated. Third, any missed dose, irregular follow-up or financial difficulty in purchasing immunosuppressive medicines should be documented and addressed early. Fourth, hospitals should maintain a transplant registry with standardized variables for rejection, ABMR, TCMR, IF/TA, infection, recurrent disease, medication non-adherence and graft outcome. Finally, because the only graft-loss event in this study was related to non-adherence, patient education, family involvement, reminder systems and affordable access to immunosuppressive drugs may help reduce preventable graft loss.

CONCLUSION

In conclusion, graft loss was documented in one out of 100 renal transplant recipients, giving a frequency of 1.0%. The graft was lost six months after transplantation due to non-adherence to immunosuppressive medication, followed by necrotizing glomerulonephritis/nephropathy. Most recipients had preserved graft status during the observed follow-up period. The presence of only one graft-loss event limited statistical assessment of determinants; therefore, subgroup findings were interpreted descriptively. The study highlights that even a low frequency of graft loss is clinically important when the cause is preventable. Strict medication adherence, regular follow-up, serum creatinine monitoring, proteinuria assessment and complete documentation of rejection and histopathological findings are essential to improve long-term renal graft survival..

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