ORIGINAL ARTICLE

Reversal of graft dysfunction following treatment of urinary tract infections in renal transplant recipients

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ABSTRACT

Background: UTI is one of the most common complications of renal transplantation. The aim of the study is to assess the reversal of graft malfunction following the management of urinary tract infection in kidney transplant patients.

Material and Methods: This was a prospective observational cohort study done at the Department of Transplant Nephrology, Sindh Institute of Urology and Transplantation. Non-probability consecutive sampling was used to select a total of 70 renal transplant recipients aged between 18 - 65 years with UTI and graft dysfunction. Graft dysfunction reversal was measured at 6 weeks of antibiotic treatment and was divided into complete, partial, and no reversal. The analysis of data was performed by SPSS 22.0 with $p<0.05$ as significant value.

Results: Graft dysfunction reversal was complete in 38 out of 70 patients (54.3%), partial in 20 out of 70 (28.6%), and absent in 12 out of 70 (17.1%). On multivariable logistic regression analysis, diabetes mellitus (AOR = 0.38, p = 0.041), hypertension (AOR = 0.42, p = 0.048), high baseline creatinine ≥2 mg/dL (AOR = 0.31, p = 0.023), and infection with multidrug-resistant organisms (AOR = 0.56, p = 0.019) were independently associated with decreased odds of complete reversal. The most frequent pathogens were *Escherichia coli* (32/70, 45.7%) and *Klebsiella pneumoniae* (18/70, 25.7%).

Conclusions: Graft dysfunction related to UTI is reversible in a high percentage of renal transplant recipients. Comorbidities, impaired baseline renal functions, and multidrug-resistant infections have a negative impact on recovery.

Keywords: Creatinine; Drug Resistance, Graft Dysfunction, Kidney Transplantation.

BACKGROUND

UTIs are common infectious complications after kidney transplantation and are an important cause of patient morbidity and graft-related adverse outcomes.[1] Immunosuppression, prolonged indwelling urinary catheterization, ureteric stenting and repeated urological procedures make kidney transplant recipients vulnerable to recurrent and complicated UTIs.[2] UTIs remain a longstanding challenge in clinical practice worldwide, despite major advances in transplant care and infection control measures.[3]

Post-transplant UTI is a significant cause of post-transplant hospital admissions, which have been reported in almost 20–60% of kidney transplant recipients (KTRs) internationally, and are associated with rising concerns about antimicrobial resistance and recurrent infection in KTRs.[4] Regional studies in South Asia, including Pakistan, have reported a meaningful burden of post-transplant UTIs, with increased concerns about antimicrobial resistance and recurrent infection observed in KTRs.[5] These infections contribute to higher healthcare costs and increased hospitalization rates for KTRs, and may have a negative impact on long-term survival of the graft and patient.[6]

Although transplant care has advanced, there are still a number of controversies. These involve the clinical importance and treatment of asymptomatic bacteriuria, Optimal antibiotic duration and the selection of the preferred treatment, as well as the long-term effects of repeated or late-onset infection on graft survival. [7] Certain studies indicate that recurrent UTI is a major cause of graft dysfunction and hospitalization whereas others report mixed results regarding the association changes with early diagnosis and prompt intervention. [8] Also, the contribution of asymptomatic infections is controversial, and it is not unanimously agreed that regular treatment of grafts can lead to a better result. [9]

The intensity of infections with multidrug-resistant organisms including Klebsiella pneumoniae that are correlated with worse clinical outcomes and higher risk of graft impairment is another critical issue of concern.

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[10] These issues imply the necessity of additional studies to explain the connection between UTIs and graft functioning with an emphasis on the possibility to reverse graft dysfunction under the condition of proper treatment. Hence, the purpose of the study is to assess the reversal of graft dysfunction following the management of urinary infection in kidney transplant patients.

MATERIAL AND METHODS

The research study was a prospective observational cohort study carried out in the Department of Transplant Nephrology at Sindh Institute of Urology and Transplantation within a period of six months following the approval of the research synopsis and ethical clearance by the Research Ethics Committees of CPSP and SIUT (Approval No: SIUT-ERC-2025/A-528 dated 28th January 2025), from 1st February 2025 till 30th July 2025. Every subject gave informed consent before being enrolled. The study population was renal transplant recipients who presented with urinary tract infection (UTI) and graft dysfunction, as specified in the operational criteria. The operational definition and clinical setting of UTI and graft dysfunction were modified based on the existing literature on kidney transplant recipients.[1, 3]

The WHO sample size calculator was used to calculate the sample size with a reference standard deviation (σ) of 6.23, which was based on the previously published literature that assessed change of serum creatinine levels following treatment of urinary tract infection in patients of a renal transplant. The precision (d) that was used was 1.5 units and the confidence level was 95%.[7]

Non-probability consecutive sampling method was used and all qualified patients who met the inclusion criteria were recruited in a sequence until the sample size was attained.

The criteria used were that the participants had to be male and female aged between 18 and 65 years and had UTI and graft dysfunction with an estimated glomerular filtration rate of greater than 30 ml/min before the infection. Patients were not eligible when they had any evidence of graft rejection or a history of rejection within 30 days of UTI, a pre-existing growing trend of serum creatinine before UTI, co-infection at other sites, or an eGFR of less than 30 ml/min before infection.

Data was collected using a structured proforma, which included demographics, comorbidities, variables relating to transplant, length of time since transplant, UTI history, immunosuppressive therapy, previous antibiotic exposure, length of time on antibiotics, and vital signs. These variables were incorporated because of their possible impact on the outcome of the graft and the severity of the infection.

At the time of admission, laboratory tests such as complete blood count (CBC), urinalysis, urine culture, serum urea, creatinine, electrolytes (sodium, potassium, chloride), calcium, and phosphorus were done. Serum creatinine was also measured at the end of the antibiotic treatment and at the end of 6 weeks of treatment to determine graft function. Analysis of CBC was done in Sysmex XN-1000 and Beckman Coulter UniCel DxH 800, whilst biochemical parameters were analyzed in Beckman Coulter AU5800. The urinalysis was done with Cobas U411 and DIRUI FUS 2000. In case of urine culture, the samples were inoculated on the cysteine lactose electrolyte-deficient agar with a calibrated loop and allowed to incubate at 36 ^{o}C, over a period of 48 hours. Microorganisms were identified based on standard microbiological guidelines, and the antibiotic susceptibility was conducted on the basis of the Kirby-Bauer disc diffusion technique based on the standard parameters. The Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula was used to estimate the glomerular filtration rate. Renal biopsy was conducted when clinically necessary and graft pyelonephritis was determined based on a histopathological examination. Graft dysfunction was operationally defined as an increase in serum creatinine of more than 50% above baseline.

Urinary tract infection was considered a positive urine culture with growth of over 10^5 CFU/ml in addition to one or more of the following: fever, urinary symptoms including dysuria, frequency or turbid urine, or leukocytosis, or biopsy evidence of graft pyelonephritis which was neutrophil-dominant inflammatory infiltrate in the interstitium with tubular micro abscesses. Renal failure was considered to be impairment of renal activity, which was indicated by increased serum creatinine by over 50 percent over the baseline in which the baseline creatinine was considered to be average of two levels of creatinine before UTI. Graft dysfunction reversal was assessed 6 weeks after initiation of antibiotic therapy. Outcomes were classified as complete reversal when serum creatinine returned to within 20% of the pre-infection baseline value. Partial reversal was defined as a reduction in serum creatinine

of 20% to <50% from the peak level during illness. No reversal was defined as a reduction in serum creatinine of <20% from the peak level after treatment.[20]

Data were analyzed using IBM SPSS Statistics v.22. Mean ± SD was calculated for age, BMI, baseline serum creatinine, and post-treatment serum creatinine. Median (IQR) was used for duration of transplant, symptoms, and hospital stay. Frequencies and percentages were computed for gender, comorbidities, type of UTI, and reversal of graft dysfunction. A paired t-test was applied to compare pre- and post-treatment serum creatinine to assess reversal of graft dysfunction. An independent (two-sample) t-test was used to compare mean creatinine between reversal vs. non-reversal groups. The Chi-square test assessed associations between reversal of graft dysfunction and categorical variables (gender, comorbidities, infection type). Stratification was done for age, gender, BMI, and comorbidities to control confounders. A p-value ≤ 0.05 was considered significant. Bias was minimized through uniform eligibility criteria and standardized data collection.

RESULTS

Total of 70 renal transplant recipients were studied with urinary tract infection (UTI) and graft dysfunction. Demographic and clinical characteristics at baseline are shown in Table-I. The average age of the study population was 42.4 ± 11.8 years. There were 42 (60.0%) males and 28 (40.0%) females. 41 patients (58.6%) had hypertension and 24 (34.3%) had diabetes mellitus. The mean BMI was 26.1 ± 3.8 kg/m², and the median baseline serum creatinine was 2.1 mg/dL (IQR: 1.7–2.8).

Reversal of graft dysfunction was evaluated after antibiotic treatment, at 6 weeks. A complete reversal was seen in 38 (54.3%) patients, partial reversal was seen in 20 (28.6%) patients and no reversal was seen in 12 (17.1%) patients as shown in Table-II.

Before and after treatment comparison of renal function revealed that there was a significant improvement in serum creatinine level. The mean serum creatinine decreased from 2.48 ± 0.91 mg/dL at presentation to 1.82 ± 0.74 mg/dL at six weeks post-treatment ($p < 0.001$), as shown in Table-III.

In the multivariable logistic regression model, diabetes mellitus, high baseline creatinine, and infection with a multidrug-resistant (MDR) organism were independently associated with complete reversal. Patients with diabetes mellitus had significantly lower odds of complete reversal (AOR = 0.38, 95% CI: 0.14–0.98, p = 0.041). Similarly, high baseline creatinine (≥2 mg/dL) was associated with reduced odds of complete reversal (AOR = 0.31, 95% CI: 0.11–0.85, p = 0.023), as was the presence of an MDR organism (AOR = 0.56, 95% CI: 0.34–0.91, p = 0.019).

Hypertension showed a trend toward reduced odds of complete reversal, but this association did not reach statistical significance (AOR = 0.42, 95% CI: 0.16–1.05, p = 0.067). Age greater than 45 years (AOR = 0.79, 95% CI: 0.32–1.94, p = 0.609) and male gender (AOR = 1.12, 95% CI: 0.45–2.78, p = 0.808) were not significantly associated with the outcome Table-IV.

Microbiological results revealed that Escherichia coli (45.7%) was most frequently isolated followed by Klebsiella pneumonia (25.7%), Pseudomonas aeruginosa (11.4%), Enterococcus species (10.0%) and Acinetobacter species (7.1%). Overall, 24 (34.3%) isolates were MDR Table-V.

Table-I: Baseline demographic and clinical characteristics (n = 70).

Variable	N(%)/ Mean ± S.D/ median (IQR)
Age (years)	42.4 ± 11.8
Male	42 (60.0%)
Female	28 (40.0%)
Hypertension	41 (58.6%)
Diabetes Mellitus	24 (34.3%)
BMI (kg/m²)	26.1 ± 3.8
Baseline Creatinine (mg/dL)	2.1 (1.7–2.8)

Table-II: Reversal of graft dysfunction at 6 weeks (n = 70).

Outcome	Frequency (%)
Complete Reversal	38 (54.3%)
Partial Reversal	20 (28.6%)
No Reversal	12 (17.1%)

Table-III: Serum creatinine before and after treatment (n = 70).

Parameter	Mean ± SD	p-value
Pre-treatment Creatinine (mg/dL)	2.48 ± 0.91	
Post-treatment Creatinine (mg/dL)	1.82 ± 0.74	<0.001

Table-IV: Multivariable Logistic Regression for Complete Reversal

Variable	AOR	95% CI	p-value
Diabetes Mellitus	0.38	0.14–0.98	0.041
Hypertension	0.42	0.17–0.99	0.048
High Baseline Creatinine (≥2 mg/dL)	0.31	0.11–0.85	0.023
MDR Organism	0.56	0.34–0.91	0.019
Age >45 years	0.79	0.32–1.94	0.609
Male Gender	1.12	0.45–2.78	0.808

Table-V: Microorganisms isolated and antimicrobial resistance pattern.

Organism	Frequency (%)	MDR n (%)	Resistance Pattern	Sensitive Antibiotics
E. coli	32 (45.7%)	11 (34.4%)	Ciprofloxacin, Ceftriaxone, Cotrimoxazole	Meropenem, Amikacin
K. pneumoniae	18 (25.7%)	7 (38.9%)	Ceftriaxone, Piperacillin-Tazobactam	Meropenem, Colistin
P. aeruginosa	8 (11.4%)	3 (37.5%)	Ceftazidime, Ciprofloxacin	Colistin, Meropenem
Enterococcus spp.	7 (10.0%)	2 (28.6%)	Ampicillin, Ciprofloxacin	Vancomycin, Linezolid
Acinetobacter spp.	5 (7.1%)	1 (20.0%)	Cefepime, Ciprofloxacin	Colistin, Tigecycline

DISCUSSION

The present study showed that, in patients with renal transplant graft dysfunction associated with urinary tract infection (UTI), more than half of these patients (54.3%) had complete recovery of graft dysfunction after antibiotic therapy, and 28.6% had partial recovery of graft dysfunction and 17.1% showed no improvement in graft dysfunction after antibiotic therapy. The results suggest that graft dysfunction due to UTI is mostly reversible, but there is a significant rate of patients who did not get completely back to normal renal function even after receiving appropriate treatment.[11]

The complete recovery rate observed in our study is similar to some earlier studies in renal transplant recipients, where recovery rates between 45 to 65% have been reported, depending on the time of therapy, the baseline graft function and the severity of infection. Other studies have revealed that early detection and early treatment with the right antimicrobial treatment is related to the better recovery of the graft, while the late treatment is associated with incomplete recovery and persistence of graft dysfunction.[12] This is in line with our observation that a substantial part of patients is not completely reversed despite treatment.

Diabetes mellitus and hypertension were significantly associated with reduced chances of complete remission of graft function in the current study. Further, high baseline serum creatinine (BSC) ≥2mg/dl was an independent predictor of poor recovery. These results indicated that pre-existing metabolic and vascular comorbidities have a negative impact on renal recovery following infectious injury. The same applies to transplant recipients, in whom diabetes and hypertension lead to suboptimal immune function and diminished renal reserve, along with delayed recovery from infection-induced damage.[13]

In addition, complete recovery was also significantly associated with being not infected with multidrug-resistant (MDR) organisms. This emphasizes the importance of antimicrobial resistance in predicting graft outcome. MDR infections can cause a delay in the initiation of appropriate therapy, persistence of bacteria and greater inflammatory damage to the transplanted kidney, with the end result being incomplete recovery of graft function.[14]

In our study, microbiological analysis showed that Escherichia coli (45.7%) was the most frequently isolated microorganism followed by Klebsiella pneumoniae (25.7%), Pseudomonas aeruginosa (11.4%), Enterococcus species (10.0%) and Acinetobacter species (7.1%). The results of this study are in keeping with earlier studies on renal transplant recipients, all of which have pointed to Gram-negative pathogens as the major cause of post-transplant UTIs.[15] Our cohort is similar to previous studies suggesting that E. coli and K. pneumoniae were the most common among transplant recipients. The relatively high proportion of MDR (34.3%) isolates found in this study is however worrisome and mirrors the global rise in antimicrobial resistance in immunocompromised patients. This is consistent with previously published

data [16] that shows an increase in antimicrobial resistance, creating a therapeutic challenge.

Our study found that the resistance profile was similar to other reports from the region and the rest of the world, with high resistance to ciprofloxacin and ceftriaxone but not to the carbapenems or to colistin. This highlights the need for local antibiogram-guided therapy in transplant recipients to make sure that appropriate empirical antibiotic therapy is given.[17]

In our study, importantly, MDR organisms were independently associated with poorer graft recovery. This has been supported by previous studies showing that antimicrobial resistance leads to delayed recovery from infection, treatment failure, and longer inflammatory periods in the graft, all of which adversely affect renal recovery.[18]

This study's main findings highlight that, despite being reversible in many cases, outcomes of graft dysfunction associated with UTI are strongly dependent on baseline patient characteristics, comorbid conditions and microbial resistance patterns. Early detection, early targeted therapy and antimicrobial stewardship are still key to ensure optimal graft function and to prevent long-term graft dysfunction.[19]

The main strength of this study is that it is a concentrated assessment of graft dysfunction reversal in kidney transplant recipients with well-known outcomes and analysis of clinically relevant predictors including comorbidities, baseline creatinine, and multidrug-resistant organisms. Its clinical applicability is also increased by the addition of microbiological profiles. The research is however constrained by small size of sample used and a one center design which might have some implications on generalization. There is a limitation to the short follow-up period to evaluate the long-term graft and the observational nature prevents the determination of causality. Also, the possible confounders, including immunosuppressive treatments and compliance, were not fully examined.

CONCLUSION

UTIs are a serious, potentially reversible, cause of graft dysfunction in patients of renal transplants. Over half of the patients in this study returned to full recovery after proper antibiotic treatment; a significant number of patients were only partially or not reverted. The most significant issues related to worse outcomes were diabetes mellitus, hypertension, a high baseline creatinine and infection by multidrug-resistant organisms. The results of these studies indicate a crucial role of host-related and microbiological factors on graft recovery, and prompt diagnosis and specific management.

RECOMMENDATIONS

UTI in renal transplant recipients should be treated early and with culture-based antibiotic therapy in order to enhance the graft outcomes. High-risk patients and especially diabetics, hypertension, and patients with impaired baseline renal function should be screened on a regular basis. The developing burden of multidrug-resistant organisms must be overcome by strengthening the antimicrobial stewardship programs. Furthermore, it is suggested that the findings should be supported by multicenter studies that will involve larger samples and a longer follow-up time to determine the role of immunosuppressive regimens and preventive measures in maximizing graft survival.

CONFLICT OF INTEREST

None

GRANT SUPPORT & FINANCIAL DISCLOSURE

Declared none

AUTHOR CONTRIBUTION

Mehak Abro: Substantial contribution to study design and data acquisition, manuscript drafting and critical review, final approval of the version to be published

Ejaz Ahmed: Supervision of study design and methodology, critical manuscript review, final approval of the version to be published

Ranjeet Kumar: Analysis and interpretation of data, critical manuscript review, final approval of the version to be published.

Imran Ali: Contribution to concept and study design, critical manuscript review, final approval of the version to be published

Sidra German: Data acquisition and management, manuscript drafting and review, final approval of the version to be published.

Huda Narejo: Data collection and preliminary analysis, manuscript drafting and review, final approval of the version to be published.

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