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Evaluation of the Prevalence of Acute Rejection in Post-Kidney Transplant Patients with Acute Tubular Necrosis in Nemazee Hospital During 2010 - 2016

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Abstract

Background: Delayed graft function (DGF), defined as the need for dialysis within the first postoperative week, is a common complication after kidney transplantation and is associated with reduced graft survival. Acute tubular necrosis (ATN) and acute rejection are the main underlying causes; however, distinguishing between them is essential for appropriate management.

Objectives: This study aimed to determine the histopathological prevalence of ATN and acute rejection in kidney transplant recipients with DGF and to identify the associated immune and nonimmune risk factors.

Methods: In this retrospective observational study, 195 adult kidney transplant recipients with DGF were evaluated at a single center between 2010 and 2016. All patients underwent allograft biopsy. Donor and recipient characteristics, induction immunosuppression, and clinical variables were analyzed. Patients were categorized according to biopsy findings as having isolated ATN, isolated acute rejection, or combined ATN and rejection.

Results: Acute tubular necrosis was identified in 155 patients (79.5%), whereas acute rejection was present in 86 patients (44.1%). Concurrent ATN and rejection occurred in 55 patients (28.2%). Isolated ATN and isolated rejection were observed in 100 patients (51.3%) and 31 patients (15.9%), respectively. Biopsy findings were significantly associated with several factors. Donor cause of death differed significantly between the isolated ATN and isolated rejection groups (P = 0.01). Recipient sex distribution also differed significantly among groups: ATN versus rejection (P = 0.03), ATN versus ATN plus rejection (P = 0.01), and rejection versus ATN plus rejection (P = 0.001). In addition, the induction immunosuppressive regimen differed significantly across all pairwise comparisons: ATN versus rejection (P = 0.03), ATN versus ATN plus rejection (P = 0.002), and rejection versus ATN plus rejection (P = 0.009).

Conclusions: Acute tubular necrosis is the predominant histopathological finding in kidney transplant recipients with DGF; however, acute rejection, either alone or concomitant with ATN, remains common. These findings underscore the critical role of an early allograft biopsy in guiding targeted therapy and optimizing transplant outcomes.

Keywords: Kidney Transplantation, Graft Rejection, Delayed Graft Function, Acute Kidney Injury, Allografts

1. Background

Chronic kidney disease (CKD) affects more than 500 million individuals worldwide, representing approximately 10% of the adult population. More than 1.5 million people rely on renal replacement therapies, such as dialysis or transplantation, to survive (1). Kidney transplantation remains the preferred treatment for patients with end-stage renal disease (ESRD), offering superior quality of life and long-term survival compared with dialysis (2). Global estimates suggest that more than 1.4 million patients with CKD have undergone

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kidney transplantation, with an annual growth rate of 8% (3). In addition to recent advances in surgical techniques and pharmacotherapeutic protocols for renal transplantation, perioperative fluid and acid-base management plays a critical role in ensuring adequate graft perfusion and optimizing clinical outcomes. Furthermore, educational interventions have significantly improved all domains of health-related quality of life and reduced the incidence of acute post-transplant complications, including infection, acute rejection, and hospital readmission, among patients undergoing kidney transplantation (4, 5). Despite these therapeutic advances and improvements in graft survival over recent decades, acute rejection (AR) and DGF remain major obstacles to successful transplantation outcomes (6). Acute rejection, an immune-mediated response against the transplanted kidney, has decreased in prevalence but continues to compromise graft longevity (7). Delayed graft function, which is typically observed after deceased-donor transplantation, is defined by the requirement for dialysis within the first postoperative week and is frequently associated with ischemia-reperfusion injury leading to ATN (8). This condition not only increases the risk of AR but also negatively affects graft and patient survival (9-11).

The incidence of DGF varies according to donor type and immunosuppression regimen, ranging from less than 5% in living-donor transplants to more than 80% in non-heart-beating donors (12, 13). Despite medical advances, DGF rates have remained relatively unchanged, partly because of the increasing use of marginal donors, including expanded-criteria donors and donors after cardiac death (14). Delayed graft function presents both diagnostic and therapeutic challenges because its causes are diverse and include immunologic rejection, surgical complications, drug toxicity, recurrent disease, and ATN (8).

2. Objectives

Given the clinical significance and multifactorial etiology of DGF, timely differentiation among its causes is essential for effective management. This study aimed to determine the prevalence of ATN and acute rejection in kidney transplant recipients with DGF and to identify associated immune and nonimmune risk factors.

3. Methods

3.1. Study Design and Population

This retrospective observational study included adult patients who underwent kidney transplantation at our center between 2010 and 2016. Using a census sampling method, we reviewed the medical records of kidney donors and recipients. The inclusion criteria were as follows: 1) recipient age ≥ 18 years; 2) clinically diagnosed DGF, in accordance with the aforementioned definition, followed by allograft biopsy; and 3) availability of complete medical records for donor and recipient variables. Patients were excluded if they had 1) a primary nonfunctioning graft, defined as a graft that never achieved sufficient function to allow discontinuation of dialysis; 2) death due to non-transplant-related causes, such as accidental death or unrelated malignancy, occurring before graft biopsy or within the first postoperative week; or 3) an inadequate biopsy specimen, defined as a biopsy containing fewer than 7 glomeruli or no arterial cross-section.

3.2. Definitions and Data Collection

All cases of DGF were confirmed by histopathological evaluation of allograft biopsy specimens. Biopsy findings were categorized as ATN, acute rejection according to the diagnostic criteria of the Banff 2017 Kidney Meeting Report, both ATN and rejection, or the absence of both conditions. The following variables were extracted from patient records: recipient and donor age, sex, body mass index (BMI), blood group and Rh status, underlying kidney disease, induction immunosuppressive regimen, donor cause of death, and serum creatinine levels.

3.3. Statistical Analysis

Data were analyzed using SPSS software version 17.0 (SPSS Inc., Chicago, IL, USA). Normality of distribution was assessed using the Kolmogorov-Smirnov test and visual inspection of histograms. Descriptive statistics were reported as mean ± standard deviation (SD) for continuous variables and frequency (percentage) for categorical variables. Group comparisons were performed using the Pearson chi-square test or Fisher exact test, as appropriate, for categorical variables and the independent t-test for continuous variables. A P value of < 0.05 was considered statistically significant.

4. Results

A total of 203 kidney transplant recipients who underwent allograft biopsy due to DGF were initially included. However, 8 patients were excluded because of inadequate biopsy specimens, resulting in a final study population of 195 patients. The mean recipient age was

42.46 ± 14.21 years (range, 19 - 75 years), including 105 males (53.8%) and 90 females (46.2%). Recipient baseline characteristics are summarized in Table 1.

Table 1. Baseline Characteristics of Recipients

Variables	Recipients (n = 195)
Recipient age (y)	42.46 ± 14.21
Recipient sex	
Male	105 (53.8)
Female	90 (46.2)
Recipient BMI (kg/m^2)	21.74 ± 4.81
Recipient blood group	
A	66 (33.8)
B	49 (25.1)
O	67 (34.4)
AB	13 (6.7)
Recipient Rh status	
Positive	183 (93.8)
Negative	12 (6.2)
Underlying kidney disease	
Hypertension	41 (21.0)
Diabetes mellitus	29 (14.9)
Polycystic kidney disease	9 (4.6)
Renal stone	11 (5.6)
Glomerulonephritis	18 (9.2)
Unknown	85 (29.7)
Other	29 (14.9)
Induction regimen	
None	45 (23.1)
Thymoglobulin	126 (64.6)
Alemtuzumab	8 (4.1)
Basiliximab	3 (1.5)
Daclizumab	13 (6.7)

Donors had a mean age of 38.67 ± 13.84 years (range, 18 - 80 years), including 110 males (56.4%) and 85 females (43.6%). The primary causes of donor death were trauma (43.1%), intracranial hemorrhage (19.5%), cardiac events (12.3%), and other causes (25.1%). The mean serum creatinine levels on the first and third post-transplant days were 3.71 ± 3.72 mg/dL and 6.26 ± 3.27 mg/dL, respectively. Donor baseline characteristics are presented in Table 2.

Table 2. Baseline Characteristics of Donors

Variables	Donors (n = 195)
Donor age (y)	38.67 ± 13.84
Donor sex	
Male	110 (56.4)
Female	85 (43.6)
Donor blood group	
A	65 (33.3)
B	52 (26.7)
O	66 (33.8)
AB	12 (6.2)
Donor Rh status	
Positive	184 (94.4)
Negative	11 (5.6)
First creatinine level (mg/dL)	3.71 ± 3.72
Third creatinine level (mg/dL)	6.26 ± 3.27
Cause of death	
Trauma	84 (43.1)
Intracranial hemorrhage	38 (19.5)
Cardiac events	24 (12.3)
Other	49 (25.1)

Histopathological evaluation of kidney biopsies revealed ATN in 155 patients (79.5%) and rejection in 86 patients (44.1%). Among these patients, 55 (28.2%) showed evidence of both ATN and rejection, 100 (51.3%) had isolated ATN, and 31 (15.9%) had isolated rejection. Notably, 9 patients (4.6%) had neither ATN nor rejection. The distribution of biopsy findings is illustrated in Figure 1.

A total of 186 kidney transplant recipients were categorized into 3 groups: patients with ATN (n = 100), patients with graft rejection (n = 31), and patients with both conditions (ATN plus rejection; n = 55). Further analyses compared clinical and donor-related variables among these categories. Donor age was comparable across all groups ($P > 0.05$). However, donor sex distribution differed significantly, with the ATN plus rejection group having a higher proportion of female donors (60%) than the ATN group (41%; $P = 0.02$) and rejection group (29.1%; $P = 0.006$). The cause of donor death also differed significantly between the ATN and rejection groups ($P = 0.01$); trauma was most frequent in the ATN group (45%), whereas intracranial hemorrhage was most common in the rejection group (41.9%).

Recipient age and BMI were similar across groups. In contrast, recipient sex distribution varied substantially, with P values of 0.03 for the ATN versus rejection comparison, 0.01 for the ATN versus ATN plus rejection comparison, and 0.001 for the rejection versus ATN plus rejection comparison. The rejection group was predominantly male (77.4%), whereas the ATN plus rejection group was predominantly female (65.5%). The distribution of recipients' underlying kidney diseases did not differ significantly between groups ($P > 0.05$). Clinically, the first recorded creatinine level was significantly higher in the ATN plus rejection group (5.03 ± 3.74 mg/dL) than in the rejection group (1.76 ± 1.55 mg/dL; $P = 0.001$). The level in the ATN group (3.89 ± 4.10

Table 3. Comparison of Immune and Nonimmune Characteristics Between Patients with ATN and Rejection in Biopsy Results

Variables	ATN group (n = 100)	Rejection group (n = 31)	ATN + rejection group (n = 55)	ATN vs rejection P value	ATN vs ATN + rejection P value	Rejection vs ATN + rejection P value
Donor age (y)	38 ± 13.88	41.64 ± 14.35	38.38 ± 13.94	0.21	0.87	0.31
Donor sex				0.29	0.02	0.006
Male	59 (57.3)	22 (21.4)	22 (21.4)			
Female	41 (49.4)	9 (10.8)	33 (39.8)			
First creatinine level (mg/dL)	3.89 ± 4.10	1.76 ± 1.55	5.03 ± 3.74	0.006	0.09	0.001
Third creatinine level (mg/dL)	6.78 ± 3.77	6.05 ± 2.95	5.64 ± 2.56	0.71	0.17	0.77
Cause of death				0.01	0.87	0.92
Trauma	45 (57.0)	10 (12.7)	24 (30.4)			
Intracranial hemorrhage	17 (47.2)	13 (36.1)	6 (16.7)			
Cardiac events	11 (45.8)	0	13 (54.2)			
Other	27 (57.4)	8 (17.0)	12 (25.6)			
Recipient age (y)	41.81 ± 13.52	46.32 ± 15.38	41.61 ± 14.78	0.11	0.93	0.16
Recipient sex				0.03	0.01	0.001
Male	56 (56.6)	24 (24.2)	19 (19.2)			
Female	44 (50.6)	7 (8.0)	36 (41.4)			
Recipient BMI (kg/m^2)	21.21 ± 5.62	23 ± 6.23	22.03 ± 4.36	0.76	0.59	0.83
Underlying kidney disease				0.06	0.47	0.94
Hypertension	20 (48.8)	4 (9.8)	17 (41.4)			
Diabetes mellitus	13 (46.4)	8 (28.6)	7 (25.0)			
Polycystic kidney disease	3 (37.5)	5 (62.5)	0			
Renal stone	5 (50.0)	1 (10.0)	4 (40.0)			
Glomerulonephritis	13 (72.2)	2 (11.1)	3 (16.7)			
Unknown	32 (58.2)	9 (16.4)	14 (25.4)			
Other	14 (53.8)	2 (7.7)	10 (38.5)			
Induction regimen				0.03	0.002	0.009
None	17 (37.8)	1 (2.2)	27 (60.0)			
Thymoglobulin	68 (58.1)	30 (25.6)	19 (16.2)			
Alemtuzumab	4 (50.0)	0	4 (50.0)			
Basiliximab	2 (66.7)	0	1 (33.3)			
Daclizumab	9 (69.2)	0	4 (30.8)			

was found in third creatinine levels. Significant differences were also observed in the use of induction immunosuppressive regimens (P = 0.03 for ATN vs rejection, P = 0.002 for ATN vs ATN plus rejection, and P = 0.009 for rejection vs ATN plus rejection). Thymoglobulin was used most frequently in the rejection (96.7%) and ATN (68%) groups, whereas nearly half of the ATN plus rejection group (49.1%) received no induction therapy (Table 3).

5. Discussion

Kidney transplantation remains the optimal treatment for patients with ESRD, providing substantial improvements in quality of life and survival compared with maintenance dialysis (15). However, despite advances in immunosuppressive regimens and postoperative care, acute renal allograft rejection remains a major clinical challenge that threatens long-term graft survival. This issue underscores the complexity of managing transplant recipients, who are inherently at risk because of both their underlying condition and the immunosuppressive therapies required to maintain graft viability (15).

This study highlights that, although the incidence of acute rejection has decreased substantially over the past few decades, particularly with the use of potent agents such as calcineurin inhibitors and mycophenolate mofetil, its presence, even when subclinical, can have lasting implications for graft function (15, 16). Notably, subclinical rejection, often identified through protocol

biopsies, may progress silently and result in interstitial fibrosis and tubular atrophy, ultimately contributing to chronic allograft nephropathy and death-censored graft loss (16).

One of the most pressing concerns is the apparent disconnect between decreased rates of acute rejection and the plateau in long-term graft survival. This paradox suggests that other factors, including overimmunosuppression, calcineurin inhibitor toxicity, and undetected or inadequately treated subclinical antibody-mediated rejection, may offset the benefits achieved through improved early rejection control (17). The distribution of induction immunosuppressive regimens differed among our study groups. The high use of thymoglobulin in the isolated rejection group reflects our center protocol for patients perceived to be at high immunological risk. Conversely, the finding that nearly half of the ATN plus rejection group received no induction therapy is striking. This observation raises the question of whether inadequate initial immunosuppression contributed to the development of rejection in patients who were also experiencing ischemic injury (ATN).

This study also emphasizes the importance of identifying and mitigating risk factors such as presensitization, human leukocyte antigen mismatches, donor quality, particularly extended-criteria donors, and DGF (18). Similarly, the findings of this manuscript suggest that donor sex was significantly associated with biopsy outcomes, with female donors overrepresented in the ATN plus rejection group. This aligns with some prior studies suggesting that donor-recipient sex mismatch, particularly female-to-male donation, may be associated with differential immunological risks, potentially mediated by minor histocompatibility antigens. Additionally, the difference in donor cause of death between the ATN and rejection groups, with more trauma in the ATN group and more intracranial hemorrhage in the rejection group, may relate to differences in donor management, inflammatory milieu, or organ quality, which could predispose grafts to different types of injury.

The complex pathophysiology of DGF, involving ischemia-reperfusion injury, inflammation, and immune activation, represents a multifactorial process that adversely affects post-transplant outcomes (19). Innovations in donor management and organ preservation, such as machine perfusion and hypothermic strategies, may help reduce its incidence in the future (20).

Furthermore, evolving tools for rejection surveillance, including noninvasive biomarkers such as donor-derived cell-free DNA, show promise for the early detection of rejection episodes and may offer alternatives to protocol biopsies (19). However, their predictive value remains modest, emphasizing the continued need for histological confirmation in many cases (21).

This study has several limitations. Its single-center retrospective design may limit generalizability. The sample size, particularly in the isolated rejection group (n = 31), reduces the statistical power of some comparisons. Another limitation is that our center exclusively uses deceased donors. Regrettably, data on cold ischemia time were unavailable.

5.1. Conclusions

In conclusion, although substantial progress has been made in reducing acute rejection rates, improving long-term kidney allograft survival will likely depend on more nuanced immunosuppression strategies, early identification of subclinical rejection, personalized risk assessment, and optimization of donor organ quality. Close collaboration between transplant centers and general nephrologists is essential for long-term success, particularly as care transitions from specialized to community settings.

Footnotes

AI Use Disclosure: For the purpose of Text Editing, the During The Preparation Of This Work The Authors Used Chatgpt In Order To Improve Language And Readability. After Using This Tool, The Authors Reviewed And Edited The Content As Needed And Take Full Responsibility For The Content Of The Publication. was used Minor in the Etc section.

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References

1. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. *Lancet*. 2013;**382**(9888):260-72. [PubMed ID: 23727169]. https://doi.org/10.1016/S0140-6736(13)60687-X.
2. Saatchi M, Poorolajal J, Amirzargar MA, Mahjub H, Esmailnasab N. Long-term survival rate of kidney graft and associated prognostic factors: a retrospective cohort study, 1994 - 2011. *Ann Transplant*. 2013;**18**:153-60. [PubMed ID: 23792515]. https://doi.org/10.12659/AOT.883873.
3. White SL, Chadban SJ, Jan S, Chapman JR, Cass A. How can we achieve global equity in provision of renal replacement therapy? *Bull World Health Organ*. 2008;**86**(3):229-37. [PubMed ID: 18368211]. [PubMed Central ID: PMC2647401]. https://doi.org/10.2471/BLT.07.041715.
4. Hashemian M, Ebadzadeh MR, Mohammadi R, Mirzaei M, Abdoli M, Mohajerani SA. Predictors of Post-operative ABG Changes to Pre-operative Levels in Kidney Transplant Patients, a Cross-Sectional Study. *Nephro-Urology Monthly*. 2024;**16**(4). https://doi.org/10.5812/numonthly-146899.
5. Matourypour P, Daliri N, Ghiyasvandian S, Zakerimoghadam M. Effects of an Educational Package on Quality of Life and Acute Complications in Kidney Transplant Recipients: A Clinical Trial. *Middle East Journal of Rehabilitation and Health Studies*. 2024;**11**(11). e140366. https://doi.org/10.5812/mejrh-129235.
6. Mikhalski D, Wissing KM, Ghisdal L, Broeders N, Touly M, Hoang AD, et al. Cold ischemia is a major determinant of acute rejection and renal graft survival in the modern era of immunosuppression. *Transplantation*. 2008;**85**(7 Suppl):S3-9. [PubMed ID: 18401260]. https://doi.org/10.1097/TP.0b013e318169c29e.
7. Shlipak MG, Fried LF, Stehman-Breen C, Siscovick D, Newman AB. Chronic renal insufficiency and cardiovascular events in the elderly: findings from the Cardiovascular Health Study. *Am J Geriatr Cardiol*. 2004;**13**(2):81-90. [PubMed ID: 15010654]. https://doi.org/10.1111/j.1076-7460.2004.02125.x.
8. Halloran PF, Hunsicker LG. Delayed graft function: state of the art, November 10 - 11, 2000. Summit meeting, Scottsdale, Arizona, USA. *Am J Transplant*. 2001;**1**(2):115-20. [PubMed ID: 12099358]. https://doi.org/10.1034/j.1600-6143.2001.10204.x.
9. Perico N, Cattaneo D, Sayegh MH, Remuzzi G. Delayed graft function in kidney transplantation. *Lancet*. 2004;**364**(9447):1814-27. [PubMed ID: 15541456]. https://doi.org/10.1016/S0140-6736(04)17406-0.
10. Szwarc I, Garrigue V, Delmas S, Deleuze S, Chong G, Mourad G. [Delayed graft function: a frequent but still unsolved problem in renal transplantation]. *Nephrol Ther*. 2005;**1**(6):325-34. [PubMed ID: 16895703]. https://doi.org/10.1016/j.nephro.2005.08.003.
11. Johnston O, O'Kelly P, Spencer S, Donohoe J, Walshe JJ, Little DM, et al. Reduced graft function (with or without dialysis) vs immediate graft function--a comparison of long-term renal allograft survival. *Nephrol Dial Transplant*. 2006;**21**(8):2270-4. [PubMed ID: 16720598]. https://doi.org/10.1093/ndt/gfl103.
12. Brennan TV, Freise CE, Fuller TF, Bostrom A, Tomlanovich SJ, Feng S. Early graft function after living donor kidney transplantation predicts rejection but not outcomes. *Am J Transplant*. 2004;**4**(6):971-9. [PubMed ID: 15147432]. https://doi.org/10.1111/j.1600-6143.2004.00441.x.
13. Brook NR, Waller JR, Nicholson ML. Nonheart-beating kidney donation: current practice and future developments. *Kidney Int*. 2003;**63**(4):1516-29. [PubMed ID: 12631369]. https://doi.org/10.1046/j.1523-1755.2003.00854.x.
14. Troncoso P, Ortiz AM, Jara A. Induction immunosuppressive therapy stratified according to risk categories in renal transplantation. *Transplant Proc*. 2003;**35**(7):2495-9. [PubMed ID: 14611995]. https://doi.org/10.1016/j.transproceed.2003.09.092.
15. Schnuelle P, Lorenz D, Trede M, Van Der Woude FJ. Impact of renal cadaveric transplantation on survival in end-stage renal failure: evidence for reduced mortality risk compared with hemodialysis during long-term follow-up. *J Am Soc Nephrol*. 1998;**9**(11):2135-41. [PubMed ID: 9808102]. https://doi.org/10.1681/ASN.V9112135.
16. Nankivell BJ, Chapman JR. The significance of subclinical rejection and the value of protocol biopsies. *Am J Transplant*. 2006;**6**(9):2006-12. [PubMed ID: 16796717]. https://doi.org/10.1111/j.1600-6143.2006.01436.x.
17. Serón D, Moreso F. Protocol biopsies in renal transplantation: prognostic value of structural monitoring. *Kidney Int*. 2007;**72**(6):690-7. [PubMed ID: 17597702]. https://doi.org/10.1038/sj.ki.5002396.
18. Thaunat O, Legendre C, Morelon E, Kreis H, Mamzer-Bruneel MF. To biopsy or not to biopsy? Should we screen the histology of stable renal grafts? *Transplantation*. 2007;**84**(6):671-6. [PubMed ID: 17893596]. https://doi.org/10.1097/01.tp.0000282870.71282.ed.
19. Salvadori M, Rosso G, Bertoni E. Update on ischemia-reperfusion injury in kidney transplantation: Pathogenesis and treatment. *World J Transplant*. 2015;**5**(2):52-67. [PubMed ID: 26131407]. [PubMed Central ID: PMC4478600]. https://doi.org/10.5500/wjt.v5.i2.52.
20. O'Callaghan JM, Knight SR, Morgan RD, Morris PJ. Preservation solutions for static cold storage of kidney allografts: a systematic review and meta-analysis. *Am J Transplant*. 2012;**12**(4):896-906. [PubMed ID: 22221739]. https://doi.org/10.1111/j.1600-6143.2011.03908.x.
21. Snyder TM, Khush KK, Valantine HA, Quake SR. Universal noninvasive detection of solid organ transplant rejection. *Proc Natl Acad Sci U S A*. 2011;**108**(15):6229-34. [PubMed ID: 21444804]. [PubMed Central ID: PMC3076856]. https://doi.org/10.1073/pnas.1013924108.