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Histopathological Pattern of Live Related Renal Allograft Biopsy in A Tertiary Care Hospital of Dhaka, Bangladesh

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ABSTRACT

Background: Renal allograft dysfunction is a significant challenge in kidney transplantation, often leading to graft failure. Histopathological evaluation of renal biopsies plays a critical role in identifying the underlying causes of dysfunction. This study aimed to analyze the histopathological patterns of renal biopsies in live-related renal allograft dysfunction.

Methods: This retrospective cross-sectional study was carried out at the Department of Nephrology, Evercare Hospital, Dhaka, Bangladesh, over a period spanning from January 2018 to December 2024. The study included 96 adult patients who had undergone renal transplantation and subsequently underwent allograft biopsies. These biopsies were performed to investigate graft dysfunction without any obvious cause. The pathological findings were categorized based on the updated Banff classification system.

Results: In this study of 96 renal allograft recipients, the majority were male (78%) with a mean age of 36.4±3.8 years. The mean serum creatinine increased from 1.33±0.21 mg/dL to 2.39±0.37 mg/dL during biopsy. According to Banff classification, 34.4% had antibody-mediated rejection (ABMR), 26.0% had T-cell-mediated rejection (TCMR), and 7.3% showed interstitial fibrosis/tubular atrophy (IF/TA).

Conclusion: Renal biopsy in live-related renal allograft dysfunction revealed common patterns of antibody-mediated rejection, T-cell-mediated rejection, and interstitial fibrosis/tubular atrophy. Elevated serum creatinine levels correlated with graft dysfunction, emphasizing the importance of early detection for improved management.

Keywords: Allograft biopsy, Calcineurin inhibitors, Graft dysfunction, Allograft kidney Biopsy, T-cell mediated rejection, antibody mediated rejection, Banff classification.

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Introduction

Chronic kidney disease (CKD) is a significant global public health issue, contributing substantially to morbidity, mortality, and the economic burden worldwide.[1] According to the US Renal Data System (USRDS), there were 120,688 new cases of end-stage renal disease (ESRD) reported in 2014, reflecting an annual increase of 1.1% .[2] Recent data suggest that approximately 60% of kidney transplant recipients can expect a 20-year graft survival rate.[3] In Pakistan, the incidence of ESRD is estimated at 100 cases per million populations.[4] Many patients who initiate hemodialysis either discontinue treatment or succumb within the first three months due to financial constraints, with less than 2% transitioning to ambulatory peritoneal dialysis. Despite being a more cost-effective option, renal transplantation is accessible to only 4 – 5% of patients with ESRD.[5] Renal transplantation is recognized as the preferred treatment for patients with end-stage renal disease (ESRD) globally.[6] In recent years, the success rates of renal transplantation have

significantly improved.[7] Current data indicate that approximately 60% of kidney transplant recipients can achieve a 20-year graft survival.[3] This improvement in short-term transplant outcomes over the past few decades is attributed to advancements in surgical techniques, enhanced medical care, early infection diagnosis and treatment, and progress in transplant immunology.[3,7] Despite these advancements, renal allograft dysfunction remains a frequent challenge after transplantation. The primary causes of graft dysfunction include acute or chronic rejection, calcineurin inhibitor (CNI) toxicity, infections, and recurrence or development of de novo primary kidney disease. The aforementioned causes of renal allograft dysfunction require distinct therapeutic strategies, making accurate diagnosis crucial for effective management. Currently, this can only be reliably achieved through renal allograft biopsy. However, there is limited published data specifically focusing on the causes of graft dysfunction in living-related kidney transplant programs.[8,9] Renal transplant biopsy (RTB) is widely recognized as the gold standard for identifying the underlying etiology of allograft dysfunction.[10] Previous studies have reported that biopsy findings alter clinical diagnoses in approximately 36% of cases and lead to changes in therapy in 59% of patients.[11] The field of renal transplant pathology is intricate and rapidly advancing, with the analysis of pathological lesions providing valuable insights into prevailing clinical practices and emerging trends in transplantation medicine.[8] This study was therefore conducted to observe the histopathological patterns of kidney transplant biopsies from our local cohort.

Methodology

This cross-sectional study was conducted at the Department of Nephrology, Evercare Hospital, Dhaka, Bangladesh, spanning from January 2018 to December 2024. The study included adult post-renal transplant patients who underwent allograft biopsy to investigate graft dysfunction with no appearent cause. Data were collected retrospectively from hospital database. The pathological findings were classified according to the updated Banff classifications (https://banfffoundation.org/central-repository-for-banff-classification-resources-3). As per the inclusion criteria, patients with allograft biopsy and graft dysfunction were enrolled in the study. Conversely, those with biopsies performed outside the center were excluded based on the exclusion criteria. The study received approval from the hospital's ethical committee. Data analysis was performed using MS Office tools and SPSS version 23.0.

Result

Total number of patients were 96.

The age distribution of participants shows that the majority were within the 15 – 30 years age group (39.6%). The mean age of participants was 36.4±3.8 years (Figure Ⅰ). In gender distribution, majority of patients (78%) were male (Figure Ⅱ). The findings of the investigation revealed that the mean serum creatinine (SC) level at baseline was 1.33±0.21 mg/dL, which increased to 2.39±0.37 mg/dL during the biopsy. The mean tacrolimus level was 6.89±1.22 ng/mL. The distribution of patients according to Banff categories showed that 34.4% fell under Category 2 (Antibody-mediated rejection, ABMR). Category 3 (Borderline changes) accounted for 17.7%, and 26.0% were in Category 4 (T-cell-mediated rejection, TCMR). Category 5 (Interstitial fibrosis/tubular atrophy, IF/TA) represented 7.3% of patients, with mild, moderate, and severe IF/TA observed in 2.1%, 3.1%, and 2.1% of these cases, respectively. Lastly, 14.6% of patients were categorized as Category 6, with the causes being calcineurin inhibitor (CNI) toxicity (7.3%), recurrent or de novo glomerulonephritis (5.2%) and infection (2.1%) (Table 1).

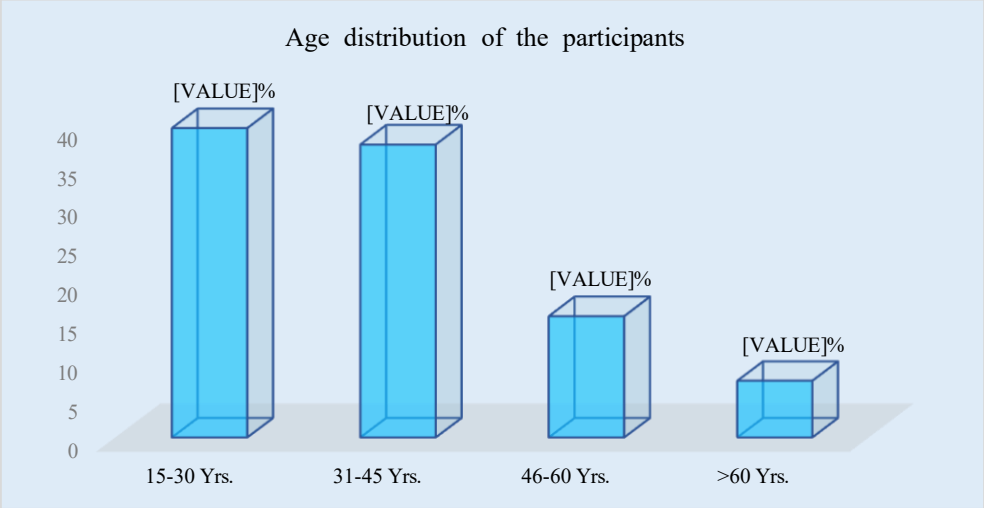

Figure I: Column chart showing age wise patients distribution among kidney transplant patients (N=96)

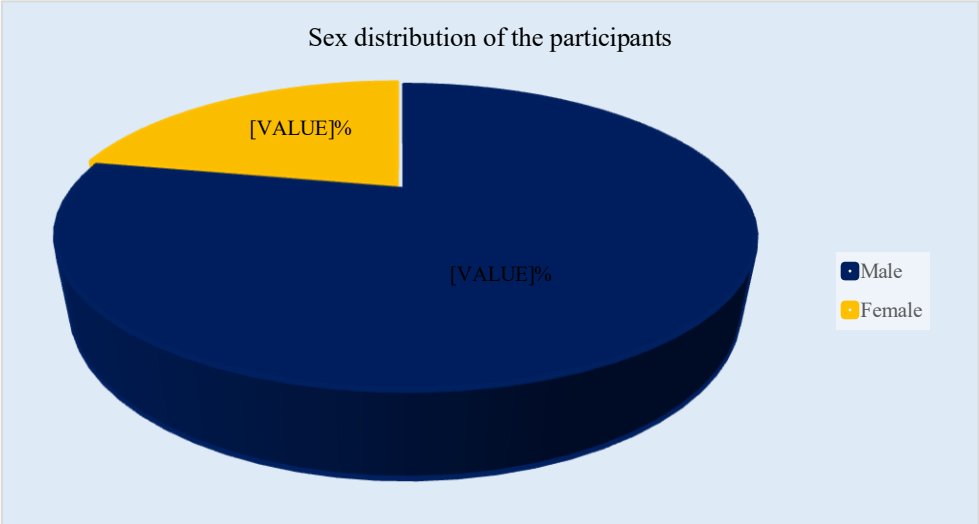

Figure 2: Pie chart showing gender wise patients distribution among kidney transplant patients (N=96)

Table 1: Banff categories of patients (N=96)

Banff categories	n	%
Category 2 (ABMR)	33	34.4%
Category 3 (Borderline)	17	17.7%
Category 4 (TCMR)	25	26.0%
Category 5 (IF/TA)	7	7.3%
Mild IFTA	2	2.1%
Moderate IFTA	3	3.1%
Severe IFTA	2	2.1%
Category 6 (Others)	14	14.6%
CNI toxicity	7	7.3%
R/de novo GN	5	5.2%
Infection	2	2.1%

Discussion

This study evaluated the histopathological patterns of renal biopsies in live-related renal allograft dysfunction in a tertiary care hospital in Dhaka, Bangladesh. The findings revealed a diverse spectrum of allograft dysfunction causes, categorized based on Banff criteria. These findings align with and diverge from the existing literature, reflecting unique patterns and variations in renal allograft dysfunction. The age distribution of participants in this study showed that most patients (39.6%) were aged between 15 – 30 years, with a mean age of 36.4±3.8 years. This is consistent with studies conducted in other regions where younger patients often represent the majority of transplant recipients due to their better overall health and suitability for transplantation.[2] However, some studies reported an older age group as predominant, possibly due to demographic differences and disease prevalence.[12] Gender distribution revealed a male predominance which is similar to findings in other studies.[13,14] This gender disparity may be attributed to higher rates of end-stage renal disease (ESRD) in males and better access to transplantation compared to females in certain regions.[14] However, some studies report more balanced gender distributions, suggesting regional differences in healthcare access and gender dynamics.[15]

In this study, the mean serum creatinine (SC) level at baseline was 1.33±0.21 mg/dL, which increased

to 2.39±0.37 mg/dL during biopsy. Elevated SC is a well-established marker of renal allograft dysfunction, ahowever may not predict the outcomes in a patient with acute rejection.[16] Additionally, the mean tacrolimus level was 6.89±1.22 ng/mL, which aligns with therapeutic ranges observed in previous research.[17] However, variations in tacrolimus levels and their association with allograft outcomes highlight the need for individualized dose monitoring to minimize toxicity.

The distribution of histopathological categories, as per the Banff classification mostly aligns with other studies, where a proportion of biopsies yield no significant abnormalities, possibly due to subclinical rejection or functional abnormalities undetected histopathologically.[18,19] Antibody-mediated rejection (ABMR) was observed in 34.4% of patients, consistent with global reports identifying ABMR as a leading cause of graft dysfunction.[20] ABMR is often associated with poor graft survival due to its progressive nature. Conversely, some studies report lower ABMR rates, possibly due to differences in immunosuppressive protocols or diagnostic criteria.[21] Borderline changes accounted in nearly one-fifth of the cases in this study. T-cell-mediated rejection (TCMR) represented 28% of cases, which is in line with previous research emphasizing TCMR as a major contributor to early and late graft dysfunction.[22] TCMR outcomes are generally better than ABMR if promptly identified and managed.[22] Interstitial fibrosis/tubular atrophy (IF/TA) was observed in 7.3% of patients, with mild, moderate, and severe forms occurring in 2.1%, 3.1%, and 2.1% of cases, respectively. These findings are comparable to previous studies identifying IF/TA as a common cause of chronic allograft dysfunction.[23] The high rates of moderate and severe IF/TA highlight the importance of early detection and intervention to prevent irreversible damage.[24] Category 6 comprised 14.6% of patients, attributed to calcineurin inhibitor (CNI) toxicity, recurrent or de novo glomerulonephritis, and infection. Recurrent/ De novo GN seen in 5.2% cases. CNI toxicity, seen in 7.3% of these cases, is a well-recognized complication of prolonged immunosuppressive therapy.[17] Infection-related dysfunction (2.1%) underscores the delicate balance between immunosuppression and infection risk in transplant recipients.[25] This study highlights the diverse histopathological patterns in live-related renal allograft dysfunction. The findings emphasize the importance of routine biopsy evaluations and individualized management to optimize graft outcomes. Future studies are needed to explore the long-term implications of these patterns and refine treatment strategies.

Conclusion

The histopathological findings of renal biopsy in live-related renal allograft dysfunction revealed a high prevalence of antibody-mediated and T-cell-mediated rejections, along with interstitial fibrosis/tubular atrophy. The progression of graft dysfunction was associated with elevated serum creatinine levels. Early identification of these patterns can aid in better management and outcomes in renal transplant recipients.

References

1. GBD 2023 Chronic Kidney Disease Collaborators. Global, regional, and national burden of chronic kidney disease in adults, 1990-2023, and its attributable risk factors: a systematic analysis for the Global Burden of Disease Study 2023. Lancet. 2025 Nov 22;406(10518):2461-2482. doi: 10.1016/S0140-6736(25)01853-7. .
2. United States Renal Data System. *2024 USRDS Annual Data Report: Epidemiology of Kidney Disease in the United States*. National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2024. https://usrds-adr.niddk.nih.gov/2025. Last accessed 10 March, scotman
3. (Severova-Andreevska G, et al. The spectrum of histopathological changes in the renal allograft - a 12 months protocol biopsy study. Open Access Maced J Med Sci 2018; 6(4):606-12.
4. Ullah K, Butt G, Masroor I, Kanwal K, Kifayat F. Epidemiology of chronic kidney disease in a Pakistani population. Saudi J Kidney Dis Transplant 2015; 26(6):1307-10. doi: 10.4103/ 1319-2442.168694.
5. Rizvi SA, Naqvi SA, Zafar MN, Hussain Z, Hashmi A, Hussain M, et al. A renal transplantation model for developing countries. Am J Transplant 2011; 11(11):2302-7.
6. Zhang H, Wang Z, Zhang J, Zhang X, Gui Z, Sun L, et al. The synergism of B and T lymphocyte attenuator

(BTLA) and cytotoxic T lymphocyte associated antigen-4 (CTLA-4) attenuated acute T-cell mediated rejection and prolonged renal graft survival. Transl Androl Urol. 2020 Oct;9(5):1990-1999. doi: 10.21037/tau-20-728.

7. Han Y, Guo H, Cai M, Xiao L, Wang Q, Xu X, et al. Renal graft biopsy assists diagnosis and treatment of renal allograft dysfunction after kidney transplantation: A report of 106 cases. Int J Clin Exp Med 2015; 8(3):4703-7.
8. Kazi JI, Mubarak M. Biopsy findings in renal allograft dysfunction in a live-related renal transplant program. J Transplant Technol Res 2012; 2:108.
9. Puntambekar A, Parameswaran S, Nachiappa RG. Evaluation of clinicopathological spectrum in renal allograft biopsies at JIPMER. J Kidney 2017; 03(03
10. Schmid A. Percutaneous renal transplant biopsy: Is the safety profile adequate for short-term postprocedure monitoring? Transpl Int 2016; 29(2):165-6.
11. Qasim H, Abuuqteish H, Khattab K, Leoni MLG, Varrassi G. Renal Allograft Pathology Classifications: Contemporary Updates and Diagnostic Utility. Cureus. 2025 Sep 24;17(9):e93134. doi: 10.7759/cureus.93134.
12. Tegzess E, Gomes Neto AW, Pol RA, de Boer SE, Peters-Sengers H, Sanders JF, Berger SP. Comparative survival of elderly renal transplant recipients with a living donor versus a deceased donor: A retrospective single center observational study. Transpl Int. 2021 Dec;34(12):2746-2754. doi: 10.1111/tri.14130.
13. Katz-Greenberg G, Shah S. Sex and Gender Differences in Kidney Transplantation. Semin Nephrol. 2022 Mar;42(2):219-229. doi: 10.1016/j.semnephrol.2022.04.011.
14. Nautiyal A, Bagchi S, Bansal SB. Gender and kidney transplantation. Front Nephrol. 2024 Apr 22;4:1360856. doi: 10.3389/fneph.2024.1360856.
15. Salas MAP, Chua E, Rossi A, Shah S, Katz-Greenberg G, Coscia L, Sawinski D, Adey D. Sex and gender disparity in kidney transplantation: Historical and future perspectives. Clin Transplant. 2022 Dec;36(12):e14814. doi: 10.1111/ctr.14814.
16. Parajuli S, Zhong W, Pantha M, Sokup M, Aziz F, Garg N, Mohamed M, Mandelbrot D. The Trend of Serum Creatinine Does Not Predict Follow-Up Biopsy Findings Among Kidney Transplant Recipients With Antibody-Mediated Rejection. Transplant Direct. 2023 May 24;9(6):e1489. doi: 10.1097/TXD.0000000000001489
17. Bentata Y. Tacrolimus: 20 years of use in adult kidney transplantation. What we should know about its nephrotoxicity. Artif Organs. 2020 Feb;44(2):140-152. doi: 10.1111/aor.13551.
18. Rodriguez-Ramirez S, Al Jurdi A, Konvalinka A, Riella LV. Antibody-mediated rejection: prevention, monitoring and treatment dilemmas. Curr Opin Organ Transplant. 2022 Oct 1;27(5):405-414. doi: 10.1097/MOT.0000000000001011.
19. Haas, M. "The revised (2013) Banff classification for antibody-mediated rejection of renal allografts: update, difficulties, and future considerations." American Journal of Transplantation 16.5 (2016): 1352-1357.
20. Rodriguez-Ramirez S, Al Jurdi A, Konvalinka A, Riella LV. Antibody-mediated rejection: prevention, monitoring and treatment dilemmas. Curr Opin Organ Transplant. 2022 Oct 1;27(5):405-414. doi: 10.1097/MOT.0000000000001011.
21. Rodrigo, Emilio, et al. "Acute rejection following kidney transplantation: state-of-the-art and future perspectives." Current Pharmaceutical Design 26.28 (2020): 3468-3496.
22. Rampersad C, Balshaw R, Gibson IW, Ho J, Shaw J, Karpinski M,et al. The negative impact of T cell-mediated rejection on renal allograft survival in the modern era. Am J Transplant. 2022 Mar;22(3):761-771. doi: 10.1111/ajt.16883.
23. Mubarak M. Kidney Allograft Interstitial Fibrosis and Tubular Atrophy Due to Specific Causes: Bacterial Infection/Pyelonephritis. Pak J Kidney Dis. 2022;6(4):34-38 DOI: 10.53778/pjkd6421
24. García-Carro C, Dörje C, Åsberg A, Midtvedt K, Scott H, Reinholt FP, et al. Inflammation in Early Kidney Allograft Surveillance Biopsies With and Without Associated Tubulointerstitial Chronic Damage as a Predictor of Fibrosis Progression and Development of De Novo Donor Specific Antibodies. Transplantation. 2017 Jun;101(6):1410-1415. doi: 10.1097/TP.0000000000001216.
25. Thongprayoon C, Hansrivijit P, Leeaphorn N, Acharya P, Torres-Ortiz A, Kaewput W, Kovvuru K, Kanduri SR, Bathini T, Cheungpasitporn W. Recent Advances and Clinical Outcomes of Kidney Transplantation. J Clin Med. 2020 Apr 22;9(4):1193. doi: 10.3390/jcm9041193.