**Etiology of Renal Allograft Dysfunction: A Single Center-based Study**

**Abstract**

**Background**: Multiple factors interplay in development of acute or chronic renal allograft dysfunction. Biopsy is the gold standard modality of evaluating the cause of transplant dysfunction. **Objective**: The present study was aimed to assess the etiology behind allograft dysfunction. **Method**: A longitudinal, observational approach was carried forward to investigate 79 renal transplant recipients during the period of September 2022 to March 2025 in Center for Kidney Diseases & Urology Hospital in Dhaka, Bangladesh. **Result**: Findings revealed, mean age of the participants was 35.03 ± 9.351 years; 78.5% were males, mean serum creatinine level was 262.4 ± 143.04 µmol/L and mean duration between transplant and diagnosis of allograft dysfunction was 1.60 ± 2.783 years. In addition, the native kidney disease of 84.5 % of the study population was not identified prior to transplantation. Active antibody-mediated rejection (ABMR), calcineurin inhibitor (CNI) toxicity and acute tubular injury were the prevalent etiologies revealed in this study (16.5%, 13.5% and 10.1%) respectively. The highest mean serum creatinine level (384.0 ± 118.7 µmol/L) was detected for combination etiology of acute T-cell mediated rejection (TCMR) and active ABMR. The longest duration between transplant and diagnosis (4.50 ± 4.44 years) was found in case of chronic active ABMR. **Conclusion**: Early detection and prompt evaluation are crucial for the management of this grievous condition and the prevention of long-term complications.

**Keywords**

*Renal allograft; kidney transplant; allograft dysfunction; chronic kidney disease; end-stage renal disease; nephrology*

**Introduction**

From the perspective of noncommunicable diseases, chronic kidney disease is a significant contributor to morbidity and mortality as well as a crucial public health issue. [1] Recently, kidney transplantation has emerged as a preferred modality in terms of renal replacement therapy for the treatment of end-stage renal disease owing to increased life expectancy and better quality of life for the patients. [1-2] However, certain factors exert an influence on the functioning of the graft which can lead to graft dysfunction. [2] Allograft dysfunction following renal transplantation is often clinically silent, with uncontrolled hypertension being one of the few observable signs. It typically comes to light through diagnostic evaluations, such as a rise in serum creatinine levels and a corresponding decline in glomerular filtration rate (GFR). [2-3] Further assessments may involve blood tests, drug-level monitoring, urinalysis which would reveal proteinuria and active urinary sediments, transplant ultrasonography and allograft biopsy. [2-3] Nonetheless, allograft biopsy can be declared as the gold standard for the detection of graft dysfunction; as its results assist in determination of the etiology of the renal injury, severity of rejection and the optimum treatment strategy. [4-6]

Concerning the global incidence and prevalence of allograft rejection, it will be fair enough to say that it has reduced dramatically and the survival of grafts have improved markedly owing to newer and more efficacious immunosuppressive agents for induction and maintenance therapy. [6] For instance, the current global incidence rate of acute rejection within the first year is around 7.9%. [6]

An appropriate approach to illustrate the pathologies behind renal transplant dysfunctions came across in the year of 1991, by Professor Kim Solez and Loraine Racusen, which is known as the Banff classification of allograft pathology. [2] As the treatment modalities vary according to the etiology of allograft loss, it is essential to distinguish the exact reason for the condition. [5] Banff system maps out the rejections, encompassing progresses in molecular landscapes of injury and imparting the diagnostic schemes of acute and chronic rejection. [5, 7] According to this classification, the topmost important causes for dysfunction are antibody-mediated rejection (ABMR) and T-cell mediated rejection (TCMR). [2] Other leading causes are BK polyomavirus nephropathy and calcineurin inhibitor (CNI) toxicity. [5] However, evidence suggests it is not wise to neglect peri-operative complications and recurrent infections as crucial factors for graft rejections. [8]

Additionally, post-transplant time remains a key factor in distinguishing early and late graft rejections; owing to the fact that some causes frequently occur early after transplant, whereas others take place much later. [5, 8] Another point that should be shed light on is intercurrent health conditions and morbidities contribute profoundly to allograft dysfunction and late allograft failure. [8]

Nonetheless, graft dysfunction should be detected and treated promptly, as there are various complications associated with this. Since the management involves immunosuppressive agents in enormous doses, the patients are already in a severely immunocompromised state and there are various side effects associated with these agents such as increased cardiovascular risks, development of post-transplant diabetes mellitus, dyslipidemia, various malignancies such as post-transplant lymphoproliferative diseases, opportunistic infections such as CMV, histoplasmosis and other atypical microorganisms and so on. [6] Furthermore, it should be kept in mind that managing immunosuppressive agents after rejection is always a double-edged sword. [6]

Recent evidences suggest that treatment non-compliance is one of the core risk factors for rejection of graft, hence patient education and awareness is of paramount importance. This study aims to discover the etiology behind the renal allograft dysfunction occurring in the context of Bangladesh. The findings would be imperative for clinicians, public health experts, stakeholders and policymakers for making better decisions for detecting and managing allograft dysfunction cases and in raising awareness for prevention.

**Methodology**

**Study design, participants and settings**

This longitudinal, observational study involved 79 alive renal allograft recipients between the period of September 2022 to March 2025. The study was a single institute study and it was conducted in Center for Kidney Diseases & Urology Hospital (CKDU), Dhaka, Bangladesh. The sampling was done by convenient technique.

The selection criteria were as follows:

**Inclusion criteria**

* Renal allograft recipients of living donors.
* Those patients who had persistent unexplained raised serum creatinine 25% above the baseline value.

**Exclusion criteria**

* Patients with obstructive changes after kidney transplant.
* Patients with uncontrolled bleeding diathesis.
* Patients with uncontrolled, severe hypertension.
* Those with active renal and/or peri-renal infection.
* Those who suffered from any skin infection at the site of biopsy.

As it was an observational study, all patients during study period with written, informed consent and fulfilling eligibility criteria at CKDU hospital were included in this study.

**Investigations and statistical analysis**

Investigations were performed as listed below:

* Complete blood count
* Serum creatinine
* Urine R/M/E
* Random blood sugar
* HbA1c
* Serum CMV DNA (PCR)
* Serum BKV DNA (PCR)
* Serum albumin
* ANA
* Anti-ds DNA antibody
* Urinary Albumin Creatinine Ratio
* HBsAg
* Anti-HCV
* Bleeding time, Clotting time
* Prothrombin time, Activated Partial Thromboplastin time
* Duplex study of transplanted kidney
* Tacrolimus (trough level)
* Ultrasound-guided renal biopsy.

The staining details are listed below:

* For low microscopic technique, formalin was used.
* For direct immunofluorescence technique, normal saline was used.

The following types of stains were used in the process:

* Hematoxylin & Eosin stain
* Silver stain
* Masson’s Trichrome stain
* Periodic Acid-Schiff stain
* C4d & SV40 (Immunohistochemical) stain

After all these procedures, we arrived at the diagnoses for determination of the etiology of renal transplant dysfunction.

Data were collected by face-to-face interview and using hospital records. Recipient and donor characteristics including age, gender, native kidney disease, donor relationship, induction agent (s) used and graft function post transplantation were documented. Physical examination was performed and reports of laboratory investigations using a structured questionnaire containing all the variables of interest. All the data were compiled in a preformed structured case record form.

Renal allograft dysfunctions were classified using the Banff criteria (2017). It is described below:

* Category 1: Normal or non-specific change
* Category 2: ABMR a. Active ABMR

b. Chronic Active ABMR

c. C4d staging without evidence of rejection

* Category 3: Borderline change (suspicious for active TCMR)
* Category 4: TCMR a. Acute TCMR

b. Chronic Active TCMR

* Category 5: IFTA a. Grade I

b. Grade II

c. Grade III

* Category 6: Others

After collection, all the data were compiled, edited and analyzed using Statistical Package for Social Sciences (SPSS) version 26. Continuous variables were expressed as mean and standard deviation. Categorical variables were expressed as frequency distribution. Data were presented in tables and figures where relevant.

**Induction and maintenance regimen used for immunosuppression**

* Injection Methylprednisolone (500 mg) I/V daily for 3 days
* Injection Basiliximab (20 mg) I/V: Day 0 & Day 4
* Injection Anti T-lymphocyte Globulin (Rabbit) (3-5 mg/kg/day) I/V: 3-5 days
* Tacrolimus (0.1 mg/kg/day) oral: in two divided doses
* Mycophenolate mofetil (500-1000 mg) oral: twice daily
* Mycophenolate sodium (360-720 mg) oral: twice daily

**Results**

Regarding gender, 62 (78.5%) participants were males. Figure 1 below shows the gender distribution of the allograft recipients.

**Figure 1: Pie chart demonstrating the gender distribution of the participants (n=79).**

**Table 1: Clinical profile of the participants (n=79).**

|  |  |
| --- | --- |
| **Characteristics** | **Mean (± SD)** |
| Age (in years) | 35.03 ± 9.351 |
| S. creatinine (µmol/L) | 262.4 ± 143.04 |
| Time lapse from transplant to diagnosis (in years) | 1.60 ± 2.783 |
| Hemoglobin (g/dL) | 9.7 ± 2.69 |
| Mean 24-hour proteinuria (g) | 1.15 ± 1.11 |

\*SD: Standard deviation

The mean age of the recipients at the time of conducting the renal biopsy was 35.03 ± 9.351 years. The mean hemoglobin level at the time of diagnosis was 9.7 ± 2.69 g/dL. The mean serum creatinine level of the patients was found to be 262.4 ± 143.04 µmol/L. Additionally, recording the time lapse between the renal transplant and diagnosis of allograft dysfunction revealed a range from 0 to 15 years, with the majority [33 (41.8%)] found to be within the first year, followed by [13 (16.5%) came out to be 1 year time duration. The mean time lapse was 1.60 ± 2.783 years. It is quite clear that, majority of the patients can be declared as early graft dysfunction patients. Lastly, the mean 24-hour proteinuria at the time of diagnosis of graft dysfunction was found to be 1.15 ± 1.11 g. Table 1 highlights the clinical characteristics of the patients.

Figure 2 depicts a column bar showing the distribution of native renal disease present in the allograft recipients. Majority of the participants, 84.5% had no known native kidney diseases.

**Figure 2: Column bar showing distribution of known or unknown native kidney disease present in the recipients (n=79).**

**Table 2: Characteristics of allograft dysfunction (n=79).**

|  |  |  |  |
| --- | --- | --- | --- |
| **Cause of allograft dysfunction based on biopsy** | **Frequency (f)/ Percentage (%)** | **Mean S. Creatinine at the time of dysfunction (****µmol/L)** | **Mean time gap between transplant to diagnosis (in years)** |
| Active ABMR | 13 (16.5) | 310.92 ± 294.26 | 0.84 ± 1.66 |
| CNI toxicity | 11 (13.5) | 245.09 ± 49.48 | 0.936 ± 1.30 |
| Acute tubular injury | 8 (10.1) | 251.5 ± 107.67 | 0.30 ± 0.45 |
| TMA | 7 (8.9) | 212.42 ± 38.14 | 0.643 ± 1.10 |
| Pyelonephritis | 7 (8.9) | 285.28 ± 139.6 | 0.429 ± 0.786 |
| Chronic Active ABMR | 7 (8.9) | 254.57 ± 88.4 | 4.50 ± 4.44 |
| FSGS | 6 (7.6) | 233.5 ± 88.01 | 4.167 ± 5.60 |
| IFTA | 4 (5.1) | 256.50 ± 68.94 | 2.60 ± 4.28 |
| IgA Nephropathy | 3 (3.8) | 192.66 ± 50.80 | 3.00 ± 3.46 |
| Acute TCMR + Active ABMR | 2 (2.5) | 384.0 ± 118.7 | 2.50 ± 3.53 |
| Chronic Active ABMR + IgA Nephropathy | 2 (2.5) | 313.0 ± 118.7 | 2.50 ± 2.47 |
| Active ABMR + TMA | 2 (2.5) | 372.5 ± 147.7 | 3.00 ± 0.00 |
| Borderline changes | 2 (2.5) | 161.0 ± 50.91 | 0.50 ± 0.70 |

\*ABMR: Antibody Mediated Rejection; ATN: Acute Tubular Necrosis; TMA: Thrombotic Microangiopathy; FSGS: Focal Segmental Glomerulosclerosis; IgA: Immunoglobulin A; TCMR: T-cell Mediated Rejection; CNI toxicity: Calcineurin Inhibitor toxicity; IFTA: Interstitial Fibrosis and Tubular Atrophy.

Table 2 elaborates the characteristics of renal allograft dysfunction in the study participants. The categorization of the causes was done on the basis of Banff criteria (2017). It is clearly evident that, following the renal biopsies, active ABMR was the cause of dysfunction in majority of the cases [13 (16.5%)] followed by CNI toxicity [11 (13.5%)]. The highest mean serum creatinine level (384.0 ± 118.7 µmol/L) was detected for the combination of acute TCMR along with active ABMR and lowest mean (161.0 ± 50.91 µmol/L) was found in case of borderline. Regarding the time lapse and determination of early and late graft dysfunction, it was visible that the earliest dysfunctions were found in cases of acute tubular injury, pyelonephritis and borderline changes (0.30 ± 0.45 years; 0.429 ± 0.786 years and 0.50 ± 0.70 years respectively). On the contrary, chronic active ABMR, FSGS and IgA nephropathy had the longest time gaps from transplant to diagnosis of graft dysfunctions (4.50 ± 4.44 years; 4.167 ± 5.60 years and 3.00 ± 3.46 years respectively). It was also noted during the analysis that in 4 of the recipients, BK polyomavirus was found to be positive.

Figure 3 highlights the distribution of interstitial fibrosis and tubular atrophy (IFTA) in the transplant recipients based on the Banff criteria (2017). The findings suggest that, majority, meaning 48.1% participants did not have any type of IFTA at the time of biopsies. Besides, about 29.1% respondents reported Grade I IFTA in the biopsies. Only 3.8% participants expressed Grade III IFTA.

**Figure 3: Distribution of IFTA grading in the allograft recipients (n=79).**

**Discussion**

Renal transplant is one of the core and gold standard modalities of management of end-stage renal disease across the world. Various etiologies like diabetic nephropathy, glomerulonephritis, interstitial nephritis and obstructive nephropathy can lead to ESRD, and mostly, these could be a consequence of multiple causes altogether. Medical management and dialysis tend to give limited benefits to the patient; however, for a permanent solution, the renal transplant has been adopted as a preferred approach in the past few decades. There is scarcity of research on the etiologies and risk factors of transplant dysfunction particularly in Bangladesh, and the aim of this paper is to contribute to the existent literature in this regard; and the findings may contribute to better management of the transplant recipients.

This study found the mean age of the participants to be 35.03 ± 9.351 years; and 78.5% were males. Findings were consistent with previous study [2] which found the mean age of the respondents to be 34 ± 7 years and 84% meaning majority were males.

At the time of performing renal biopsy, mean serum creatinine level was found to be 262.4 ± 143.04 µmol/L; mean hemoglobin level 9.7 ± 2.69 g/dL, mean 24-hour proteinuria being 1.15 ± 1.11 g. Evidence from recent study performed in Bangladesh [9] suggests that, mean serum creatinine level of the participants was higher than what we found, 374.05 ± 16.24 µmol/L. Meanwhile, another paper [1] found mean hemoglobin level at the time of diagnosis of dysfunction was similar to us, meaning 9.4 ± 2.55 g/dL; mean 24-hour proteinuria being lower, meaning 0.64 ± 0.27 g. The dissimilarities can be explained by the variations in ethnicity and demography and difference in the etiologies of the renal transplant dysfunction.

We found the mean time lapse between transplantation and diagnosis of dysfunction was 1.60 ± 2.783 years. Evidence from relevant study [9] reveals the average time duration was 2.63 ± 3.95 years. The slight variations in findings could be due to smaller cohort in the reference study mentioned (n=23) compared to our study (n=79).

In addition, it was revealed that 84.5% of the renal transplant recipients had no known native renal diseases. Our findings are inconsistent with previous study performed in Bangladesh [10] between the period of 2010-2012, which demonstrated that, majority 88.58% had glomerulonephritis, 5.72% had polycystic kidney disease, 2.85% had chronic pyelonephritis and another 2.85% had diabetic nephropathy. This can be put across owing to variations in secular trends over the period of time.

Furthermore, our study found that active ABMR, CNI toxicity and acute tubular injury were the most prevalent causes for allograft dysfunction. Our findings were partly consistent with another study performed in Pakistan [2] in this regard which elaborated that, 20.24%, 9.81% and 9.20% and 9.05% of the cases were due to active ABMR, chronic active ABMR, FSGS and CNI toxicity respectively. Regional differences can be a cause of these dissimilarities.

Our mean serum creatinine levels ranged from 161.0 ± 50.91 µmol/L in case of borderline rejection to 384.0 ± 118.7 µmol/L for acute TCMR + active ABMR. In case of active ABMR, the mean value of S. creatinine was 310.92 ± 294.26 µmol/L. Similar study [9] claimed those patients with active ABMR had S. creatinine level of 273.89 µmol/L. In addition, this reference study found their highest mean level of S. creatinine to be 499.95 µmol/L in case of IgA nephropathy; whereas in our investigation it was 192.66 ± 50.80 µmol/L.

Additionally, earliest graft dysfunctions were detected in cases of acute tubular injury, followed by pyelonephritis and borderline changes. Our findings were unfamiliar to the other paper [9] which mentioned the earliest dysfunctions were reported in cases of CNI toxicity, crystal nephropathy, post-ischemic renal cortical necrosis (RCN). Furthermore, relevant study [11] suggests that, early causes of allograft dysfunction that manifest during the first 6 months after transplant include [hyperacute rejection](https://www.sciencedirect.com/topics/medicine-and-dentistry/hyperacute-graft-rejection), thrombosis, urologic causes (urine leak, ureteral obstruction), and thrombotic [microangiopathy](https://www.sciencedirect.com/topics/medicine-and-dentistry/microangiopathy). In addition, it is found that, prolonged cold ischemia, the time the kidney is kept without blood flow before transplantation, is a significant factor in early allograft dysfunction. [12]

On the contrary, chronic active ABMR, FSGS and IgA nephropathy cases took the longest time duration from the onset of renal transplantation to the diagnosis of allograft dysfunction. Evidence [13] suggests that, the main issues that may lead to the development of delayed graft dysfunction (DGF) are ischemia–reperfusion injury, the source and the quality of the donated kidney, and the clinical management of the recipient. It is of paramount importance to focus on improving early renal function after transplantation, as this can significantly enhance both short-term and long-term outcomes. In this context, technical and pharmacological strategies play crucial roles. [14]

Besides, this study found 4 cases out of the 79 renal transplant recipients to have BK polyomavirus positive. Also, 48.1% participants did not demonstrate any sort of IFTA in their biopsy reports; whereas 29.1% exhibited Grade I IFTA. Another study [15] showed, out of 56 transplant recipients, 16.7% cases were found to be diagnosed with BK nephropathy. Additionally, evidence [16] reveals that, the progression of IFTA seen on surveillance biopsies between implantation and 6-month post-transplant status predicted lower graft function at 5 y along with a higher likelihood of composite of graft loss and doubling of serum creatinine levels. However, it can be stated that, randomized clinical trials would be needed to establish the proper role of IFTA in graft loss.

Digging deep into pathogenesis, the binding of [alloantibodies](https://www.sciencedirect.com/topics/medicine-and-dentistry/alloantibody) to the donor tissue triggers [cytokine release](https://www.sciencedirect.com/topics/immunology-and-microbiology/cytokine-release) by donor endothelial cells and monocytes. *In vitro* studies of cocultured monocytes with HLA class I antibody-activated endothelial cells demonstrated secretion of proinflammatory cytokines. This [cytokine production](https://www.sciencedirect.com/topics/immunology-and-microbiology/cytokine-production) can orchestrate the recruitment and activation of deleterious immune cells that perpetuate the rejection process by eliciting direct graft injury or secreting even more cytokines. Among these, [CXC chemokine ligand 10](https://www.sciencedirect.com/topics/immunology-and-microbiology/gamma-interferon-inducible-protein-10) (CXCL10) is produced by both endothelial cells and monocytes, and its increase is well-associated with ABMR, recruiting both innate CXC [chemokine receptor](https://www.sciencedirect.com/topics/medicine-and-dentistry/chemokine-receptor) 3 (CXCR3)+ NK cells and adaptive CXCR3+ T cells. The binding of [alloantibodies](https://www.sciencedirect.com/topics/immunology-and-microbiology/alloantibody) to the donor tissue also triggers ADCC and complement-dependent cytotoxicity that can enhance effector cells. In addition to FcγR expression, NK cells and myeloid cells express [complement receptors](https://www.sciencedirect.com/topics/medicine-and-dentistry/complement-receptor) CR3 and CR5 that bind to C3 degradation fragments, and this binding increases the cytotoxicity of NK cells and macrophages, but also of CD16A+ CD8 T cells. Finally, missing self and non-complement fixing HLA-DSA synergistically enhance NK cell activity against allogeneic endothelial cells, and this combination associated with worse allograft outcome after microvascular inflammation than presence of HLA-DSA alone. Similarly, pretransplant HLA-DSA and missing self are independent and cumulative risk factors for the occurrence of microvascular inflammation after transplantation, and for the development of [transplant glomerulopathy](https://www.sciencedirect.com/topics/medicine-and-dentistry/transplant-glomerulopathy) after the diagnosis of microvascular inflammation. [17]

The study had certain limitations. One would be this being a single-center study; as including few more institutions would have expressed a larger annexure as more causes would come in the limelight. Another flaw would be not incorporating HLA profiling and donor data. Coexisting systemic chronic diseases should have also been included to establish the etiologies and correlations better. Larger, prospective studies can give better insights. Nonetheless, the generated findings would be useful for the caregivers to initiate better treatment strategies to prevent graft dysfunctions and losses.

**Conclusion**

Active ABMR was found to be the prime cause of allograft in this study which occurred mostly within 2 years of renal transplantation. The other significant causes were CNI toxicity, acute tubular injury, TMA, chronic active ABMR and pyelonephritis. An integrated approach is required to ensure post-transplant care of the allograft recipients. Patient awareness regarding compliance and regular follow-ups should be encouraged to prevent graft rejections. The study findings will pave ways for better patient management in the future with scopes for broader and prospective studies.

**Ethical approval and consent**

Proper safety measures were taken in every step of the study. Clearance was obtained from Institutional Review Board (IRB) of CKDU. According to Helsinki Declaration for Medical Research involving Human Subjects 1964, all the patients were informed about the study design, the underlying hypothesis and the right of the participants to withdraw themselves from the research at any time and for any reason whatsoever.

The following ethical issues was addressed accordingly:

* Strict confidentiality and security of data related to the patient was maintained.
* The presentation of data and information related to the patient was documented anonymously.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that they have no known competing financial interests OR non-financial interests OR personal relationships that could have appeared to influence the work reported in this paper.

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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