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Original Research Article

Clinical outcomes of plasma exchange in acute and chronic antibody-mediated rejection following kidney transplantation: a prospective observational study

Maulik Patel[1,2]*, Akash Dholakia[2]

[1]Department of Nephrology, Institute of Kidney Diseases and Research Centre (IKDRC), Ahmedabad, Gujarat, India
[2]Department of Nephrology, Shankus Hospital, Mehsana, Gujarat, India

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***Correspondence:**
Dr. Maulik Patel,
E-mail: maulikpatel88.md@gmail.com

ABSTRACT

Background: Antibody-mediated rejection (ABMR) remains a major cause of renal allograft dysfunction and graft loss after kidney transplantation. Plasma exchange (PE) is widely used for antibody removal; however, its effectiveness in acute and chronic ABMR remains incompletely defined.
Methods: This prospective observational study was conducted at the Institute of Kidney Diseases and Research Centre–Institute of Transplantation Sciences (IKDRC-ITS), Ahmedabad, Gujarat, India. The study included 60 kidney transplant recipients with biopsy-proven ABMR treated between September 2017 and November 2019. Patients were classified as acute ABMR (n=30) or chronic ABMR (n=30) according to Banff criteria. All patients received plasma exchange in combination with corticosteroids, intravenous immunoglobulin, and optimization of maintenance immunosuppression. Demographic, clinical, histopathological, immunological, and renal outcomes were assessed at baseline, 6 months, and 5 years.
Result: Baseline demographic and laboratory characteristics were comparable between groups ($p>0.05$). Acute ABMR demonstrated significant improvement in histological activity scores following treatment, including C4d, glomerulitis, peritubular capillaritis, and vasculitis ($p\leq0.002$), whereas chronic ABMR showed no significant histological improvement ($p>0.05$). At 6 months, acute ABMR was associated with significantly better renal function, lower proteinuria, and lower donor-specific antibody levels than chronic ABMR (all $p<0.001$). At 5 years, acute ABMR maintained superior renal outcomes with lower serum creatinine, lower proteinuria, lower persistent donor-specific antibody positivity, and higher estimated glomerular filtration rate ($p<0.001$). Chronic ABMR was associated with more hospitalizations ($p<0.001$), infection-related admissions ($p=0.004$), cardiovascular events ($p=0.002$), mortality ($p=0.044$), and graft loss requiring maintenance dialysis ($p=0.026$).
Conclusion: Plasma exchange is associated with substantial histological and functional recovery in acute ABMR but provides limited benefit in chronic ABMR. Early diagnosis and intervention may improve long-term allograft survival and clinical outcomes.

Keywords: Antibody-mediated rejection, Kidney transplantation, Plasma exchange, Donor-specific antibody, Renal allograft, Graft survival

INTRODUCTION

Kidney transplantation is the preferred treatment for end-stage renal disease, providing significant survival benefits and improved quality of life compared with long-term dialysis. However, long-term allograft survival is heavily constrained by immune-mediated injury, particularly antibody-mediated rejection (ABMR). ABMR has been increasingly recognized over the past two decades as the

leading cause of late kidney allograft loss, often despite maintenance immunosuppression designed predominantly to target T-cell-mediated pathways.[1] The pathogenesis of ABMR is characterized by donor-specific antibodies against HLA or non-HLA molecules expressed on the donor endothelium.[2] These antibodies bind to the microvascular endothelium and trigger complement activation, natural killer cell recruitment, and microvascular inflammation, resulting in endothelial injury, thrombosis, ischemia, and progressive graft damage.[3,4]

The accurate diagnosis of ABMR relies heavily on the Banff classification, which has undergone continuous refinement since its inception to better capture the spectrum of antibody-mediated allograft injury. The current diagnostic criteria for ABMR integrate distinct lines of evidence: histological lesions indicating tissue injury (such as glomerulitis and peritubular capillaritis), evidence of current or recent antibody interaction with the vascular endothelium (typically assessed via C4d staining or validated molecular transcripts), and serological evidence of circulating DSAs.[5-7] Recent advancements in molecular diagnostics and transcriptomic profiling have further improved our ability to detect early and subtle endothelial cell injury, enabling intervention before extensive structural remodeling occurs.[8]

The therapeutic management of ABMR remains one of the most formidable challenges in solid organ transplantation. The standard of care primarily involves strategies designed to rapidly remove circulating antibodies and modulate the B-cell immune response. Plasma exchange (PE) serves as the cornerstone of this approach, mechanically clearing DSAs and other inflammatory mediators from the patient's circulation.[9,10] PE is rarely utilized in isolation; it is most commonly administered in conjunction with intravenous immunoglobulin (IVIG) to neutralize residual antibodies and suppress further antibody production, alongside other immunomodulatory agents.[11,12]

Clinical evidence indicates that the reversibility of ABMR depends significantly on the chronicity of the underlying tissue injury at the time of intervention. Acute ABMR, which presents primarily with active microvascular inflammation and an absence of chronic structural changes, may be highly responsive to prompt antibody depletion therapies.[13,14] Conversely, chronic active ABMR represents a more advanced disease state marked by irreversible structural adaptations, including glomerular basement membrane double contours and extensive interstitial fibrosis.[15]

METHODS

Study design and participants

This prospective observational study was conducted at the Institute of Kidney Diseases and Research Centre–Institute of Transplantation Sciences (IKDRC-ITS), Ahmedabad, Gujarat, India, between September 2017 and November 2019. Adult kidney transplant recipients with biopsy-proven ABMR according to Banff 2017 criteria were enrolled. Patients were classified as acute ABMR (n=30) or chronic ABMR (n=30). Exclusion criteria included age <12 years, pregnancy, maintenance dialysis dependence, eGFR <15 ml/min/1.73 m², active infection, or incomplete follow-up.

Treatment protocol

All patients underwent plasma exchange in combination with intravenous immunoglobulin, corticosteroids, and optimization of maintenance immunosuppressive therapy according to institutional protocols. The number of plasma exchange sessions was individualized based on clinical, immunological, and histopathological severity.

Data collection and outcomes

Baseline demographic, clinical, laboratory, and transplant-related variables were recorded. Histopathological assessment included Banff lesion scores for C4d deposition, glomerulitis, peritubular capillaritis, vasculitis, chronic glomerulopathy, and interstitial fibrosis/tubular atrophy. Renal outcomes were assessed at 6 months and 5 years. Long-term clinical outcomes included hospitalization, infection-related admission, cardiovascular events, malignancy, mortality, and graft loss requiring maintenance dialysis.

Statistical analysis

Continuous variables are presented as mean±standard deviation and categorical variables as number (%). Between-group comparisons were performed using Student’s t-test, Chi-square test, or Fisher’s exact test, as appropriate. Within-group histological comparisons were performed using paired t-test or Wilcoxon signed-rank test. Hospitalization episodes were compared using the Mann–Whitney U test. A two-sided $p<0.05$ was considered statistically significant. The design, conduct, analysis, and reporting of this prospective observational study adhered to the strengthening the reporting of observational studies in epidemiology (STROBE) statement.

RESULTS

Baseline characteristics

Sixty patients with biopsy-proven ABMR were included, comprising 30 patients each with acute and chronic ABMR. Baseline demographic, transplant-related, and clinical characteristics were comparable between groups, with no significant differences in age, sex, donor age, HLA matching, prior transplantation, desensitization history, medication non-adherence, or changes in immune-suppressive therapy (Table 1).

Baseline laboratory parameters, including serum creatinine, eGFR, proteinuria, DSA levels, hematological indices, and biochemical variables, were also similar between groups (Table 2).

Histopathological response

Acute ABMR demonstrated significant improvement in active Banff lesions following plasma exchange, including C4d deposition, glomerulitis, peritubular capillaritis, and vasculitis (all p≤0.002), whereas chronic injury scores remained unchanged (Table 3). In contrast, chronic ABMR showed no significant improvement in either active or chronic histological lesions at 6 months (Table 4).

Renal outcomes

At 6 months, acute ABMR was associated with significantly better renal function, lower proteinuria, and lower DSA levels compared with chronic ABMR (all p<0.001; Table 5).

These differences persisted during long-term follow-up. At 5 years, patients with acute ABMR maintained superior renal function, lower proteinuria, and lower persistent DSA positivity than those with chronic ABMR (all p<0.001; Table 6).

Long-term clinical outcomes

Chronic ABMR was associated with significantly greater long-term morbidity. Compared with acute ABMR, chronic ABMR resulted in more hospitalization episodes (p<0.001), infection-related admissions (p=0.004), cardiovascular events (p=0.002), mortality (p=0.044), and graft loss requiring maintenance dialysis (p=0.026). Although malignancy occurred more frequently in chronic ABMR, the difference was not statistically significant (p=0.085) (Table 7).

Table 1: Baseline demographic and clinical characteristics of patients with acute and chronic ABMR.

Variables	Acute ABMR (n=30)	Chronic ABMR (n=30)	P value
Age (years), mean±SD	31.1±12.9	35.0±13.0	0.25
Male sex, N (%)	17 (56.7)	18 (60.0)	0.80
Donor age (years), mean±SD	45.7±12.5	44.9±12.6	0.82
HLA match, mean±SD	3.5±2.4	4.1±2.7	0.37
HLA <3 match, N (%)	18 (60.0)	14 (46.7)	0.29
HLA 4–6 match, N (%)	8 (26.7)	10 (33.3)	0.58
HLA 7–10 match, N (%)	4 (13.3)	6 (20.0)	0.48
Prior kidney transplantation, N (%)	5 (16.7)	2 (6.7)	0.23
Desensitization history, N (%)	4 (13.3)	1 (3.3)	0.16
History of drug non-adherence, N (%)	12 (40.0)	13 (43.3)	0.80
History of change in immunosuppressive therapy, N (%)	7 (23.3)	11 (36.7)	0.27

Values are presented as mean±SD or N (%). Continuous variables were compared using Student's t-test. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. p<0.05 was considered statistically significant, ABMR: antibody-mediated rejection; HLA: human leukocyte antigen; SD: standard deviation

Table 2: Baseline laboratory parameters at presentation in acute and chronic ABMR.

Parameters	Acute ABMR (n=30)	Chronic ABMR (n=30)	P value
Serum creatinine (mg/dl)	3.32±1.1	3.26±1.0	0.78
eGFR (ml/min/1.73 m²)	21.1±6.5	24.7±7.2	0.11
24-hour proteinuria(g/day)	2.87±1.2	2.84±1.1	0.92
DSA level (MFI)	3446±950	3420±900	0.88
Hemoglobin (g/dl)	10.5±1.4	10.2±1.5	0.41
Total leukocyte count (×10⁹/l)	6.8±1.9	7.1±2.0	0.52
Platelet count (×10⁹/l)	220±60	210±55	0.47
Serum sodium (mEq/l)	136±4	135±5	0.36
Serum potassium (mEq/l)	4.8±0.6	4.9±0.7	0.58
Serum calcium (mg/dl)	8.5±0.8	8.3±0.7	0.29
Serum phosphorus (mg/dl)	5.2±1.1	5.4±1.2	0.44

Values are presented as mean±SD, comparisons between groups were performed using student's t-test. p<0.05 was considered statistically significant, eGFR: estimated glomerular filtration rate; DSA: donor-specific antibody; MFI: mean fluorescence intensity; SD: standard deviation

Table 3: Banff histological scores in acute ABMR before plasma exchange and at 6-month follow-up.

Banff score	Before PE	At 6 months	P value
C4d score	2.20	0.43	<0.001
Glomerulitis (g score)	1.77	0.33	<0.001
Peritubular capillaritis (ptc score)	2.10	0.30	<0.001
Vasculitis (v score)	1.20	0.17	0.002
Chronic glomerulopathy (cg score)	0.17	0.17	1.000
IF/TA score	0.20	0.20	1.000

Values are presented as mean Banff scores, comparisons between baseline and 6-month follow-up were performed using the paired Student's t-test. p<0.05 was considered statistically significant, PE: Plasma exchange; C4d: complement component 4d; g: glomerulitis; ptc: peritubular capillaritis; v: vasculitis; cg: chronic glomerulopathy; IF/TA: interstitial fibrosis/tubular atrophy

Table 4: Banff histological scores in chronic ABMR before plasma exchange and at 6-month follow-up

Banff score	Before PE	At 6 months	P value
C4d score	1.17	0.90	0.118
Glomerulitis (g score)	0.53	0.43	0.317
Peritubular capillaritis (ptc score)	0.67	0.43	0.089
Vasculitis (v score)	0.40	0.27	0.206
Chronic glomerulopathy (cg score)	1.10	1.23	0.284
IF/TA score	1.63	1.77	0.248

Values are presented as mean Banff scores, Comparisons between baseline and 6-month follow-up were performed using the Wilcoxon signed-rank test. p<0.05 was considered statistically significant, PE: plasma exchange; C4d: complement component 4d; g: glomerulitis; ptc: peritubular capillaritis; v: vasculitis; cg: chronic glomerulopathy; IF/TA: interstitial fibrosis/tubular atrophy

Table 5: Renal outcomes at 6-month follow-up.

Parameter	Acute ABMR(n=30)	Chronic ABMR(n=30)	P value
Serum creatinine (mg/dl)	1.43±0.3	3.30±0.9	<0.001
eGFR (ml/min/1.73 m²)	57.7±9.0	27.1±7.5	<0.001
24-hour proteinuria (g/day)	0.29±0.1	2.23±0.7	<0.001
DSA level (MFI)	850±200	2630±600	<0.001

Values are presented as mean±SD, Comparisons between groups were performed using student's t-test. p<0.05 was considered statistically significant, eGFR: estimated glomerular filtration rate; DSA: donor-specific antibody; MFI: mean fluorescence intensity; SD: standard deviation

Table 6: Renal outcomes at 5-year follow up.

Parameter	Acute ABMR	Chronic ABMR	P value
Serum creatinine (mg/dl)	1.8±0.5	3.8±1.2	<0.001
eGFR (ml/min/1.73 m²)	52±12	25±8	<0.001
24-hour proteinuria (g/day)	0.6±0.3	2.5±1.0	<0.001
Persistent DSA positivity (%)	28±12	68±15	<0.001

Values are presented as mean±SD or percentage (%), comparisons between groups were performed using Student's t-test. p<0.05 was considered statistically significant, DSA: donor-specific antibody; eGFR: estimated glomerular filtration rate; SD: standard deviation

Table 7: Long-term clinical outcomes at 5-year follow-up.

Outcome	Acute ABMR (n=30)	Chronic ABMR (n=30)	P value
Hospitalization episodes	32	87	<0.001
Infection-related admissions	10	21	0.004
Malignancy	1	5	0.085
Cardiovascular events	4	15	0.002
Mortality	1	6	0.044
Graft loss requiring maintenance dialysis	1	8	0.026

Values are presented as number of events; hospitalization episodes were compared using the Mann-Whitney U test. Other categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. p<0.05 was considered statistically significant, ABMR: antibody-mediated rejection

DISCUSSION

The therapeutic landscape for antibody-mediated rejection emphasizes the critical distinction between acute active injury and chronic structural damage. A comprehensive review of the contemporary literature strongly supports the observation that outcomes following conventional antibody removal therapies, specifically plasma exchange, vary drastically based on the chronicity of the rejection episode. Numerous studies have documented that the prompt initiation of plasma exchange in the setting of acute ABMR can substantially ameliorate active immune-mediated injury.[16] When applied during the early phases of rejection, plasma exchange efficiently reduces the circulating burden of donor-specific antibodies, thereby directly mitigating endothelial cell activation and halting the complement cascade. Histologically, this translates to a marked reduction in active Banff lesions, including C4d deposition in peritubular capillaries, glomerulitis, and peritubular capillaritis.[17-19] The capacity of plasma exchange to reverse these active inflammatory lesions underscores the critical window of opportunity that exists before persistent microvascular inflammation transitions into permanent tissue remodeling.[20]

In stark contrast, the literature paints a considerably more sobering picture regarding the efficacy of plasma exchange and standard immunomodulatory therapies in the context of chronic active ABMR. Chronic ABMR is defined by established structural deterioration, predominantly manifesting as transplant glomerulopathy, multi-layering of peritubular capillary basement membranes, interstitial fibrosis, and tubular atrophy.[21,22] These pathognomonic features represent the culmination of indolent, sustained endothelial injury and repetitive cycles of tissue repair. Extensive transcriptomic and histopathological studies have confirmed that the architectural distortions seen in transplant glomerulopathy are essentially irreversible.[23,24] Consequently, while plasma exchange may successfully lower circulating DSA titers in these patients, it often fails to restore normal allograft architecture or meaningfully alter the trajectory of declining renal function.[25,26]

The functional clinical outcomes documented in large-scale transplant registries closely mirror these histological realities. Patients diagnosed and treated during the acute phase of ABMR frequently experience significant improvements in clinical parameters, including robust reductions in serum creatinine, stabilization of estimated glomerular filtration rate (eGFR), and resolution of proteinuria.[27,28] Furthermore, successful clearance or significant reduction of DSA levels following acute intervention correlates tightly with improved mid-to-long-term graft survival. Conversely, patients with established chronic ABMR characteristically exhibit inexorable disease progression despite aggressive standard-of-care interventions. Persistent allograft dysfunction, progressive proteinuria, and relentless deterioration of eGFR remain the norm in this population.[29-31]

Long-term observational cohorts and registry analyses further accentuate the profound prognostic divergence between acute and chronic ABMR. Sustained successful treatment of acute ABMR has been firmly linked to superior renal preservation, significantly lower rates of chronic DSA persistence, reduced hospitalization frequencies, and lower overarching healthcare burdens.[32] In these cohorts, preserving allograft function directly translates to a reduction in cardiovascular morbidity and overall mortality, given the well-established survival advantage of a functioning kidney transplant over return to dialysis.[33] The inability of conventional therapies to arrest the progression of chronic ABMR dictates a poor long-term prognosis for these patients, characterized by high rates of return to end-stage renal disease.[32,33]

Recognizing the stark limitations of plasma exchange and IVIG in reversing chronic tissue injury, the transplant community has increasingly pivoted towards exploring novel therapeutic avenues that target alternative nodes of the humoral immune response. Emerging literature highlights the potential of therapies aimed specifically at the cellular sources of antibody production and the downstream amplifiers of tissue injury. Interleukin-6 (IL-6) signaling blockade, for instance, has demonstrated promise in modulating the inflammatory milieu and directly suppressing terminal B-cell differentiation, offering a potential strategy for stabilizing chronic active ABMR.[34] Similarly, biological agents targeting CD38-positive long-lived plasma cells are being actively investigated to deplete the cellular reservoirs responsible for continuous DSA generation.[35] Complement cascade inhibitors, while highly effective at blocking terminal membrane attack complex formation, have shown mixed results but remain a critical area of investigation for specific phenotypes of complement-activating ABMR.[36] The integration of these targeted therapies with highly sensitive molecular phenotyping holds the promise of ushering in an era of personalized medicine in transplantation. By tailoring interventions to the specific immunological and transcriptomic profile of the allograft, clinicians may finally bridge the therapeutic gap in the management of chronic ABMR.

Limitations

The present study has several limitations. It was conducted at a single center with a relatively small sample size. Donor-specific antibody subclass analysis and molecular profiling were not performed. Larger multicenter studies are required to validate these findings and determine optimal therapeutic strategies for chronic antibody-mediated rejection.

CONCLUSION

Antibody-mediated rejection remains a major cause of kidney allograft dysfunction and loss. Standard therapies, particularly plasma exchange–based regimens, are most effective when initiated early before irreversible chronic

injury develops. Once chronic structural changes such as transplant glomerulopathy and interstitial fibrosis are established, treatment responses are limited. Therefore, early diagnosis, timely intervention, and the development of targeted immunomodulatory therapies are essential to improve long-term graft survival and patient outcomes.

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