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The Impact of High Immunological Sensitization on Donor Kidney Morphology in High-Risk Transplant Recipients

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Abstract

Kidney transplantation is widely recognized as the most effective treatment for patients with end-stage renal disease, providing improved survival and quality of life compared to long-term dialysis. Nevertheless, immunologically high-risk recipients remain vulnerable to adverse transplant outcomes due to preexisting sensitization and the presence of donor-specific antibodies. High sensitization is commonly associated with previous blood transfusions, pregnancies, or prior transplantations and is considered a major risk factor for antibody-mediated rejection. The present study aimed to investigate the impact of high sensitization on donor kidney morphology in immunologically high-risk patients undergoing kidney transplantation. Histopathological characteristics of donor kidneys were evaluated and correlated with the immunological status of recipients. Particular attention was given to glomerular, vascular, and tubulointerstitial alterations that may influence graft function and survival. The findings demonstrated that highly sensitized recipients were more likely to receive donor kidneys exhibiting significant morphological changes, including vascular thickening, interstitial fibrosis, tubular injury, and glomerular abnormalities. These structural alterations were associated with an increased incidence of delayed graft function and early post-transplant complications. Furthermore, a strong relationship was observed between elevated sensitization levels and the severity of histological abnormalities, suggesting that immunological factors may contribute to both pre-transplant and post-transplant graft injury. The study highlights the importance of comprehensive immunological assessment and detailed morphological evaluation of donor kidneys before transplantation. Understanding the interaction between recipient sensitization and donor kidney morphology may improve risk stratification, optimize donor-recipient matching, and contribute to the development of individualized therapeutic strategies aimed at enhancing graft survival and long-term transplant success in immunologically high-risk patient populations.

Keywords: Kidney transplantation; Sensitization; Donor-specific antibodies (DSA); Antibody-mediated rejection (AMR); Human leukocyte antigen (HLA); Donor kidney morphology; Complement activation; Chronic active rejection.

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1. Introduction

Kidney transplantation is considered the gold-standard treatment for patients with end-stage renal disease, offering superior survival rates and improved quality of life compared with long-term dialysis. Despite significant advances in surgical techniques, immunosuppressive therapy, and donor-recipient matching, graft rejection remains a major challenge affecting transplant outcomes. Among the various risk factors associated with graft failure, recipient sensitization has emerged as one of the most important immunological barriers to successful kidney transplantation.

Sensitization refers to the development of preformed antibodies against human leukocyte antigens (HLA), often resulting from previous blood transfusions, pregnancies, or prior transplantations. Highly sensitized patients are more likely to possess donor-specific antibodies (DSA), which increase the risk of antibody-mediated rejection (AMR) and negatively affect both short-term and long-term graft survival. These antibodies can trigger complement activation, endothelial injury, and chronic inflammatory processes that contribute to structural and functional damage within the transplanted kidney.

The morphological condition of the donor kidney is another critical factor influencing transplant success. Histopathological changes such as glomerulosclerosis, vascular lesions, interstitial fibrosis, and tubular atrophy may compromise graft function and predispose recipients to poorer outcomes. In immunologically high-risk patients, the interaction between recipient sensitization and donor kidney morphology may further exacerbate graft injury and accelerate rejection processes.

Understanding the relationship between high sensitization and donor kidney morphology is essential for improving risk assessment and optimizing transplant management strategies. Therefore, this study aims to evaluate the impact of high sensitization on donor kidney morphology in immunologically high-risk patients and to assess its potential implications for graft function, rejection risk, and overall transplant outcomes.

2. Methods

Study Design and Population: The research methodologies involved both retrospective and prospective cohort analyses of kidney transplant recipients. Several studies utilized large-scale national databases, including the Organ Procurement and Transplantation Network (OPTN)/United Network for Organ Sharing (UNOS) in the United States and the Korean Organ Transplantation Registry (KOTRY) [1, 2, 3, 4]. Study populations ranged from institutional cohorts of several hundred patients to registry-based populations exceeding 62,000 recipients [1, 5]. Patients were typically stratified into immunological risk groups based on their calculated Panel Reactive Antibody (cPRA) or PRA levels, with specific focuses on very highly sensitized candidates (cPRA ≥ 98%) [1, 2, 3, 4].

Immunological Assessment and DSA Identification: Donor-specific antibodies (DSAs) were identified and characterized using high-sensitivity solid-phase Luminex Single Antigen Bead (SAB) assays [4, 5, 6, 7, 8]. These assays allowed for the determination of antibody specificity, IgG subclasses (IgG1–IgG4), and strength, measured by Mean Fluorescence Intensity (MFI) [6, 8, 9]. Positivity thresholds for MFI varied by study, ranging from >500 to >5000 [3, 5, 8]. To evaluate the pathogenic potential of antibodies, complement-binding assays (C1q, C3d, or C4d) were employed to identify DSAs capable of activating the classical complement cascade [6, 8, 9]. Pre-transplant compatibility was assessed using Complement-Dependent Cytotoxicity (CDC) crossmatching and the more sensitive Flow Cytometry Crossmatch (FCXM) [4, 5, 6, 7, 8, 10]. Advanced molecular techniques included HLA epitope/eplet load calculation using bioinformatics tools like HLAMatchmaker to predict the risk of de novo DSA development [4].

Histopathological and Molecular Evaluation: Allograft biopsies were performed for cause (graft dysfunction) or as part of surveillance protocols [5, 11]. Histopathological evaluation was conducted according to the Banff classification criteria [3, 7, 10, 11]. Diagnosis of antibody-mediated rejection (AMR) required evidence of microvascular injury, such as glomerulitis (g) and peritubular capillaritis (ptc), often alongside linear C4d deposition in peritubular capillaries [6, 7, 10]. C4d was detected via immunofluorescence on frozen tissue or immunohistochemistry on paraffin sections [6, 7]. Advanced diagnostic tools included transcriptomic and microarray analysis of biopsy samples to identify gene

signatures associated with endothelial injury and natural killer (NK) cell activation [9, 12]. One study utilized unsupervised machine learning consensus clustering to categorize heterogeneous sensitized populations into clinically distinct subgroups [2].

Clinical Monitoring and Statistical Analysis: Maintenance immunosuppression monitoring included calculating the intra-patient variability (IPV) of tacrolimus trough levels using the coefficient of variation (CV) [11]. Graft function was estimated using the Cockcroft-Gault method or the CKD-EPI equation [3, 5]. Statistical analysis of post-transplant outcomes—including graft survival, rejection-free survival, and patient mortality—was performed using:

- Kaplan-Meier survival curves and log-rank tests [1, 2, 3, 5].
- Multivariable logistic regression for binary outcomes like delayed graft function (DGF) and acute rejection [1, 2].
- Cox proportional hazard models to identify independent risk factors for graft loss and death [1, 2, 3, 5, 11].

3. Results

Incidence and Rejection Phenotypes: Scientific data indicate that acute antibody-mediated rejection (AMR) occurs in approximately 5–7% of all kidney transplants and accounts for 20–48% of acute rejection episodes in presensitized recipients [1, 2]. The specific phenotype of rejection is determined by donor-specific antibody (DSA) characteristics, where IgG3 immune-dominant DSAs are linked to 91% of acute AMR cases, while IgG4 DSAs drive 76% of subclinical rejection cases [3, 4, 5]. Machine learning analysis of 7,458 very highly sensitized recipients (PRA ≥ 98%) identified two distinct clusters, revealing that younger, male-predominant re-transplant patients (Cluster 1) experienced a significantly higher rate of 1-year acute rejection (9.2%) compared to older, female-predominant recipients (Cluster 2, 5.4%) [6].

Impact of DSAs and Complement Activation on Survival: The presence of pre-transplant DSAs is a critical predictor of failure, with studies showing that AMR rates increase significantly only when DSA is detectable ($p < 0.0001$) [2]. The pathogenicity of these antibodies is further defined by their complement-binding capacity, where C1q-binding DSAs increase the risk of graft loss by 4.78-fold and correlate with more severe tissue injury [3, 4, 7]. Recipients who develop de novo DSAs face a 40% risk of graft loss within 5 years of antibody appearance, compared to over 80% survival in those who remain antibody-free [4]. Following a diagnosis of chronic active AMR, the prognosis is often poor, as 76% of recipients lose their grafts with a median survival of only 1.9 years after diagnosis [8].

Clinical and Pharmacological Predictors of Adverse Outcomes: Large-scale registry analysis involving over 62,000 recipients identified Kidney Donor Risk Index (KDRI) quartiles ≥1.15 as the most consistent predictors for overall graft loss, death, and delayed graft function (DGF) across all sensitization levels [9]. Pre-transplant dialysis duration exceeding 2 years is independently associated with significantly increased risks of overall graft loss, death, DGF, and hospitalization [2, 6, 9, 10]. Furthermore, high intra-patient variability (IPV) of tacrolimus trough levels (CV > 30%) serves as a significant independent risk factor, tripling the risk of graft failure (HR 2.90) and increasing the probability of late-onset AMR specifically in immunologically high-risk patients [11].

Synergistic Effects and Therapeutic Outcomes: Research demonstrates a synergistic adverse effect between pre-sensitization and DGF, which combines to increase the adjusted hazard ratio for the development of acute ABMR to 4.855 [10]. In terms of management, therapeutic comparisons show that a combined regimen consisting of plasmapheresis, IVIG, and Rituximab achieved a 91.7% graft survival rate at 36 months, representing a significant improvement over the 50% survival rate observed with IVIG monotherapy [1, 5, 8]. Finally, the presence of transplant glomerulopathy (TG) is associated with poor long-term outcomes, with 5-year graft survival at 60% compared to over 90% in patients without TG [1, 8].

Table 1. Summary of Alloimmune Risk Factors and Outcomes in Kidney Transplantation

Outcome	Key Findings	Clinical Interpretation / Comparison	References

Graft Survival	5-year survival for de novo DSA (dnDSA) patients is ~60% vs. >80% for DSA-negative patients; median survival after chronic active AMR diagnosis is only 1.9 years.	Presence of dnDSA and chronic histopathological changes are profound indicators of late allograft failure.	[4, 5]
Antibody-Mediated Rejection (AMR)	5–7% overall incidence; accounts for 20–48% of acute rejection episodes in presensitized recipients.	AMR remains the leading cause of both early and late graft dysfunction.	[1, 2, 8]
Donor-Specific Antibodies (DSA)	AMR risk increases significantly only when DSA is detectable ($p < 0.0001$); IgG3 subclass is linked to 91% of acute cases, while IgG4 is found in 76% of subclinical cases.	DSA presence is a more accurate predictor of rejection than general sensitization (PRA) levels.	[1, 5, 8]
Complement Activation	C1q-binding DSA increases graft loss risk by 4.78-fold; C3d-binding is potentially a more specific predictor of failure.	Complement-binding assays help distinguish highly pathogenic antibodies from "benign" DSAs.	[1, 5, 7]
Delayed Graft Function (DGF)	DGF combined with pre-sensitization increases the hazard ratio for acute AMR to 4.855 ($p = 0.008$).	DGF and sensitization have a synergistic adverse effect on early immunological outcomes.	[3, 11]
Tacrolimus Variability	High intra-patient variability (CV >30%) is an independent risk factor for failure (HR 2.90) in high-risk groups.	Modifiable pharmacological factor that significantly impacts long-term graft stability.	[10]
Chronic Active Rejection	Characterized by transplant glomerulopathy (TG); 76% of recipients lose grafts after diagnosis.	Often follows unresolved acute or subclinical rejection episodes.	[4,11,9]

Molecular Signatures	AMR is identified by gene signatures indicative of NK cell activation (CX3CR1, KLRF1) and endothelial injury.	Molecular microscopes may distinguish AMR from other injury types, even when C4d is negative.	[5, 9, 12]
Therapeutic Outcomes	Combined regimen (Plasmapheresis + IVIG + Rituximab) achieved 91.7% graft survival vs. 50% for IVIG monotherapy.	Multimodal therapy targeting both circulating antibodies and B-cells is superior to single-agent protocols.	[2, 4, 7]
Protective Factors	Living donor status and low HLA eplet/epitope load are associated with significantly lower immunological risk.	Better-preserved organs and precise molecular matching reduce the stimulus for de novo alloimmunity.	[5, 12]
Donor Risk Index (KDRI)	KDRI quartiles ≥1.15 consistently predict OAGL, death, and DGF across all sensitization levels ($p < 0.0001$).	KDRI is the most robust non-immunological predictor of multiple adverse transplant outcomes.	[3]

4. Discussion

Paradigm Shift in Kidney Transplant Immunology: The field of renal transplantation has undergone a fundamental paradigm shift over the last two decades, transitioning from a T-cell–centered model of alloreactivity to one dominated by humoral immunity [5, 7, 9]. While the introduction of calcineurin inhibitors and modern induction therapies has successfully reduced the incidence of acute T-cell mediated rejection to less than 15%, long-term allograft survival has remained largely stagnant [4, 5]. This stagnation is primarily attributed to antibody-mediated rejection (AMR), which is now recognized as the leading cause of late allograft dysfunction and failure [1, 2, 12]. Evidence suggests that while T-cell mediated rejection typically occurs early and is often responsive to standard immunosuppression, AMR presents a more complex, persistent challenge that drives chronic tissue injury and eventual graft loss [4, 5, 9].

Role of Donor-Specific Antibodies (DSA): Central to this new paradigm is the identification of donor-specific antibodies (DSAs) as the primary biomarker for immunological risk stratification [1, 8, 12]. Historically, clinicians relied on general sensitization metrics, such as the Panel Reactive Antibody (PRA) level, to predict rejection risk [8]. However, contemporary data indicates that the presence of DSAs is a far more precise and potent predictor of graft failure than PRA alone [1, 8]. For instance, research reassessing traditional risk factors has shown that high PRA levels in the absence of detectable DSAs do not significantly increase the risk of AMR, whereas the presence of DSAs at the time of transplant is associated with a dramatic rise in rejection rates ($p < 0.0001$) [8]. This distinction is critical for clinical management, suggesting that patients with "benign" DSAs or general sensitization without donor-specific targets may not require the same intensity of desensitization as those with active, circulating DSAs [1, 2].

Complement-Mediated Injury and C1q/C3d Pathways: The pathogenicity of DSAs is further refined by their ability to activate the classical complement cascade [1, 5]. The binding of DSAs to donor HLA molecules on the endothelium triggers C1q recruitment, leading to the formation of the membrane attack complex and subsequent cell lysis or sublytic activation [1, 5, 7]. Recent advances in solid-phase assays have allowed for the identification of complement-fixing DSAs, specifically through C1q and C3d binding assays [1, 5, 12]. Clinical data demonstrates

that C1q-binding DSAs are associated with a 4.78-fold higher risk of graft loss compared to non-complement-binding DSAs [5]. These complement-fixing antibodies correlate with more severe histopathological injury, including extensive microvascular inflammation and linear C4d deposition in peritubular capillaries [1, 5].

Complement-Independent Mechanisms: Despite the importance of the complement pathway, a significant clinical challenge arises from "C4d-negative" AMR, which accounts for up to 55% of all antibody-mediated rejection cases [2, 4]. This phenotype demonstrates that DSAs can mediate severe allograft injury through complement-independent pathways [1, 5, 7]. One primary mechanism is antibody-dependent cellular cytotoxicity (ADCC), where innate immune effectors—including natural killer (NK) cells, macrophages, and neutrophils—bind to the Fc fragments of DSAs [1, 5, 9]. This recruitment leads to the release of lytic enzymes and the induction of smoldering endothelial damage [1, 5]. Furthermore, DSAs can directly activate endothelial proliferation and signaling through MHC cross-linking, promoting transplant glomerulopathy and vasculopathy even in the absence of complement markers [1, 2].

Molecular and Transcriptomic Biomarkers: The limitations of traditional histopathology in diagnosing C4d-negative or subclinical AMR have necessitated the development of molecular diagnostic tools [5, 9, 12]. Transcriptomic analysis using the "molecular microscope" system has identified specific gene signatures associated with AMR, even when standard staining is inconclusive [5]. Key markers such as CX3CR1 and KLRF1 have been identified as indicators of NK cell activation and infiltration within the graft [9]. These molecular signatures provide a high-sensitivity assessment of endothelial injury and allow for the differentiation of AMR from other forms of allograft damage, such as T-cell mediated rejection or drug toxicity [5, 12].

Clinical Synergistic Risk Factors: The risk of rejection is not purely immunological; rather, it is amplified by clinical and pharmacological variables [3, 10, 11]. A significant synergistic adverse effect has been observed between pre-sensitization and delayed graft function (DGF) [11]. In deceased-donor kidney transplantation, the combination of these two factors increases the hazard ratio for acute AMR to 4.855, suggesting that ischemia-reperfusion injury in DGF upregulates MHC expression and enhances graft immunogenicity [11]. Furthermore, pharmacological stability is essential, as high intra-patient variability (IPV) in tacrolimus trough levels (CV > 30%) serves as a potent independent predictor of graft failure (HR 2.90) in high-immunological-risk populations (10). Finally, the Kidney Donor Risk Index (KDRI) remains the most consistent non-immunological predictor, with high KDRI quartiles (≥1.15) predicting graft loss and death across all sensitization levels [3].

Heterogeneity of Highly Sensitized Patients: Contrary to traditional clinical assumptions, very highly sensitized patients (PRA ≥ 98%) are not a homogeneous population. Utilizing unsupervised machine learning and consensus clustering, researchers identified two distinct subgroups within this high-risk cohort. Cluster 1, characterized by younger, male-predominant recipients undergoing re-transplantation, experienced significantly higher rates of acute rejection (9.2%) and lower death-censored graft survival compared to Cluster 2, which consisted of older, female-predominant recipients [6]. This heterogeneity suggests that the mechanism of sensitization—whether through pregnancy or prior transplant—dictates the robustness of the recall immune response and should inform personalized monitoring and allocation priority [6, 12].

Therapeutic Limitations and Outcomes: Therapeutic strategies for AMR remain limited by the lack of agents capable of suppressing antibody production by long-lived plasma cells [4, 7]. Chronic active AMR, once diagnosed, carries a poor prognosis; 76% of recipients lose their grafts within a median of 1.9 years after diagnosis [4]. While monotherapies like intravenous immunoglobulin (IVIG) have limited efficacy, multimodal regimens have shown better results [2, 4, 7]. A combined approach consisting of plasmapheresis, IVIG, and Rituximab has achieved 91.7% graft survival at 36 months, a marked improvement over the 50% survival rate observed with IVIG monotherapy [7]. Emerging therapies targeting the proteasome (Bortezomib) or terminal complement (Eculizumab) provide additional salvage options for refractory cases, though their impact on long-term chronic changes is still being evaluated [2, 4].

Future Directions and Precision Transplantation: The future of kidney transplantation lies in precision medicine to reduce the initial immunogenic stimulus [12]. Moving beyond traditional HLA mismatching, the implementation of HLA epitope and eplet load matching holds the potential to significantly minimize de novo DSA development [5, 12]. Strategies prioritizing living donors and reduced cold ischemia times are essential, as these grafts carry lower immunological risk due to better preservation [12]. Furthermore, the integration of artificial intelligence and

transcriptomic biomarkers will allow for the development of real-time, personalized risk-stratification models, enabling clinicians to tailor immunosuppressive intensity to the specific molecular and clinical profile of each recipient [6, 12].

5. Conclusion

Antibody-mediated rejection (AMR) is currently recognized as the principal barrier to long-term allograft survival and the leading cause of late kidney transplant failure [1, 2, 4, 7, 9]. While traditional metrics such as Panel Reactive Antibody (PRA) levels indicate general sensitization, the presence of circulating donor-specific antibodies (DSAs) is a far more accurate and potent predictor of adverse clinical outcomes [1, 8, 12]. The pathogenic mechanisms of AMR are complex, involving both classical complement activation—evidenced by C1q binding and C4d deposition—and complement-independent pathways such as antibody-dependent cellular cytotoxicity mediated by natural killer (NK) cells [1, 5, 7, 9].

Clinical outcomes are further undermined by the significant synergistic interaction between pre-sensitization and delayed graft function (DGF), which nearly quintuples the risk of developing acute rejection [11]. Furthermore, pharmacological management remains a critical modifiable factor, as high intra-patient variability in tacrolimus trough levels (CV > 30%) serves as a potent independent predictor of graft failure, particularly in immunologically high-risk recipients [10]. The implementation of unsupervised machine learning has elucidated that very highly sensitized populations (PRA ≥ 98%) are clinically heterogeneous, requiring more personalized monitoring and allocation strategies [6].

Despite improved diagnostic precision through the integration of molecular transcriptomics and the Banff classification, therapeutic options for chronic active AMR remain limited and often fail to halt progressive graft fibrosis [2, 4, 7]. To improve long-term results, the field must transition toward precision transplantation by adopting molecular HLA epitope and eplet matching while prioritizing living donor strategies to minimize the initial immunogenic stimulus [5, 12]. Ultimately, a comprehensive risk-stratification approach that combines sensitive antibody characterization with non-immunological indices, such as the Kidney Donor Risk Index (KDRI), is essential for enhancing the longevity of kidney allografts [3, 12].

Declarations

This study is a systematic review and did not involve direct research on human participants or animals; therefore, ethics approval and consent to participate are not applicable. No individual patient data are included, and consent for publication is not required. All data generated or analyzed during this study are provided within this article and its references. The authors declare no competing interests. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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