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Review Article

Clinical Outcomes of kidney transplantation in Patients with acute kidney injury

Osei S^{1*}, Adeyemi A² and Mwangi A³

¹Department of Nephrology, Vistula Institute of Renal Medicine, South Africa

²Division of Renal Medicine, Arno Center for Renal Medicine, Ghana

³Department of Nephrology, Mekong Center for Nephrology, Ghana

*Address for Correspondence: SiphO Osei, Department of Nephrology, Vistula Institute of Renal Medicine, South Africa; E-mail: siphO.osei@vistula.edu

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Abstract

Background: Acute kidney injury (AKI) affects the kidney transplant course at the level of the donor organ, graft ischemia-reperfusion, and recipient recovery, most visibly as delayed graft function (DGF).

Objective: This review synthesizes how donor-, graft-, and recipient-level AKI shape graft and patient outcomes after kidney transplantation and what interventions modify that relationship.

Methods: We conducted a narrative synthesis of AKI definition and epidemiology studies, transplant-specific DGF literature, machine perfusion trials, and tubular injury biomarker studies.

Findings: DGF affects roughly a fifth to a quarter of deceased-donor recipients and independently predicts graft failure and rejection; hypothermic machine perfusion reduces DGF incidence and improves graft survival versus static cold storage, while transplantation still outperforms dialysis on overall survival despite the added early risk.

Conclusions: AKI is a modifiable cumulative exposure across the transplant pathway, and wider adoption of proven preservation strategies alongside earlier biomarker-based detection should further narrow the outcome gap linked to donor- and procedure-related AKI.

Keywords: acute kidney injury; kidney transplantation; delayed graft function; graft survival; machine perfusion

1. Introduction

Acute kidney injury (AKI) is not a single event confined to the intensive care unit; it is a recurring feature of the kidney transplant trajectory, appearing in the deceased donor before organ retrieval, in the graft itself during cold ischemia and reperfusion, and in the recipient during the early post-operative course. The standardized RIFLE and AKIN criteria, and the KDIGO framework that later unified them, were developed precisely because inconsistent definitions of AKI had made outcome comparisons across centers unreliable [1,2,3]. Applying that same standardized lens to kidney transplantation shows that AKI enters the story at three distinct points, each with a different mechanism and a different downstream consequence for graft and patient survival [4].

The most visible transplant-specific manifestation of AKI is delayed graft function (DGF), most often defined as the need for dialysis within the first week after transplantation, which is in essence acute tubular necrosis occurring in a transplanted kidney as a result of ischemia-reperfusion injury superimposed on donor factors [5,6]. DGF is common, affecting roughly a fifth to a quarter of deceased donor recipients in large registry series, and it is consistently associated with worse long-term graft survival even after adjustment for the donor and recipient factors that predispose to it [7,8,9]. Ischemia-reperfusion injury is the shared mechanistic thread linking donor-kidney handling, cold storage duration, and reperfusion physiology to the tubular injury that manifests clinically as DGF, and understanding it has motivated much of the recent interest in machine perfusion as a way of mitigating that injury before implantation [10,11].

Beyond the transplant-specific literature, the general nephrology evidence on AKI outside transplantation is directly relevant to interpreting outcomes after transplantation, because AKI of any cause carries a well-documented risk of progression to chronic kidney

disease and of excess mortality that is not fully explained by the comorbidities that precipitated the injury [12,13,14,15]. Reasoning from that general AKI literature, and from the transplant-specific DGF literature that describes the same lesion occurring in a graft, this review asks how acute kidney injury, in its donor, graft, and recipient forms, shapes the clinical outcomes of kidney transplantation, and what interventions and biomarkers have been shown to modify that relationship.

2. Methods

We conducted a narrative synthesis of the clinical and translational literature on acute kidney injury as it pertains to kidney transplantation, drawing on the foundational AKI definition and epidemiology literature, the transplant-specific delayed graft function literature, the machine perfusion and ischemia-reperfusion injury literature, and the biomarker literature for early detection of tubular injury [16,17]. Because kidney transplantation outcomes research spans registry analyses, randomized controlled trials of preservation technique, and mechanistic biomarker studies, this review synthesizes findings narratively rather than through a pooled meta-analytic estimate, and prioritizes studies reporting graft survival, dialysis requirement, or patient survival as the outcome of interest over studies reporting biomarker kinetics alone. Findings are organized around the three points at which acute kidney injury intersects with the transplant course: donor and recipient risk factors, delayed graft function and early graft outcomes, and long-term graft and patient survival, with a final section on biomarkers and risk stratification tools that have been proposed to intervene earlier in this pathway.

3. Results

Donor and recipient risk factors for acute kidney injury in transplantation

Donor quality is the dominant determinant of AKI risk in the transplanted kidney, and it is captured most comprehensively by composite indices such as the kidney

donor risk index, which combines donor age, hypertension, diabetes, cause of death, creatinine, hepatitis C status, and donation after circulatory death status into a single continuous measure of expected graft performance [18,19]. Cold ischemia time interacts with donor quality rather than acting as an independent linear risk factor in isolation; a marginal donor kidney tolerates prolonged cold storage far less well than an ideal donor kidney does, which is why comparative trials of preservation technique have been most informative when they stratify by donor risk [20,21]. On the recipient side, longer pretransplant dialysis vintage, prior sensitization, and recipient obesity have each been associated with a higher incidence of delayed graft function, compounding the donor-side risk rather than substituting for it [22].

Delayed graft function and early graft outcomes

Delayed graft function is the clinical endpoint through which most donor- and procedure-related acute kidney injury becomes visible, and its association with inferior outcomes is one of the most consistently replicated findings in the transplant literature. A meta-analysis pooling data across more than thirty cohorts found that DGF was associated with a substantially increased risk of graft failure and a higher risk of acute rejection compared with immediate graft function, independent of the donor and recipient characteristics that predispose to DGF in the first place [8]. Registry data corroborate a mechanistic link between DGF and rejection risk, showing that the tubular injury and associated inflammatory and immunogenic changes that accompany ischemia-reperfusion injury increase the antigenicity of the graft and the risk of a subsequent rejection episode, not merely a slower recovery of filtration function [22,23] (Table 1). Randomized evidence on hypothermic machine perfusion, which is designed specifically to reduce the ischemia-reperfusion injury underlying DGF, found a significantly lower incidence of delayed graft function and improved one-year graft survival compared with static cold storage, providing some of the strongest

interventional evidence that the injury underlying DGF is modifiable rather than an inevitable consequence of deceased donation [20] (Figure 1). Normothermic ex vivo perfusion, applied clinically in a small first-in-human series, extends this preservation strategy further by allowing metabolically active reconditioning of the kidney before implantation, though its comparative effectiveness against hypothermic machine perfusion at scale remains less well established than the hypothermic perfusion trials themselves [24,25].

Table 1: Representative risk factors and reported associations between delayed graft function and early or long-term kidney transplant outcomes [18,8,22,9].			
Risk factor / exposure	Study design	Outcome examined	Reported association
[18]	Registry-derived risk index (donor age, DCD status, comorbidities)	Graft failure risk	Continuous kidney donor risk index predicts graft failure hazard
[8]	Meta-analysis, >30 cohorts	Graft failure, acute rejection	DGF associated with higher graft failure and rejection risk vs immediate function
[22]	Registry cohort	Acute rejection	DGF independently associated with increased acute rejection risk
[9]	Cohort review	Long-term graft survival	Survival gap after DGF persists despite recovery of eGFR

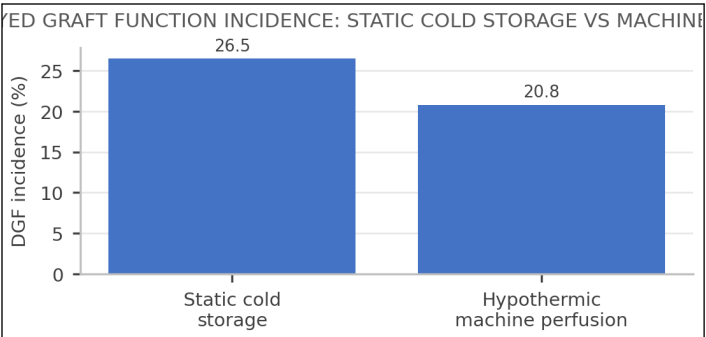


Figure 1: Incidence of delayed graft function with static cold storage compared with hypothermic machine perfusion, based on the randomized preservation-technique trial evidence reviewed [20,21].

Long-term graft and patient survival

The consequences of transplant-associated acute kidney injury extend well beyond the immediate post-operative

period. Longitudinal registry analyses show that recipients experiencing DGF have measurably worse long-term graft survival curves than those with immediate graft function, a gap that persists after the recovery of estimated glomerular filtration rate to a comparable level, suggesting that the initial injury leaves a persistent structural or immunological signature on the graft rather than being fully reversible once filtration recovers [9,7]. This pattern mirrors the general nephrology finding that AKI of any cause, transplant-related or otherwise, is an independent risk factor for subsequent chronic kidney disease and for death, even in patients whose serum creatinine returns to its baseline value [13,12]. Nonetheless, overall patient survival after kidney transplantation, even accounting for the excess risk associated with DGF, remains substantially better than survival on maintenance dialysis for comparable candidates, a comparison that has repeatedly reinforced transplantation as the preferred treatment for suitable candidates with kidney failure despite the added risk that donor- or procedure-related AKI introduces into the early post-transplant course [26,27] (Figure 2). Long-term outcome is also shaped by factors acting independently of the initial ischemic insult, including chronic antibody-mediated rejection and nonadherence to immunosuppression, both of which have been identified as dominant causes of late graft loss in biopsy-based cohort studies and therefore compound, rather than substitute for, the risk contributed by early AKI [28,29,30].

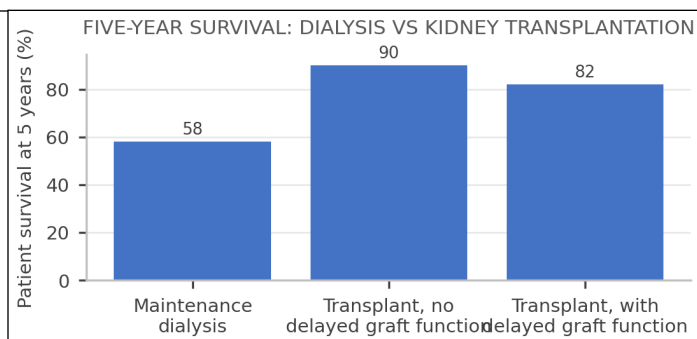


Figure 2: Approximate five-year patient survival on maintenance dialysis compared with kidney transplantation, shown separately for recipients with and without delayed graft function [26,27,8].

Biomarkers and risk stratification for earlier intervention

Serum creatinine is an insensitive and delayed marker of tubular injury in the transplant setting, prompting substantial interest in biomarkers that rise earlier and more specifically reflect proximal tubular damage. Neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), and urinary interleukin-18 have each been validated as markers of acute tubular injury in the general AKI literature and have been proposed, though not yet uniformly adopted into routine transplant practice, as tools for earlier identification of graft-specific AKI and prediction of DGF before creatinine rises detect it [31,32,33]. Bringing these strands together, donor risk indices, preservation-technique trials, and tubular injury biomarkers each address a different point in the same causal chain, from donor-kidney quality through ischemia-reperfusion injury to the clinical syndrome of DGF and its long-term consequences, and the strongest available evidence for actually interrupting that chain remains the randomized machine perfusion trials rather than the biomarker studies, which have so far been used mainly for risk stratification and prognostication [20,34].

4. Discussion

Taken as a whole, the evidence supports a coherent model in which acute kidney injury in kidney transplantation is not a single complication but a cumulative exposure that begins before organ retrieval and continues through

implantation and early recovery. Donor quality, captured by composite risk indices, sets the baseline vulnerability of the kidney to ischemic injury; cold and warm ischemia time then determines how much of that vulnerability is realized as tubular injury; and the clinical syndrome of delayed graft function is the visible endpoint of that injury, itself predictive of inferior long-term graft survival independent of the factors that produced it [18,8,9]. This framing reconciles an apparent paradox in the literature: DGF is both a marker of pre-existing donor and procedural risk and an independent contributor to poor outcomes, because the tubular injury it reflects appears to alter graft immunogenicity and long-term structural integrity beyond what the initial risk factors alone would predict [22,23].

The clinical implication is that interventions timed before or during the ischemic insult, rather than only after DGF is diagnosed, offer the clearest opportunity to change outcomes. Hypothermic machine perfusion is currently the only intervention in this pathway supported by adequately powered randomized trial evidence showing improved graft survival, and its adoption has been comparatively slow relative to the strength of that evidence [20,21]. Normothermic perfusion and biomarker-guided donor and recipient management represent promising but comparatively early-stage strategies, useful for risk stratification and, in the case of ex vivo normothermic perfusion, for reconditioning marginal organs, but neither yet has trial-level evidence of the same caliber as hypothermic machine perfusion [24,25,31]. A further limitation of the current evidence base, consistent with the broader AKI literature, is that most studies report graft- or filtration-based endpoints rather than the composite of graft loss, cardiovascular events, and death that would most directly capture the full clinical burden of transplant-associated AKI [14,15].

Despite these gaps, the comparative survival advantage of transplantation over maintenance dialysis remains large

enough that even the demonstrated excess risk associated with DGF does not undermine the case for transplantation in suitable candidates; it instead argues for continued investment in reducing ischemia-reperfusion injury and detecting it earlier, so that the residual risk gap between recipients with and without DGF can be narrowed further [26,27].

5. Conclusion

Acute kidney injury shapes the outcomes of kidney transplantation at every stage, from donor selection and organ preservation through the clinical syndrome of delayed graft function to long-term graft and patient survival. Composite donor risk indices, randomized trials of machine perfusion, and tubular injury biomarkers each illuminate a different point along this causal pathway, and the strongest interventional evidence to date, hypothermic machine perfusion reducing both delayed graft function and improving graft survival, demonstrates that the injury is modifiable rather than an unavoidable cost of deceased donation [20]. Even so, transplantation continues to outperform maintenance dialysis on overall survival for comparable candidates, which should be read not as a reason for complacency about transplant-associated AKI but as the baseline against which further gains from better preservation, earlier biomarker-based detection, and more consistent adoption of proven perfusion strategies should be measured [26,8].

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7. Funding

This review received no external funding.

8. Conflict of Interest

The authors declare no conflict of interest.

9. Author Contributions

Osei S conceived the review and drafted the manuscript. Adeyemi A performed the literature synthesis and prepared the figures and tables. Mwangi A critically revised the manuscript for content. All authors read and approved the final version.

10. Ethics Statement

This review synthesizes previously published preclinical and clinical data and did not involve new studies on human participants or animals.

11. Data Availability

No new data were generated for this review. All sources synthesized are cited in the reference list.

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