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Recovery of renal function and structure following acute kidney injury

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ABSTRACT

Recovery of renal function and structure following AKI is a complex and multifactorial process involving coordinated tubular epithelial regeneration, resolution of inflammation, restoration of microvascular integrity, and remodeling of the extracellular matrix. Clinical recovery is variable and influenced by injury severity, comorbidities, and therapeutic interventions. Advances in biomarkers and imaging techniques enhance early prediction and monitoring of recovery trajectories. Although no definitive therapies currently exist to reliably speed recovery, promising approaches, including stem cell secretome administration and growth factor modulation, are in development. Long-term consequences of incomplete structural repair highlight the importance of sustained patient follow-up after apparent recovery from AKI.

Keywords: Chronic kidney disease, Acute kidney injury, Renal recovery, Kidney function, Renal structure, Nephron regeneration, Tubular repair, Histology

Implication for health policy/practice/research/medical education:
Acute kidney injury (AKI) is a highly prevalent clinical syndrome characterized by an abrupt decline in renal function that manifests over hours to days. It is associated with increased morbidity and mortality worldwide and represents a significant challenge in both hospital and critical care settings. Although many patients experience some degree of recovery after AKI, the extent and completeness of renal functional and structural restoration can vary widely, with profound implications for long-term outcomes, including progression to chronic kidney disease and end-stage renal disease. Identification of mechanisms, clinical course, and predictors of renal recovery after AKI is thus critical for optimizing patient management and developing therapies to enhance repair.

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Review

Introduction

The recovery of renal function and structure following acute kidney injury (AKI) is a complex, multifaceted, and dynamically regulated biological process that engages a wide spectrum of cellular, molecular, and systemic mechanisms aimed at restoring the kidney's filtration, secretory, metabolic, and endocrine capacities after an insult (1-3). Acute kidney injury, a clinical syndrome characterized by a rapid decline in glomerular filtration rate, accumulation of nitrogenous waste products, and dysregulation of fluid, electrolyte, and acid-base homeostasis, affects millions of individuals annually worldwide and carries significant morbidity and mortality, particularly when complicated by multi-organ failure or when recovery is

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incomplete (4,5). Despite advances in supportive care, including renal replacement therapy and hemodynamic optimization, specific pharmacological interventions to accelerate or ensure complete renal recovery remain elusive (6). Consequently, identification of mechanisms underpinning renal repair and regeneration after AKI has become a central focus in nephrology research, with the critical goal of identifying therapeutic targets to enhance recovery, prevent transition to chronic kidney disease (CKD), and improve long-term patient outcomes (1).

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using various keywords like; chronic kidney disease, acute kidney injury, renal recovery, kidney function, renal structure, nephron regeneration, tubular repair, and histology

Renal function recovery following AKI

Recovery of kidney function following AKI is defined as a return toward baseline glomerular filtration rate (GFR) and resolution of biochemical abnormalities such as creatinine elevation and electrolyte derangements (6,7). However, the definition of renal recovery remains inconsistent and heterogeneous in the literature, complicating comparison across studies (8,9). Some define recovery as a return of serum creatinine to within 25% of baseline, others as dialysis independence, and yet others employ novel biomarkers to assess functional restoration or structural repair (10-12). Despite this variability, it is clear that complete recovery — reflected by normalization of both kidney function and histological integrity — occurs in a subset of patients, while others achieve only partial restitution or sustain permanent loss of nephron mass and function (1,13,14). Several factors influence the likelihood and speed of recovery including the initial severity and etiology of AKI, patient comorbidities such as pre-existing CKD or diabetes, and therapeutic interventions (15,16). At the molecular and cellular level, recovery from AKI entails tubular epithelial cell regeneration, resolution of interstitial inflammation, restoration of the microvascular and endothelial compartments, and remodeling of extracellular matrix (17). Injured renal tubular epithelial cells exhibit both necrotic and apoptotic death during the acute insult, leading to denudation of tubular basement membranes and loss of functional epithelial integrity (18,19). Surviving epithelial cells adjacent to denuded areas proliferate and migrate to repopulate the tubule, undergo de-differentiation to a progenitor-like state, and subsequently re-differentiate to restore mature functional cell types (20). Growth factors such as epidermal growth factor (EGF) and hepatocyte growth factor (HGF), as well as signaling pathways including hypoxia-inducible factor-1α (HIF-1α) and Wnt/β-catenin, play essential roles in promoting tubular repair and regeneration (21,22). Activation of the epidermal growth factor receptor (EGFR) has shown to stimulate tubular cell proliferation and enhance recovery after AKI experimentally, although persistent or excessive activation may contribute to maladaptive tubular atrophy and fibrosis in chronic injury contexts (23). Simultaneously, the renal microvascular compartment undergoes significant injury during AKI, with loss of peritubular capillaries and endothelial cell dysfunction contributing to local hypoxia and inflammation (24,25). Angiogenesis, the process of new capillary formation, is crucial in restoring oxygen delivery and supporting tubular repair (26). This vascular regeneration is modulated by factors such as vascular endothelial growth factor (VEGF), angiopoietins, and Notch signaling pathways (17,27). Controlled angiogenesis promotes recovery by reinstating perfusion and preventing ongoing ischemic damage, but dysregulated or excessive neovascularization can drive fibrotic remodeling and chronic kidney damage (28,29). Therapeutic approaches targeting angiogenesis, including exogenous VEGF administration and endothelial progenitor cell therapies, are under investigation to enhance recovery (30). The inflammatory milieu within the kidney after AKI also improves during recovery (30). Initially, AKI incites a robust inflammatory response characterized by activation of resident immune cells and infiltration of neutrophils, macrophages, and lymphocytes (31). Although necessary for removal of cell debris and initiation of repair, prolonged or dysregulated inflammation results in persistent interstitial fibrosis and loss of normal renal architecture (32,33). Resolution of inflammation is mediated by anti-inflammatory cytokines, regulatory immune cells, and clearance of pro-inflammatory mediators, marking a critical transition toward tissue repair (34). Experimental models highlight the role of mesenchymal stromal cells and their secreted factors in modulating inflammation and accelerating renal recovery, offering promising therapeutic avenues (35). It should remember that, clinically, renal recovery after AKI can be heterogeneous based on patient factors, underlying cause, and severity of injury (8,36). Recovery is often characterized into complete, partial, or no recovery (8). Complete recovery entails return of serum creatinine and GFR approximately to baseline levels, and independence from dialysis if it was initiated during AKI (8,37). Partial recovery reflects improvement from peak injury but with persisting renal dysfunction relative to baseline, while no recovery implies continued dialysis dependence or a persistent need for renal replacement therapy (RRT) (6). The time course of recovery from AKI can range from days

to weeks or months, with most patients achieving dialysis independence within one to three months after the injury (1,37). However, residual subclinical kidney injury often persists undetected by conventional markers even after apparent recovery and is associated with increased risks of CKD and long-term mortality (38).

Focus on predictors of recovery

Several clinical predictors of recovery have been identified (39). Patients with less severe AKI stages, absence of pre-existing CKD, younger age, and absence of multi-organ failure have higher likelihood of recovery (8,40). Biomarkers including urinary kidney injury molecule-1 (KIM-1), neutrophil gelatinase-associated lipocalin, and serum cystatin C have been studied for their roles in early prediction of recovery potential (41). Novel plasma and urine biomarkers reflecting tubular damage, repair, and inflammation provide more granular insights into the recovery process, potentially guiding prognostication and therapeutic decision making (42,43). Advanced imaging techniques such as multi-parametric magnetic resonance imaging (MRI) have been applied to assess renal structural and functional changes during AKI and recovery (44). Likewise, MRI parameters, including total kidney volume, cortical thickness, renal perfusion via arterial spin labeling, and blood oxygen level-dependent R2* mapping, reveal dynamic alterations in renal parenchymal oxygenation and microvascular function correlating with recovery phases (44). Despite biochemical normalization, some patients demonstrate persistent microvascular rarefaction and reduced cortical perfusion by MRI months to a year after AKI, indicating incomplete structural recovery, which may predispose to progressive CKD (44).

Treatment modalities

Renal replacement therapy techniques employed during AKI to support kidney function have been evaluated for their potential impact on recovery (45). Different modalities such as intermittent hemodialysis, continuous renal replacement therapy, and peritoneal dialysis show varying profiles in terms of hemodynamic stability and clearance of inflammatory mediators (46,47). However, no dialysis modality has conclusively demonstrated superiority in promoting renal recovery (48). Timing of initiation of RRT remains controversial, with recent prospective trials suggesting no clear benefit of early compared to delayed initiation on recovery rates (49,50). Focus has thus shifted toward refining patient selection and optimizing supportive care during the AKI recovery phase (51,52). Emerging therapeutic strategies to enhance renal recovery after AKI include administration of growth factors, stem cell and secretome-based therapies, and modulation of signaling pathways involved in repair (53). Mesenchymal stem cell-derived secretome, comprising cytokines, growth factors, and extracellular vesicles, has demonstrated in preclinical models the ability to accelerate recovery by reducing inflammation, promoting tubular regeneration, and enhancing angiogenesis (54,55). Translation to clinical practice is ongoing with early phase trials evaluating safety and efficacy (55). Pharmacologic agents targeting renal fibrosis, tubular cell proliferation, and microvascular repair are also under investigation (56). Long-term follow-up studies reveal that patients who recover from AKI, even those with normalized creatinine, remain at increased risk for future adverse outcomes including CKD progression, recurrent AKI episodes, cardiovascular events, and mortality (57,58). This underscores that renal recovery is a dynamic process extending well beyond the acute phase and necessitates ongoing surveillance (1, 8). Strategies to modulate maladaptive repair processes and fibrotic remodeling during convalescence may improve long-term renal health (59,60).

Structural recovery in the kidney post-AKI

Renal structural changes include tubular cell injury, interstitial inflammation, and fibrosis development, which can contribute to incomplete recovery and progression to CKD (61,62). Experimental treatments targeting inflammatory pathways, such as inhibition of IκB kinase, can improve structural and functional renal recovery and reduce fibrosis, underscoring the role of inflammation in repair processes (63). Meanwhile, antioxidants and anti-inflammatory agents have been shown experimentally to attenuate kidney injury and promote functional recovery (64,65). Additionally, cellular therapies like adipose tissue-derived stromal vascular fraction injections also demonstrate potential to improve structural recovery after ischemia/reperfusion injury in experimental models (66).

Conclusion

The recovery of renal function and structure following AKI is a highly orchestrated, multicellular, and multilayered process that integrates epithelial regeneration, immune modulation, vascular repair, matrix remodeling, metabolic adaptation, and epigenetic reprogramming. While the kidney possesses a remarkable capacity for self-repair, this capacity is finite and can be overwhelmed by severe or repetitive insults, comorbidities, or maladaptive responses. Advances in molecular biology, immunology, metabolomics, and systems biology are providing unprecedented insights into the mechanisms of renal repair, revealing novel therapeutic targets and strategies. Future directions include the development of combination therapies that simultaneously target multiple pathways (e.g., anti-inflammatory + pro-regenerative +

antifibrotic), the use of biomimetic scaffolds or organoids to replace irreversibly lost nephrons, and the application of artificial intelligence to integrate multi-omics data and predict individual repair trajectories. Finally, the goal is not merely to restore renal function to baseline but to achieve true biological restoration—reconstitution of nephron architecture, microvascular networks, and metabolic homeostasis—thereby preventing the insidious progression to CKD and improving the quality and longevity of life for AKI survivors.

Authors' contribution

Conceptualization: Sina Salem Ahim, Rasoul Jafari Arismani, and Shirin Shamsghahfarokhi.
Data curation: Iman Zafar Asoodeh, Hamid Rastad, and Farid Rahbari.
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Resources: All authors.
Supervision: All authors.
Validation: Sina Salem Ahim and Hamid Rastad.
Visualization: Shirin Shamsghahfarokhi and Hooman Esfahani
Writing–original draft: All authors.
Writing–review and editing: All authors.

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity.ai to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, and double publication) have been completely observed by the authors.

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