Opinion

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Journal of Renal Medicine

2025
Vol 08, No.1:51

Acute Kidney Injury: Pathophysiology, Biomarkers and Therapeutic Strategies

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Received date: February 01, 2025, Manuscript No. ipjrm-25-20533; **Editor assigned date:** February 03, 2025, PreQC No. ipjrm-25-20533 (PQ); **Reviewed date:** February 15, 2025, QC No. ipjrm-25-20533; **Revised date:** February 22, 2025, Manuscript No. ipjrm-25-20533 (R); **Published date:** February 28, 2025, DOI: 10.36648/ipjrm.8.1.51

Citation: Rassy A (2025) Acute Kidney Injury: Pathophysiology, Biomarkers and Therapeutic Strategies. J Ren Med Vol.8 No.1: 51.

Introduction

Acute kidney injury (AKI) is a common clinical syndrome characterized by a sudden decline in renal function, leading to impaired clearance of metabolic waste products, electrolyte imbalance, and fluid dysregulation. It is frequently encountered in hospitalized patients, particularly in critical care and surgical settings, and is associated with high rates of morbidity and mortality. The causes of AKI are multifactorial, ranging from prerenal factors such as hypovolemia and sepsis, to intrinsic renal damage from ischemia or nephrotoxic agents, and postrenal obstruction. Despite advances in medical care, AKI remains a major healthcare challenge due to its heterogeneous etiology, complex pathophysiology, and long-term risk of progression to chronic kidney disease (CKD). Recent research has emphasized the need for early detection and timely intervention in AKI to improve patient outcomes. Traditional diagnostic markers such as serum creatinine and urine output are limited by their delayed response and poor sensitivity to early renal injury. Novel biomarkers and emerging therapeutic strategies are now under investigation to provide more accurate diagnosis, risk stratification, and effective treatment options. Understanding the underlying pathophysiological mechanisms of AKI, coupled with advancements in biomarker discovery and therapeutic interventions, offers the potential to significantly reduce the burden of this condition and improve patient prognosis [1].

Description

The pathophysiology of AKI is complex and involves multiple mechanisms including ischemia-reperfusion injury, inflammation, oxidative stress, and tubular cell apoptosis. In prerenal AKI, reduced renal perfusion from hypovolemia, shock, or heart failure leads to decreased glomerular filtration rate (GFR). If prolonged, this can progress to intrinsic renal injury, particularly acute tubular necrosis (ATN). Intrinsic AKI is often the result of ischemia, sepsis, or nephrotoxic insults that cause direct injury to renal tubular cells and endothelial dysfunction. Postrenal AKI, on the other hand, results from urinary tract obstruction, which raises intratubular pressure and impairs filtration. Across all etiologies, common features include disruption of renal microcirculation, activation of inflammatory pathways, mitochondrial dysfunction, and cell death. These processes contribute to renal injury that, if unresolved, can lead to fibrosis and permanent structural damage [2].

Biomarkers have gained increasing attention as tools for the early detection and monitoring of AKI. Traditional measures such as serum creatinine and urine output, though widely used, often fail to reflect acute changes in kidney function until significant damage has occurred. Novel biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), interleukin-18 (IL-18), cystatin C, and tissue inhibitor of metalloproteinases-2 (TIMP-2) combined with insulin-like growth factor-binding protein 7 (IGFBP7) offer more sensitive and specific detection of renal injury. These biomarkers can differentiate between functional and structural renal impairment, identify the type and severity of AKI, and help guide therapeutic decisions. For example, TIMP-2/IGFBP7 has been validated as a predictor of AKI risk in critically ill patients, aiding in early preventive measures. The incorporation of biomarker-guided approaches into clinical practice holds promise for improving AKI management and outcomes [3].

Therapeutic strategies for AKI remain largely supportive, focusing on hemodynamic optimization, fluid management, avoidance of nephrotoxic agents, and Renal Replacement Therapy (RRT) when necessary. Early recognition and correction of underlying causes, such as sepsis or obstruction, are essential for reversing renal dysfunction. Advances in critical care have emphasized individualized fluid therapy, with careful balancing between avoiding hypovolemia and preventing fluid overload. Pharmacological interventions such as antioxidants, anti-inflammatory agents, and vasodilators have shown promise in experimental models, but clinical translation remains limited. The use of RRT, including intermittent hemodialysis and continuous renal replacement therapies, provides life-saving support in severe cases, though optimal timing and modality are still debated. Emerging regenerative therapies, such as stem cell-based interventions and novel pharmacological agents targeting mitochondrial dysfunction, represent exciting areas of ongoing research [4].

Despite these advancements, challenges remain in the global

management of AKI. Limited access to diagnostic resources, delayed recognition, and variability in treatment practices contribute to poor outcomes, especially in low- and middle-income countries. Moreover, AKI survivors face an increased risk of developing CKD, End-Stage Renal Disease (ESRD), and cardiovascular complications, highlighting the importance of long-term follow-up and preventive strategies. The integration of electronic health records, predictive analytics, and biomarker-based risk stratification into clinical workflows may help overcome these barriers by facilitating earlier intervention. International initiatives, such as the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines, have provided standardized definitions and management recommendations, yet ongoing research is essential to refine diagnostic tools and therapeutic approaches. A multidisciplinary approach involving nephrologists, intensivists, pharmacists, and nurses is critical to achieving optimal outcomes for patients with AKI [5].

Conclusion

Acute kidney injury remains a major clinical challenge with significant short- and long-term consequences. Understanding its multifactorial pathophysiology has paved the way for the identification of novel biomarkers that enable earlier diagnosis and more precise risk stratification. While current therapeutic strategies are primarily supportive, emerging approaches targeting inflammation, oxidative stress, and regeneration offer new hope for improved patient care. Addressing gaps in early detection, access to care, and long-term management is essential for reducing the global burden of AKI. With continued advances in biomarker discovery and therapeutic innovation, combined with international collaboration and guideline implementation, the management of AKI can be transformed to improve both survival and long-term outcomes.

Acknowledgment

None.

Conflict of Interest

None

References

1. Coca SG, Singanamala S, Parikh CR. (2012). Chronic kidney disease after acute kidney injury: A systematic review and meta-analysis. Kidney Int 81: 442-448.

2. Sohaney R, Yin H, Shahinian V, Saran R, Burrows NR, et al. (2022). In-hospital and 1-year mortality trends in a national cohort of US veterans with acute kidney injury. Clin J Am Soc Nephrol 17: 184-193.

3. Al-Jaghbeer M, Dealmeida D, Bilderback A, Ambrosino R, Kellum JA (2018). Clinical decision support for in-hospital AKI. J Am Soc Nephrol 29: 654-660.

4. Hoste EA, Bagshaw SM, Bellomo R, Cely CM, Colman R, et al. (2015). Epidemiology of acute kidney injury in critically ill patients: The multinational AKI-EPI study. Intensive Care Med 41(8), 1411-1423.

5. Hoste EA, Clermont G, Kersten A, Venkataraman R, Angus DC, et al. (2006). RIFLE criteria for acute kidney injury are associated with hospital mortality in critically ill patients: A cohort analysis. Crit Care 10: R73.