Acute kidney injury: From pathophysiology to clinical management in nephrology.

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Introduction

Acute Kidney Injury (AKI) is a common and serious condition characterized by a rapid decline in kidney function over a short period. It is associated with increased morbidity, mortality, and healthcare costs. Understanding the pathophysiology of AKI is essential for early detection, accurate diagnosis, and appropriate management to prevent further kidney damage and improve patient outcomes [1].

AKI can result from various insults to the kidneys, including ischemia, nephrotoxins, and inflammatory processes. The pathophysiology of AKI involves complex interactions between hemodynamic changes, tubular injury, inflammation, and oxidative stress. Hemodynamic alterations, such as decreased renal blood flow and Glomerular Filtration Rate (GFR), play a crucial role in the pathogenesis of AKI. Reduced perfusion pressure leads to ischemia and hypoxia in the renal tubules, contributing to tubular injury [2].

Tubular injury is a hallmark feature of AKI, characterized by epithelial cell dysfunction, apoptosis, and necrosis. Ischemic insults and nephrotoxins can directly damage tubular cells, impairing their ability to reabsorb solutes and maintain electrolyte balance. Inflammatory processes contribute to the pathogenesis of AKI through the release of pro-inflammatory cytokines and recruitment of immune cells to the kidney. Activation of innate immune pathways exacerbates tissue damage and impairs renal function [3,4].

Oxidative stress, resulting from an imbalance between Reactive Oxygen Species (ROS) production and antioxidant defenses, further exacerbates kidney injury in AKI. ROS-mediated damage to cellular components, including lipids, proteins, and DNA, contributes to renal dysfunction. The clinical presentation of AKI can vary widely depending on the underlying cause, severity, and patient characteristics. Common manifestations include oliguria, fluid overload, electrolyte abnormalities, and uremia. Early recognition and diagnosis of AKI are crucial for initiating timely interventions and preventing complications [5,6].

Diagnostic criteria for AKI typically include changes in serum creatinine levels and urine output over a defined period. The Kidney Disease: Improving Global Outcomes (KDIGO) criteria provide guidelines for staging AKI based on these parameters. Additional tests, such as urinalysis, imaging studies, and biomarkers, may be used to aid in the diagnosis and assessment of AKI severity. Optimal fluid management is essential for maintaining hemodynamic stability and preventing volume overload or dehydration in AKI patients. Careful monitoring of fluid balance and adjustment of fluid therapy based on hemodynamic parameters and renal function are crucial [7].

Electrolyte abnormalities, such as hyperkalemia, hyperphosphatemia, and metabolic acidosis, commonly occur in AKI and can have serious consequences if left untreated. Prompt identification and correction of electrolyte imbalances are essential for preventing complications. In severe cases of AKI, especially those complicated by fluid overload, severe electrolyte abnormalities, or uremia, Renal Replacement Therapy (RRT) may be necessary to provide temporary support for kidney function. RRT modalities include hemodialysis, peritoneal dialysis, and Continuous Renal Replacement Therapy (CRRT) [8].

Pharmacological interventions may be used to manage specific complications or underlying causes of AKI. For example, diuretics may be prescribed to enhance urine output and reduce fluid overload, while vasopressors may be used to maintain adequate perfusion pressure in hemodynamically unstable patients. Supportive care measures, such as nutritional support, pain management, and prevention of complications (e.g., infections, pressure ulcers), are essential components of AKI management. A multidisciplinary approach involving nephrologists, intensivists, pharmacists, and other healthcare professionals is often required to optimize patient care [9].

The prognosis of AKI varies depending on factors such as the underlying cause, severity, comorbidities, and timely intervention. While some patients may experience complete renal recovery, others may develop Chronic Kidney Disease (CKD) or progress to End-Stage Renal Disease (ESRD) requiring long-term dialysis or kidney transplantation [10].

Conclusion

Acute kidney injury is a complex and multifactorial condition associated with significant morbidity and mortality. Understanding the pathophysiology of AKI is essential for early detection, accurate diagnosis, and appropriate management to prevent further kidney damage and improve patient outcomes. A comprehensive approach involving fluid

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management, correction of electrolyte imbalances, renal replacement therapy, pharmacological interventions, and supportive care is necessary to optimize the management of AKI and enhance patient survival and recovery.

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