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RISK FACTORS AND EARLY DIAGNOSIS OF ACUTE KIDNEY INJURY IN CRITICALLY ILL PATIENTS

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ABSTRACT

Acute kidney injury (AKI) represents one of the most frequent and consequential complications encountered in critically ill patients, with incidence rates ranging from 16% to over 50% depending on the population studied and the diagnostic criteria applied. This condition is independently associated with prolonged intensive care unit stay, increased healthcare resource utilization, and significantly elevated short- and long-term mortality. The KDIGO criteria, which incorporate serum creatinine elevation and urine output reduction, have standardized the diagnosis and staging of AKI. Risk factors for AKI in critically ill patients include advanced age, pre-existing chronic kidney disease, diabetes mellitus, hypertension, sepsis, mechanical ventilation, vasopressor use, exposure to nephrotoxic medications, and elevated inflammatory markers. Early diagnosis of AKI remains a critical challenge because conventional biomarkers such as serum creatinine and urine output exhibit delayed kinetics, often manifesting only after substantial renal functional impairment has already occurred. Emerging biomarkers, including neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, and tissue inhibitor of metalloproteinases-2, offer potential for earlier detection of renal tubular injury prior to functional decline. However, their routine clinical application is not yet established. This review examines the principal risk factors and diagnostic approaches for AKI in critically ill patients, emphasizing the clinical importance of early recognition and risk stratification.

Keywords: acute kidney injury, critically ill patients, risk factors, early diagnosis, biomarkers, KDIGO criteria

INTRODUCTION

Acute kidney injury (AKI) is a complex clinical syndrome characterized by an abrupt deterioration in renal function, typically occurring over hours to days. It affects a substantial proportion of patients admitted to intensive care units and is associated with considerable morbidity and mortality. The KDIGO (Kidney Disease: Improving Global Outcomes) definition, established in 2012, has become the internationally accepted standard for AKI diagnosis and staging, incorporating both serum creatinine elevation (≥0.3 mg/dL within 48 hours or ≥1.5 times baseline within 7 days) and urine output reduction (<0.5 mL/kg/hour for 6 hours) . This unified definition has facilitated comparative epidemiological research and improved understanding of the global burden of AKI in critical illness .

The pathophysiology of AKI in critically ill patients is multifactorial and incompletely understood. Ischemic injury resulting from renal hypoperfusion, inflammatory and oxidative stress, microcirculatory dysfunction, and direct nephrotoxic damage converge to produce tubular epithelial cell injury and subsequent functional impairment. Sepsis remains the most common contributing factor, with approximately half of all AKI cases in intensive care settings attributable to septic etiology . Other important contributors include cardiogenic shock, major surgery, trauma, and administration of nephrotoxic agents. The clinical implications of AKI in critically ill patients extend well beyond the acute episode. Patients who survive an episode of severe AKI face substantially elevated risks of persistent kidney dysfunction, progression to chronic kidney disease, and long-term mortality . The two-year incidence of major adverse kidney events among survivors of severe AKI

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has been reported at 46.6 per 100 person-years, with mortality constituting the dominant component . These observations underscore the necessity of identifying high-risk patients and detecting renal injury at its earliest stages.

MAIN PART

Established Risk Factors for Acute Kidney Injury in Critically Ill Patients - The identification of modifiable and non-modifiable risk factors for AKI in critically ill patients is essential for risk stratification and preventive intervention. A prospective cohort study conducted in a tertiary care setting in Uganda reported an AKI incidence of 70 per 1000 person-days among critically ill patients, with independent predictors including previous hospitalization within the preceding three months, elevated baseline creatinine (>1.2 mg/dL), elevated baseline white blood cell count (>12 × 10^9/L), and admission to a surgical ward . These findings highlight the contribution of both patient-specific factors and contextual elements related to the clinical environment. Nephrotoxic medication exposure represents a particularly important modifiable risk factor. In the aforementioned cohort, 60% of participants were exposed to at least one potentially nephrotoxic drug during hospitalization, with penicillins and loop diuretics being the most commonly administered agents . The nephrotoxic burden of medications is a well-recognized contributor to hospital-acquired AKI and represents a target for preventive strategies through medication review and adjustment. Cardiovascular risk factors and hemodynamic instability are consistently associated with AKI development. In a study of patients admitted to a coronary care unit, independent risk factors for AKI included pre-existing chronic kidney disease, reduced left ventricular ejection fraction, mechanical ventilation, and vasopressor use . The need for vasopressor support was a particularly strong predictor of renal replacement therapy requirement . These observations reflect the critical dependence of renal perfusion on adequate cardiac output and systemic blood pressure, and the vulnerability of the kidney to hemodynamic compromise. In patients with COVID-19 admitted to intensive care, the incidence of AKI reached 54.6%, with advanced age, hypertension, cardiovascular disease, and chronic kidney disease more frequently observed among those who developed renal injury compared with those who did not . Mortality increased progressively with AKI stage, and intensive care unit length of stay was significantly prolonged in the AKI group . Laboratory parameters including lymphopenia, elevated D-dimer, ferritin, interleukin-6, C-reactive protein, and procalcitonin were significantly higher in patients with stage 3 AKI, suggesting that systemic inflammation and hypercoagulability contribute to renal injury in this population . Other risk factors identified in the literature include increased serum chloride levels, invasive mechanical ventilation with positive end-expiratory pressure, and positive fluid balance . The association between fluid balance and AKI is complex, as both hypovolemia and fluid overload may contribute to renal injury through distinct mechanisms.

The Imperative for Early Diagnosis - The traditional reliance on serum creatinine and urine output for AKI diagnosis presents a fundamental limitation: both parameters reflect functional impairment rather than structural injury. Serum creatinine does not increase until a substantial reduction in glomerular filtration rate has occurred, typically when approximately 50% of renal function has been lost. Furthermore, creatinine concentrations are influenced by muscle mass, nutritional status, and fluid balance, rendering them imperfect indicators of renal function, particularly in the critically ill. The delay inherent in creatinine-based diagnosis may delay therapeutic intervention beyond the window during which injury is potentially reversible .

Emerging biomarkers of renal tubular injury offer the prospect of earlier diagnosis. These include neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), liver fatty acid-binding protein (L-FABP), and tissue inhibitor of metalloproteinases-2 (TIMP-2). These biomarkers become detectable in urine or plasma before conventional functional parameters change,

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reflecting the existence of a subclinical phase of AKI in which tubular injury has occurred without measurable functional decline . The integration of such biomarkers into routine clinical practice could enhance diagnostic accuracy and enable earlier risk stratification, although further validation and standardization are required before widespread implementation .

Urinalysis with microscopy remains a valuable and cost-effective diagnostic tool that can provide information about the severity and likely etiology of AKI . The presence of renal tubular epithelial cells, muddy brown casts, or evidence of glomerular injury can guide clinical decision-making and prompt additional diagnostic evaluation.

Clinical Implications and Management Considerations - The recognition of AKI in its earliest stages has significant clinical implications. Once AKI is established, management focuses on treating the underlying cause, achieving and maintaining euvolemia, optimizing nutrition, controlling blood glucose, and discontinuing nephrotoxic agents where possible . Emergent nephrology consultation is recommended for patients with stage 2 or 3 AKI, those with stage 1 AKI accompanied by decompensated comorbidity, and situations in which the etiology remains unclear . The long-term consequences of AKI in critically ill patients warrant increased attention. Among patients with severe AKI, the incidence of new chronic kidney disease and progression of pre-existing chronic kidney disease at two years post-injury was 58.6 and 35.4 per 100 person-years, respectively . Non-recovery of kidney function after AKI was identified as an independent risk factor for adverse long-term outcomes . These data underscore the importance of post-discharge follow-up and monitoring of renal function in survivors of critical illness complicated by AKI. Oliguric AKI, defined as urine output less than 0.3 mL/kg/hour for at least 24 hours, was observed in approximately one-third of intensive care patients with AKI and was associated with higher one-year mortality compared with non-oliguric AKI . This finding suggests that the duration and severity of oliguria may serve as additional prognostic indicators, beyond the conventional creatinine-based staging system.

CONCLUSION

Acute kidney injury in critically ill patients represents a high-incidence, high-consequence condition that demands early recognition and proactive management. Established risk factors include pre-existing chronic kidney disease, cardiovascular compromise, sepsis, mechanical ventilation, vasopressor requirement, and exposure to nephrotoxic medications. The limitations of conventional diagnostic parameters necessitate the exploration of novel biomarkers capable of detecting renal injury before functional decline becomes manifest. The integration of risk stratification tools and emerging diagnostic technologies into routine critical care practice has the potential to improve outcomes by enabling timely intervention and preventing progression to chronic kidney disease. Further research is needed to establish the optimal strategies for early detection and to evaluate the impact of biomarker-guided management on patient outcomes.

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