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Precipitating factors for mortality of individuals with acute kidney injury in ICU; collaboration of nephrologists and intensivists

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ABSTRACT

Acute kidney injury (AKI) is a common and critical condition faced by patients in the intensive care units (ICUs), which significantly increases morbidity and mortality rates among affected individuals. Additionally, individuals in the ICU often present with a multitude of comorbidities, which significantly influence the risk of developing acute renal failure and the associated mortality rates. Common underlying conditions such as hypertension, diabetes, heart disease, and chronic kidney disease (CKD) predispose patients to acute renal failure. Hypertension and diabetes are particularly noteworthy as they distort renal hemodynamics, leading to increased susceptibility to acute renal failure during critical illness. Moreover, older patients consistently exhibit higher mortality rates associated with AKI, as advancing age correlates with deteriorating organ function and increased prevalence of comorbid conditions.

Review

Implication for health policy/practice/research/medical education:
Acute kidney injury (AKI) is a critical condition frequently encountered in intensive care unit patients. Its complexity stems from a combination of underlying illnesses, the severity of the critical condition, and various precipitating factors that contribute significantly to mortality.

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Introduction

Acute kidney injury (AKI) is a common and serious condition that significantly increases mortality rates in hospitalized patients. Defined as a sudden decrease in kidney function, characterized by elevated serum creatinine levels or reduced urine output, AKI is a critical concern in both intensive care and general medical settings (1,2). It is associated with various precipitating factors that can worsen the clinical outcome and increase the risk of intensive care unit (ICU) mortality (1). Underlying health problems play a crucial role in determining the prognosis of patients with AKI. Common comorbidities associated with AKI, such as heart disease, diabetes mellitus, and a history of chronic renal failure, significantly raise the risk of adverse outcomes (3,4). As an example, the study by Desai et al indicated that patients with diabetes had

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an adjusted odds ratio of 3.07 for in-hospital mortality when they presented with AKI (5). Since, the presence of chronic kidney disease (CKD) increases vulnerability to acute injury; however, some studies have reported that CKD by itself does not predict mortality in all instances (6). Previous authors highlighted the severity of the initial illness at the time of AKI diagnosis is a significant predictor of in-hospital mortality. How severely ill a patient is can determine the extent of kidney damage and the body's ability to recover (7,8). Critically ill patients who develop AKI have a mortality rate exceeding those with AKI alone. Meanwhile, AKI in critically ill patients nearly doubles the risk of hospital death compared to patients in the general population with AKI (9,10). Additionally, admission severity scores such as the Acute Physiology and Chronic Health Evaluation II (APACHE II) and Sequential Organ Failure Assessment (SOFA) scores have been shown to correlate positively with in-hospital mortality rates in those with AKI (11). This review study will consider the primary precipitating factors contributing to ICU mortality among individuals with AKI, including underlying health conditions, the severity of illness, use of nephrotoxic drugs, and the presence of complications such as sepsis.

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using different keywords like; acute kidney injury, chronic kidney disease, mortality rate, intensive care unit, comorbidities, hypertension and diabetes.

Molecular mechanisms of AKI in intensive care unit

The pathophysiology of AKI in ICU patients is primarily characterized by alterations in renal perfusion and the intrinsic cellular response to injury. Inflammatory processes play a critical role in the development of AKI. Following an insult, such as sepsis or shock, there is an acute inflammatory response that attracts immune cells to the kidney, which release a variety of pro-inflammatory cytokines and further exacerbate renal injury (1,12). This dysregulated inflammatory response is believed to morph from a protective mechanism during the early phases into a pathway that exacerbates renal damage over time (13). Experimental evidence shows that during AKI, the kidney is exposed to high levels of reactive oxygen species (ROS), promoting oxidative stress and cellular damage, leading to tubular cell necrosis and apoptosis (14). Then, the infiltration of inflammatory cells such as neutrophils and macrophages activate a cascade of immune responses that significantly modulate the renal environment. Localized renal inflammation is intensified by the release of damage-associated molecular patterns, which trigger pattern recognition receptors, including Toll-like receptors on renal cells (15,16). These receptors mediate inflammatory signaling pathways that promote the expression of various cytokines, further perpetuating tissue inflammation and contributing to tubular injury (16,17). Moreover, the activation of innate immune responses plays a dual role: while it may initially facilitate healing after injury, chronic inflammation leads to maladaptive repair mechanisms, manifesting as fibrosis and further decline in renal function (18,19). In the context of sepsis-induced AKI, the adverse effects of pro-inflammatory cytokines can be particularly severe, causing acute tubular necrosis and exacerbating renal dysfunction (20). Several studies showed that, oxidative stress is recognized as a pivotal factor in AKI pathogenesis, particularly in the setting of ischemia-reperfusion injury that often occurs in critically ill patients (14,21). Oxidative injury can impair mitochondrial functions, compromising cellular energy metabolism and promoting cell death through pathways such as necroptosis and ferroptosis (22). The excessive generation of ROS inflicts damage on cellular components, including lipids, proteins, and DNA, triggering apoptotic signaling pathways that ultimately result in tubular cell death (23). The renal tubular epithelial cells are particularly vulnerable due to their high metabolic activity and reliance on mitochondrial function for ATP production. Meanwhile, the recovery process post-AKI is critically dependent on the regenerative capacity of surviving tubular cells (24). Following injury, renal tubular epithelial cells exhibit various adaptive responses, such as dedifferentiation, proliferation, and eventual re-differentiation to restore renal function (24). However, extensive or repeated injuries can lead to maladaptive repair characterized by cellular senescence and persistent inflammation, ultimately leading to CKD (24,25). Accordingly, cell cycle arrest and cellular senescence represent critical pathological features of renal tubular epithelial cells responses following AKI (25). While initially protective, prolonged cell cycle arrest can contribute to the accumulation of senescent cells that secrete pro-inflammatory and profibrotic factors (26). Senescent tubular cells can further promote inflammation and fibrosis, underscoring the importance of modulating repair mechanisms to avert the long-term consequences of AKI (26).

Determinants of mortality risk of AKI in ICU

The severity of AKI itself is a crucial determinant of mortality risk. Studies indicate a marked correlation between the stage of AKI and the mortality rates, with more severe forms of AKI (stage 2 and stage 3) associated with exponentially higher mortality risk compared to stage 1 (10). For instance, the hospital mortality rates for

Stage 1 AKI are approximately 6.3%, while for Stage 3, it escalates to 23.7% (27). Mortality odds ratios increase significantly with each increment in AKI stage; therefore, close monitoring and early intervention in patients presenting with severe AKI are paramount to mitigate these heightened risks (27). In addition, complications associated with AKI further exacerbate the risk of mortality. Metabolic disturbances like hyperkalemia, metabolic acidosis, and fluid overload are frequent in AKI cases, impacting the overall clinical status of the patient and often necessitating renal replacement therapy (RRT) (28). Notably, hyperkalemia has been identified as a major cause of sudden cardiac events, leading to increased mortality rates among AKI patients (29). Furthermore, patients requiring RRT demonstrate higher mortality rates, especially when initiated late amid stable or progressive AKI. Studies reveal that early initiation of RRT can significantly reduce AKI-related mortality, emphasizing the importance of timely intervention (30,31). Delayed treatment increases the incidence of complications which, in turn, results in worse outcomes for patients (30,31). Sepsis is another predominant factor precipitating AKI in critically ill patients and has been shown to directly contribute to increased mortality (32). The presence of sepsis triggers a cascade of inflammatory responses that can lead to multi-organ failure, worsening renal function and heightening the likelihood of mortality in these patients (32). The interaction between sepsis and AKI creates a vicious cycle that significantly complicates clinical management (32,33). The underlying mechanisms often culminate in systemic hypotension, impaired renal perfusion, and the activation of multiple organ dysfunction syndrome, all of which are associated with poorer prognosis for patients (34). Accordingly, nephrotoxic medications are pivotal factors in the onset and progression of AKI (35). Commonly prescribed medications, including non-steroidal anti-inflammatory drugs, diuretics, and certain antibiotics, can independently precipitate kidney injury, exacerbating the condition (35). For instance, the administration of diuretics was found to significantly increase the odds of developing AKI, particularly in patients who were already vulnerable due to existing health issues (36). Moreover, nephrotoxic drug exposure has been linked to higher mortality rates in patients with AKI, especially in those requiring RRT (37). One study cited a mortality rate of 62% in patients with AKI who received RRT, illustrating the critical interplay between medication management and recovery outcomes (38). Further, sepsis is a critical and common complication in patients with AKI that significantly contributes to in-hospital mortality. The relationship between sepsis and AKI is so pronounced that it is often described as the sepsis-associated AKI (SA-AKI) (33). The study by Jia et al showed that patients with AKI and concurrent sepsis have been observed to have hospital mortality rates ranging from 30% to 60%, depending on the severity of their illness (39). Moreover, septic shock, as a more severe form of sepsis, has been identified as a major contributor to mortality in AKI patients, since it can exacerbate renal injury through systemic inflammation and hemodynamic instability (40). This highlights the critical need for rapid identification and treatment of sepsis in hospitalized patients presenting with or at risk for AKI (40).

Improving outcomes for AKI patients in the ICU

The timely identification and intervention for patients exhibiting signs of AKI are vital components in the management of this life-threatening condition (41,42). Early intervention has been shown to be instrumental in improving outcomes for AKI patients in the ICU, often leading to reduced kidney injury severity, lower mortality rates, and enhanced renal recovery (41,42). Thereby, early recognition of AKI and prompt initiation of treatment can mitigate the progression of the injury to more advanced stages, which are often associated with worse outcomes (41,42). Clinical studies indicated that patients with stage 1 AKI who receive prompt intervention are less likely to progress to stage 2 or stage 3 AKI, significantly reducing the risk of complications associated with more severe forms of the condition (43). This relationship underlines the importance of timely responses to the initial signs of renal dysfunction, ensuring that patients maintain optimal renal perfusion and function (43). Likewise, the impact of early intervention on mortality rates among AKI patients in the ICU is profound (43). A systematic review of literature by Li et al, highlights that early initiation of RRT has been correlated with decreased mortality in critically ill patients with AKI; since, early RRT initiation has been linked to lower all-cause mortality rates compared to delayed initiation (44). This condition can be attributed to the prevention of severe metabolic complications, such as hyperkalemia and metabolic acidosis, which can arise from untreated AKI and lead to life-threatening conditions (42,44). Therefore, early intervention also plays a crucial role in enhancing the likelihood of renal recovery post-AKI. Patients who receive appropriate and timely treatment are more likely to recover renal function after an AKI episode (7). A previous study by Meersch et al has shown that cases with early RRT have a lower incidence of persistent renal dysfunction after one year compared to those whose RRT is initiated later. This is particularly important, as many AKI patients face a subsequent risk of CKD if renal recovery is not achieved promptly (31). Moreover, effective early intervention strategies, including appropriate fluid management, withdrawal of nephrotoxins, and overall

support for hemodynamic stability, are key in optimizing renal recovery (41,42,45).

Focus on early intervention modalities

To achieve the benefits of early intervention in AKI patients, physicians must integrate systematic protocols and guidelines to ensure timely care (46,47). Regular monitoring of kidney function, particularly in high-risk populations, is crucial. The use of biomarkers for early detection of AKI, alongside early intervention, can contribute significantly to improved outcomes (48,49). The protocols of fluid resuscitation and optimizing drug dosing based on renal function can prevent further deterioration in kidney health (50,51). Moreover, effective teamwork between nephrologists and intensivists can facilitate early referrals and optimize patient management strategies within the ICU (52).

Conclusion

The precipitating factors contributing to ICU mortality in individuals with AKI are multifaceted, involving underlying health conditions, severity of illness, use of nephrotoxic drugs, and complications such as sepsis. Underlying medical conditions such as hypertension and diabetes, coupled with the severity of AKI, the presence of complications like metabolic disturbances, and the critical impact of sepsis, all interplay to escalate the mortality risks in these patients. Therefore, a comprehensive understanding of these factors is essential for clinicians to formulate proactive strategies to monitor, treat, and improve outcomes in patients with AKI in the ICU setting. Optimizing management protocols, including early identification and appropriate intervention, remains pivotal in mitigating the associated mortality of AKI in critically ill patients. By decreasing the severity and duration of kidney injury, reducing mortality rates, and facilitating renal recovery, timely intervention can significantly impact patient prognosis and quality of life. Implementing robust monitoring systems and treatment protocols within the ICU setting is essential for managing AKI effectively. The integration of early intervention strategies not only enhances survival rates but also promotes better kidney health among critically ill patients, highlighting the necessity for ongoing research and application of best practices in AKI care.

Authors' contribution

Conceptualization: Mojgan Jahantigh and Mahshad Ghezelbash
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Visualization: Mahsa Rezaee.
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 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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