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Fibrinogen to Albumin Ratio in Acute Kidney Injury: A Readily Available Biomarker for Critical Illness Outcomes

Ashraf Khalifa Elnaggar[1], Mahmoud Mohammed Ragab Ali[2], Azza Moustafa Ahmed[3], Essam Adel Abdelrahman[4]

1. Professor of Internal Medicine, Faculty of Medicine - Zagazig University,
2. MBBCH, Faculty of Medicine - Zagazig University,
3. Assistant Professor of Clinical Pathology, Faculty of Medicine - Zagazig University,
4. Lecturer of Internal Medicine, Faculty of Medicine - Zagazig University,

Corresponding author: Mahmoud Mohammed Ragab Ali

ABSTRACT

Background: Acute kidney injury (AKI) is a frequent and serious complication in critically ill patients, associated with high morbidity, mortality, and healthcare burden. Despite advances in critical care, early recognition and precise risk stratification of AKI remain challenging, contributing to delays in intervention and poor outcomes. In recent years, a growing focus has emerged on the role of biomarkers in enhancing the timely diagnosis and prognosis of AKI. Among these, the fibrinogen to albumin ratio (FAR) has gained attention as a potential prognostic marker due to its reflection of systemic inflammation and nutritional status—both of which are crucial determinants of outcomes in critically ill populations. This review aims to explore the prognostic utility of FAR in patients with AKI, especially within the intensive care setting. We discuss the pathophysiology of AKI, the importance of biomarker-guided management, and the evolving diagnostic criteria. A special emphasis is placed on the classification and clinical roles of emerging biomarkers for diagnosis, risk stratification, and therapeutic monitoring. The review also addresses the potential for FAR to serve as a readily available, cost-effective indicator for mortality and renal outcomes in AKI, compared with conventional and novel biomarkers. Further, we examine how biomarker-guided strategies, including the use of FAR, may contribute to antimicrobial stewardship, personalized care, and reduced nephrotoxic exposure. Finally, we analyze the limitations and current research gaps in biomarker utilization, and propose directions for future studies to better integrate FAR and similar indices into clinical decision-making algorithms. Overall, this review synthesizes current knowledge on FAR and emphasizes its relevance in prognostic evaluation among critically ill AKI patients, offering insights into more precise and individualized patient management.

Keywords: *Fibrinogen to Albumin Ratio, Acute Kidney Injury, Critical Illness*

INTRODUCTION

Acute kidney injury (AKI) is a common and serious condition encountered in critical care settings, characterized by a rapid decline in kidney function leading to the accumulation of nitrogenous waste products, fluid imbalance, and electrolyte disturbances. AKI affects up to 50% of critically ill patients in intensive care units (ICUs), with mortality rates ranging from 20% to over 50% depending on severity and associated comorbidities [1]. The condition can result from a variety of insults including ischemia, nephrotoxins, sepsis, and surgery. Importantly, AKI is not only an acute problem but also a precursor to chronic kidney disease (CKD), especially when recovery is incomplete [2].

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The management of AKI is largely supportive, emphasizing hemodynamic optimization, avoidance of nephrotoxins, and timely initiation of renal replacement therapy when indicated. However, traditional diagnostic tools such as serum creatinine and urine output are often delayed and nonspecific, which underscores the need for more sensitive and timely biomarkers [3]. Emerging evidence supports the integration of novel biomarkers and clinical indices into early AKI diagnosis and risk stratification, with growing attention to inflammatory and nutritional markers such as the fibrinogen to albumin ratio (FAR) [4].

Recent research suggests that FAR, a marker derived from routine laboratory values, may reflect the severity of systemic inflammation and hypoalbuminemia—both of which are strongly associated with adverse outcomes in AKI patients. Elevated FAR has been linked to increased ICU mortality and longer hospital stays, supporting its potential as a simple, cost-effective prognostic indicator in critically ill populations [5].

A. Acute Kidney Injury: Biomarker-Guided Diagnosis and Management

Traditional diagnostic criteria for AKI, primarily based on serum creatinine levels and urine output, often fail to detect kidney damage in its early stages. These parameters are influenced by various factors such as volume status, muscle mass, and delayed kinetics, which can delay diagnosis and timely intervention [6]. This limitation has driven the exploration and implementation of biomarkers that can more sensitively and specifically reflect renal injury and functional impairment. Biomarker-guided approaches offer the advantage of identifying patients at risk or in early stages of AKI before overt clinical manifestations occur, allowing for prompt therapeutic measures [7].

Recent advances have led to the validation of several novel biomarkers including neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), tissue inhibitor of metalloproteinases-2 (TIMP-2), and insulin-like growth factor-binding protein 7 (IGFBP7). These biomarkers reflect different pathophysiological processes such as tubular injury, cell cycle arrest, and inflammation. Their integration into clinical practice has enhanced risk stratification and improved the prediction of outcomes like need for renal replacement therapy or in-hospital mortality [8].

Biomarker-guided management also supports drug stewardship, particularly in minimizing exposure to nephrotoxins. For example, biomarkers such as TIMP-2 × IGFBP7 (commercially available as NephroCheck®) can help identify patients who are more vulnerable to nephrotoxic injury, thus guiding decisions about contrast use or antibiotic choice in critically ill patients [9]. Moreover, dynamic changes in biomarker levels can serve as monitoring tools for the effectiveness of interventions and provide insights into the trajectory of kidney recovery or worsening [10].

Overall, the biomarker-guided paradigm represents a shift from reactive to proactive management of AKI, aligning with precision medicine principles. However, broader accessibility, cost-effectiveness,

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and standardization of biomarker assays remain barriers to widespread adoption in routine ICU care [11].

B. Diagnosis of AKI

The diagnosis of acute kidney injury (AKI) has traditionally relied on increases in serum creatinine and reductions in urine output, as outlined by consensus criteria such as RIFLE (Risk, Injury, Failure, Loss, and End-stage kidney disease), AKIN (Acute Kidney Injury Network), and KDIGO (Kidney Disease: Improving Global Outcomes) [12]. These criteria have standardized AKI staging, allowing for greater consistency in clinical practice and research. However, the use of serum creatinine is problematic due to its insensitivity to early kidney injury and its variability with factors like muscle mass, hydration status, and medication use [13].

Urine output, although part of the diagnostic framework, can be influenced by fluid therapy and diuretic use, and often lacks specificity for true renal injury. Furthermore, both markers reflect functional changes rather than structural injury and may lag behind the actual onset of renal insult [14]. Thus, the reliance on these traditional indicators may delay diagnosis, missing the window for early intervention and contributing to worsened outcomes in critically ill patients [15].

To address these challenges, newer approaches have advocated for the inclusion of structural damage markers in diagnostic criteria. Biomarkers like NGAL, KIM-1, and interleukin-18 (IL-18) have shown promise in identifying subclinical AKI—renal injury without an overt rise in serum creatinine. These markers provide additional granularity to the diagnostic process, enabling the identification of patients at risk for progression or complications even before traditional criteria are met [16].

Early identification through advanced biomarkers is particularly valuable in intensive care and perioperative settings, where timely intervention can mitigate complications such as fluid overload, need for renal replacement therapy, and multiorgan dysfunction. Nevertheless, incorporation of these biomarkers into routine practice remains limited due to issues of cost, assay availability, and lack of standardized cutoff values [17].

C. Definition and Diagnostic Criteria

Acute kidney injury (AKI) is defined as an abrupt decline in kidney function occurring over hours to days, resulting in the accumulation of metabolic waste, fluid imbalance, and dysregulation of electrolytes. The KDIGO (Kidney Disease: Improving Global Outcomes) clinical practice guideline remains the most widely adopted framework for AKI diagnosis, defining it as an increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours, or an increase to ≥ 1.5 times baseline within the prior 7 days, or a urine volume of <0.5 mL/kg/h for 6 hours [18].

This definition integrates criteria from previous classifications such as RIFLE and AKIN, providing a comprehensive and standardized approach to AKI staging. KDIGO stages AKI into three grades of

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increasing severity based on serum creatinine rise and urine output reduction. This stratification aids clinicians in recognizing AKI early, estimating prognosis, and guiding interventions [19]. Nevertheless, the limitations of using creatinine and urine output persist, particularly in ICU settings where baseline renal function may be unknown or confounded by non-renal factors [20].

Emerging perspectives suggest that AKI should not be viewed as a single disease entity but rather a syndrome encompassing multiple etiologies, each with distinct pathophysiological mechanisms and therapeutic implications. In this context, the definition of AKI may evolve to incorporate molecular markers of damage, inflammation, and stress, complementing traditional functional criteria [21]. Such a dual approach—combining markers of injury and function—could provide a more nuanced and clinically meaningful framework for diagnosis and risk stratification in critically ill patients.

Further refinement of AKI definitions will likely depend on the clinical validation of novel biomarkers and their integration into practice guidelines. These advancements could ultimately improve early recognition and enable a shift toward precision medicine approaches in nephrology and critical care [22].

D. Biomarkers for Diagnosis

Biomarkers have emerged as essential tools for the early detection and risk assessment of acute kidney injury (AKI), offering insights that go beyond traditional markers like serum creatinine and urine output. Unlike these conventional parameters, which reflect functional decline often delayed by several hours or days post-insult, biomarkers can indicate kidney injury at a cellular or molecular level, enabling earlier intervention [23].

Several promising biomarkers have been identified and validated in both experimental and clinical settings. Neutrophil gelatinase-associated lipocalin (NGAL) is one of the most studied, reflecting tubular injury as early as 2–4 hours after insult. It has demonstrated predictive value in a variety of settings, including cardiac surgery, sepsis, and contrast-induced nephropathy [24]. Kidney injury molecule-1 (KIM-1), expressed in proximal tubular epithelial cells following injury, is another sensitive marker of ischemic and nephrotoxic damage and has shown utility in differentiating pre-renal azotemia from intrinsic AKI [25].

Other biomarkers, such as liver-type fatty acid-binding protein (L-FABP), interleukin-18 (IL-18), and cystatin C, offer additional diagnostic and prognostic information. These markers target different aspects of renal injury, including oxidative stress, inflammation, and glomerular filtration function. Some have demonstrated synergistic value when used in combination, enhancing diagnostic performance compared to any single marker [26].

Perhaps the most clinically advanced biomarkers are TIMP-2 and IGFBP7, which indicate early cell-cycle arrest in tubular cells. These are commercially available as the NephroCheck® test and have

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shown high predictive value for moderate to severe AKI within 12 hours of ICU admission [27]. Despite their promise, the routine use of biomarkers is constrained by cost, availability, and lack of standardization across laboratories and patient populations [28].

E. Risk Stratification for AKI Assessment and Prevention

Risk stratification is a critical step in preventing the onset and progression of acute kidney injury (AKI), especially in high-risk populations such as critically ill patients, those undergoing major surgery, or individuals exposed to nephrotoxic agents. Early identification of patients at increased risk allows for timely application of nephroprotective strategies, fluid optimization, and medication adjustments to mitigate renal insult [29].

Clinical risk models, such as the Cleveland Clinic Score or Mehran Score for contrast-induced nephropathy, incorporate variables like age, comorbidities, baseline renal function, and hemodynamic status. However, these tools are limited by interpatient variability and often fail to capture dynamic physiological changes that occur in real-time in critically ill patients [30]. Consequently, there is a growing interest in incorporating biomarkers into risk stratification frameworks to enhance their predictive power and clinical utility.

The use of biomarkers such as NGAL, TIMP-2, and IGFBP7 has shown value in identifying patients at risk for AKI before the onset of overt clinical symptoms. In perioperative and ICU settings, elevated levels of these markers have been associated with higher incidence of AKI, longer hospital stays, and greater need for renal replacement therapy [31]. These biomarkers may outperform traditional risk scores, particularly in capturing subclinical injury and predicting adverse outcomes.

Integrating biomarkers with clinical algorithms creates a multidimensional risk assessment approach. For example, combining biomarker data with hemodynamic monitoring, fluid balance, and early warning scores can guide preemptive strategies such as goal-directed therapy, nephrotoxin avoidance, or early nephrology consultation [32]. Such proactive interventions may reduce the incidence and severity of AKI, improve renal recovery, and lower mortality.

Despite their promise, the widespread application of biomarker-driven risk stratification is challenged by high costs, logistical barriers, and limited availability in resource-constrained settings. More robust multicenter studies are needed to validate these approaches and determine cost-effectiveness in routine clinical practice [33].

F. Biomarkers for AKI Risk Assessment, Prediction, and Prevention

Biomarkers have transformed the landscape of AKI prediction by enabling the detection of kidney stress or injury before clinical symptoms or changes in serum creatinine become apparent. Their application in risk assessment and prevention is particularly valuable in critically ill patients, where early identification of subclinical AKI can alter clinical decisions and improve outcomes [34].

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Among the most promising biomarkers for early risk prediction are tissue inhibitor of metalloproteinases-2 (TIMP-2) and insulin-like growth factor binding protein 7 (IGFBP7), which reflect tubular cell-cycle arrest—an early event in response to cellular stress. These biomarkers have been shown to predict AKI within 12 hours in ICU patients, with higher levels associated with a greater risk of progression to severe AKI and need for renal replacement therapy. Their combined product ([TIMP-2] × [IGFBP7]) is available as a point-of-care test (NephroCheck®), approved by the FDA for AKI risk assessment in hospitalized patients [35].

Neutrophil gelatinase-associated lipocalin (NGAL), both in plasma and urine, also serves as a reliable marker for predicting AKI in various clinical scenarios, including cardiac surgery, sepsis, and contrast exposure. Studies have shown that elevated NGAL levels correlate with adverse renal outcomes and can provide prognostic information superior to traditional creatinine-based assessments [36]. Similarly, biomarkers such as KIM-1, IL-18, and cystatin C have demonstrated utility in forecasting AKI onset and severity, particularly when combined into multimarker panels [37].

Incorporating biomarker-driven prediction tools into clinical pathways enables timely application of renal-protective strategies, such as optimization of hemodynamics, dose adjustment of nephrotoxic medications, and enhanced surveillance. Preventive measures guided by biomarker profiles may reduce the incidence and severity of AKI, lessen the burden of renal replacement therapy, and ultimately improve long-term renal and patient outcomes [38].

Despite this promise, routine use of predictive biomarkers is not yet universal due to concerns over cost-effectiveness, test variability, and uncertain clinical thresholds. Further large-scale, multicenter trials are needed to define optimal biomarker thresholds, improve diagnostic algorithms, and assess the impact on patient-centered outcomes [39].

G. Drug Stewardship and Use of Biomarkers

Acute kidney injury (AKI) in critically ill patients is frequently exacerbated by exposure to nephrotoxic medications, including aminoglycosides, vancomycin, non-steroidal anti-inflammatory drugs, and iodinated contrast agents. Drug-induced nephrotoxicity remains a significant contributor to hospital-acquired AKI, underscoring the importance of antimicrobial and drug stewardship programs aimed at minimizing unnecessary exposure and optimizing dosing strategies [40].

Biomarkers have emerged as powerful adjuncts in drug stewardship, enabling early identification of patients who are at increased risk of nephrotoxic injury. For instance, elevated levels of urinary TIMP-2 × IGFBP7 or NGAL can signal early kidney stress before traditional indicators such as serum creatinine rise, allowing clinicians to adjust or discontinue potentially nephrotoxic drugs proactively [41]. This is particularly relevant in the ICU where balancing antimicrobial efficacy with renal safety is a constant challenge.

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Incorporating biomarkers into stewardship algorithms allows for a more individualized approach to pharmacotherapy. For example, in septic patients receiving high-dose vancomycin, serial monitoring of renal biomarkers can guide dose adjustment or prompt early consideration of alternative agents before irreversible damage occurs. Similarly, in the setting of contrast-enhanced imaging, pre-procedural assessment of biomarkers may help identify high-risk individuals who would benefit from enhanced hydration protocols or contrast-sparing techniques [42].

Biomarker-guided stewardship also holds promise in the context of chemotherapy-related AKI, transplant immunosuppression, and perioperative medication management. Furthermore, emerging studies suggest that combining pharmacokinetic data with dynamic changes in biomarkers may enhance the precision of dosing regimens in patients with fluctuating renal function [43].

Despite these advantages, routine integration of biomarkers into stewardship remains limited by cost considerations, lack of real-time assays in many settings, and the need for broader validation. As evidence accumulates, particularly from interventional trials, biomarker-informed stewardship is likely to become a cornerstone of kidney-protective strategies in critically ill populations [44].

H. Limitations of Novel Biomarkers and Future Research Directions

Despite the considerable promise of biomarkers in the diagnosis, prediction, and management of acute kidney injury (AKI), several limitations currently hinder their widespread implementation in clinical practice. One major concern is the lack of standardization across assay platforms, which can lead to variability in biomarker levels and difficulty in interpreting results across different settings or patient populations [45]. Differences in sample handling, storage, and timing of measurement further complicate comparison and reproducibility.

Another limitation is cost. Many of the most promising biomarkers, including [TIMP-2] × [IGFBP7] and NGAL, require specialized assays that are not routinely available in all healthcare facilities, particularly in low-resource settings. This restricts their use to specialized centers and limits their applicability in broader patient populations [46]. Furthermore, the predictive thresholds for many biomarkers are not yet universally established, making clinical decision-making difficult without clear, evidence-based cutoffs [47].

The interpretation of biomarker levels is also influenced by comorbidities, systemic inflammation, and critical illness itself. For instance, elevated NGAL can occur in infections, malignancies, or inflammatory states without underlying kidney injury, leading to false positives and diagnostic ambiguity [48]. Moreover, the clinical utility of these biomarkers is often evaluated in observational studies, and interventional trials demonstrating improved patient-centered outcomes through biomarker-guided therapy remain limited.

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Future research should focus on large-scale, multicenter randomized controlled trials assessing how biomarker-guided strategies affect clinical decisions, reduce AKI incidence, or improve mortality and renal recovery. In addition, the integration of biomarker data with artificial intelligence and electronic health records may support real-time risk prediction models for AKI, tailored to individual patient physiology and clinical context [49]. Ongoing exploration of novel biomarkers with improved specificity, lower cost, and broader applicability will also be essential to advance the field.

Ultimately, translating biomarker research into routine practice will require addressing logistical, financial, and regulatory barriers while continuously refining their clinical relevance through robust validation studies [50].

I. Prognostic Value of Fibrinogen to Albumin Ratios Among Critically Ill Patients with Acute Kidney Injury

The fibrinogen to albumin ratio (FAR) has recently garnered interest as a novel biomarker that combines inflammatory and nutritional status to predict outcomes in various critical illnesses, including acute kidney injury (AKI). Fibrinogen, a positive acute-phase reactant, rises in response to systemic inflammation and plays a role in coagulation and endothelial dysfunction. In contrast, albumin levels typically decline during critical illness due to hemodilution, malnutrition, and increased capillary permeability. The FAR therefore offers a composite index that reflects the severity of the underlying pathophysiologic milieu in critically ill patients [51].

Several retrospective and prospective studies have demonstrated the prognostic utility of FAR in critically ill populations. Elevated FAR has been associated with increased in-hospital mortality, prolonged ICU stay, and higher incidence of renal replacement therapy among patients with AKI. In a study by Wu et al., critically ill AKI patients with higher FAR levels on ICU admission had significantly worse 30-day mortality and fewer ventilator-free days compared to those with lower FAR values, independent of APACHE II and SOFA scores [52].

The appeal of FAR lies in its accessibility and cost-effectiveness, as both fibrinogen and albumin are routinely measured in ICU settings. FAR may be especially useful in resource-limited environments where advanced biomarker assays are unavailable. Moreover, FAR can complement existing clinical scores to improve risk stratification and guide therapeutic intensity. It may also serve as a target for nutritional and anti-inflammatory interventions aimed at improving patient outcomes [53].

Despite these advantages, the current body of evidence supporting FAR is limited to observational data. Further prospective studies are needed to validate optimal cutoff values, clarify temporal trends, and evaluate its role in clinical decision-making. Integration of FAR into predictive models and stewardship protocols could enhance personalized care for AKI patients in critical care settings [54].

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Conclusion

The fibrinogen to albumin ratio (FAR) is a promising, cost-effective biomarker for prognosticating outcomes in critically ill patients with acute kidney injury (AKI). By reflecting both systemic inflammation and nutritional status, FAR has been associated with increased mortality, greater need for renal replacement therapy, and longer ICU stays. Its simplicity and availability make it a practical tool for risk stratification, especially when combined with other biomarkers and clinical scores. However, further prospective studies are needed to validate its predictive power and establish standardized thresholds. With continued research, FAR could become an integral part of AKI management in critical care settings.

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