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**Review Article**

# A review on the role of urinary biomarkers in predicting renal recovery post-sepsis

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## ABSTRACT

Acute kidney injury (AKI), which is commonly caused by sepsis and contributes significantly to worldwide mortality, may affect patient outcomes and increase the risk of chronic disease. This review examines how urinary biomarkers can forecast renal recovery following sepsis. Conventional AKI diagnostics, like serum creatinine, suffer from delayed detection and poor specificity. Newer urinary biomarkers, such as neutrophil gelatinase-associated lipocalin (NGAL) and cell-free DNA (cfDNA), show potential for earlier detection and improved prognosis. NGAL, noted for its sensitivity to nephrotoxic and ischemic insults, shows potential in predicting AKI onset and recovery. Similarly, cfDNA levels, reflecting systemic cell death, correlate with sepsis severity and renal outcomes. Despite their promise, variability in biomarker levels due to comorbidities and the need for standardized diagnostic thresholds remain challenges. Combining multiple biomarkers may enhance diagnostic accuracy, offering a more comprehensive assessment of kidney function and sepsis-induced AKI (S-AKI). Further research is needed to validate these biomarkers and integrate them into clinical practice for improving patient outcomes post-sepsis.

**Keywords:** Sepsis, AKI, Urinary biomarkers, NGAL, cfDNA, Renal recovery

## INTRODUCTION

Sepsis is among the leading global causes of mortality, arising from syndromic responses to various infectious diseases. This life-threatening clinical condition occurs when the body's tissues and organs are compromised due to an infection, leading to immune suppression.[1] Sepsis is associated with multiple morbidities, including dysfunctions of the heart, kidneys, liver, and central nervous system.[2] Global sepsis cases are shown to be 48.9 million, with 11 million deaths as a result. This accounts for 19.7% of all deaths worldwide.[3,4] The overall mortality rate for sepsis can range from one-sixth to one-third of affected patients. Additionally, sepsis is one of

the most expensive hospital complications, representing 6.2% of total hospitalization costs, which amounts to $23 billion each year.[4,5]

During sepsis, the infection triggers systemic inflammatory response syndrome (SIRS), which involves the release of pro-inflammatory cytokines, adhesion molecules from damaged endothelium and immune cells, and procoagulants. Two or more symptoms, such as a body temperature that is either over 38°C or below 36°C, tachycardia, tachypnea, or a white blood cell count that is either above $12\times10^9$/L or below $4\times10^9$/L, are indicative of SIRS.[6] In severe cases, the septic process leads to the dysfunction of at least one organ. In more critical scenarios, patients experience hypotension due to severe sepsis, known as septic shock. Chronologically, septic shock represents the most severe condition, followed by severe sepsis, which is more serious than sepsis.[7]

Septic patients often experience multiple organ failures, including those affecting the kidneys, liver, lungs, circulatory system, gastrointestinal tract, hematologic system, and central nervous system.[8] Among these, sepsis-associated renal dysfunction is the primary cause of in-hospital AKI. It often leads to multiple organ failure and, consequently, increases the risk of death.[9] The epidemiology of S-AKI is not well understood, but it is estimated that sepsis accounts for half of all AKI cases, with 60% of septic patients developing AKI.[6]

It is thought that up to 11 million people may develop S-AKI annually, given that there are roughly 19 million cases of sepsis worldwide each year.[10]

Additionally, up to 25% of patients with S-AKI will require renal replacement therapy (RRT).[10] The intricate and distinct pathophysiology of sepsis gives rise to the syndromic nature of sepsis-associated AKI, setting it apart from other types of AKI and making it challenging to pinpoint the precise onset of kidney damage, which ultimately delays timely prognosis.[6] A key feature of S-AKI is its early onset during the progression of sepsis. In fact, half of the patients with septic shock develop AKI before arriving at the emergency department. As such, AKI can act as a crucial early indicator, signaling the presence of sepsis in its initial stages.[10]

Biomarkers are essential for diagnosing, treating, and predicting outcomes in sepsis and AKI. However, no individual biomarker has demonstrated sufficient specificity or discriminative ability to be used conclusively for these conditions.[11]

## LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on Augst 25, 2024, in the Medline and Cochrane databases, utilizing the medical subject headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the role of urinary biomarkers in predicting renal recovery post-sepsis. There were no restrictions on date, language, participant age, or type of publication.

## DISCUSSION

The pathophysiology of S-AKI is complex and multifaceted, involving systemic and renal inflammation, microcirculatory dysfunction, and metabolic reprogramming.[11]

Sepsis is diagnosed when there is an increase of two or more points in the sequential organ failure assessment (SOFA) score in the context of an infection. Created by the European society of intensive care medicine, the SOFA score assesses dysfunction or failure across various organ systems, including neurological, respiratory, cardiovascular, gastrointestinal, renal, hematological, and endocrinological systems. Each system is rated on a scale from 0 to 4, reflecting the severity of dysfunction.[12]

### *Role of biomarkers in the detection of sepsis and AKI*

To overcome the limitations of current diagnostic standards, the use of biomarkers offers a promising approach for enhancing early detection and management.[11] For AKI, traditional diagnosis relies on serum creatinine (Scr) levels and urine output measurements. However, Scr has several limitations, it increases only after 48 hours of kidney injury, can be underestimated due to fluid overload, and is affected by muscle mass and other variables.[11] Urine volume measurements can be unreliable, especially in cases of oliguric renal injury, where a considerable proportion of patients (25% to 80%) may not show oliguria.[13] Additionally, using urinary catheters for precise monitoring may lead to infections.

In the context of sepsis, no standard biological marker is universally accepted. Currently, procalcitonin, lactate, and C-reactive protein (CRP) are utilized in clinical practice. However, procalcitonin levels can also rise in conditions such as trauma, burns, cardiogenic shock, major surgeries, and renal injury, making its specificity for sepsis low.[14,15] Lactate, which reflects perfusion abnormalities and impaired oxidative metabolism, is not a reliable early indicator of sepsis and lacks specificity.[16] CRP is useful but lacks specificity to bacterial infections, as it can be elevated in various other inflammatory conditions.

Emerging research suggests that combining biomarkers could enhance diagnostic accuracy by addressing the heterogeneity of both AKI and sepsis. For AKI, a panel of biomarkers may offer a comprehensive assessment by identifying the type and location of injury, whether pre-

renal, renal, or post-renal, and distinguishing between ischemic, nephrotoxic, and obstructive causes. Similarly, in sepsis, a combination of biomarkers could help differentiate between various pathophysiological pathways, thereby improving risk stratification, early detection, and management. This approach could provide more detailed insights into the global dimension of kidney function and the specific pathways involved in sepsis, potentially guiding more effective antibiotic use and treatment strategies.[11]

***NGAL***

NGAL has emerged as a prominent biomarker for diagnosing both AKI and sepsis. NGAL is normally expressed at low levels in human tissues, including the kidneys, stomach, lungs, trachea, and colon.[17] It is associated with anti-inflammatory effects and is released in response to epithelial injury and inflammation.[18]

In the kidneys, NGAL is expressed in various segments, including the thick ascending limb of the loop of Henle, the collecting ducts, and the proximal tubular epithelium. NGAL's physiological role remains under investigation, but it is thought to participate in renal morphogenesis, cell proliferation, and tubular re-epithelialization. Its increase in urine during AKI may indicate nephrotoxic and ischemic insults, functioning similarly to a "kidney troponin".[11]

Serial assessments of NGAL have proven valuable for early detection of AKI in emergency settings.[19] The diagnostic and prognostic accuracy of NGAL for AKI, as well as its function in predicting course and results, are validated by meta-analyses conducted by Haase et al. and Zhou et al..[20] NGAL has also shown promise in predicting adverse medium-term outcomes, such as increased risk of mortality or progression to chronic kidney disease.[21]

NGAL is also relevant for sepsis diagnosis. NGAL reflects neutrophil activation, which is prevalent in sepsis, and can aid in early diagnosis and disease stratification. A plasma NGAL cut-off value of 570 ng/mL has been identified as a predictor of bacterial sepsis, with AUC of 0.69.[11] In neonates, NGAL cut-off values of 455 µg/L and 1104 µg/L have been associated with sepsis and non-survival, respectively.[22]

Additionally, a study found that a NGAL cut-off of 250 ng/mL had a sensitivity of 0.838 and a specificity of 0.827 for predicting 28-day mortality, showing better sensitivity than lactate.[23]

Despite its potential, NGAL's clinical application faces challenges. Variability in NGAL levels, influenced by factors such as age, gender, and comorbid conditions like chronic obstructive pulmonary disease, cardiac dysfunction, and diabetes, can impact its diagnostic performance. Additionally, differences in NGAL isoforms, such as the 25 kDa monomer and the 45 kDa homodimer, may influence its utility in diagnosing AKI and sepsis.[11]

***cfDNA***

cfDNA has garnered attention as a significant biomarker for various medical conditions, including sepsis and AKI. cfDNA comprises DNA fragments released into body fluids from nucleated cells undergoing extensive death, a phenomenon commonly observed in infection and stress-induced conditions.[11] These extracellular DNA fragments can serve as damage-associated molecular patterns (DAMPs), contributing to immune system activation, coagulation disturbances, and multiple-organ dysfunction syndrome (MODS).[11]

DAMPs, including cfDNA, are endogenous molecules that escape from cells under stress and trigger immune responses. These encompass nuclear and mitochondrial DNA, RNA, and various molecules like heat shock proteins.[24] Elevated cfDNA levels reflect cell death and tissue damage, especially in conditions such as sepsis and AKI. The increased release of cfDNA can exacerbate inflammation, affect coagulation pathways, and influence disease progression. In sepsis, cfDNA levels increase due to extensive immune cell death and tissue damage. cfDNA has been suggested as a predictive biomarker for assessing the severity of sepsis and its outcomes.[11]

cfDNA levels also rise in AKI, with studies showing that high cfDNA concentrations correlate with the development of AKI in septic patients. In S-AKI, cfDNA serves as a prognostic indicator for disease severity and potential outcomes.[11] cfDNA has demonstrated its potential as a biomarker for several conditions, including cancer, stroke, trauma, and transplantation. In sepsis, cfDNA's role in assessing disease severity and prognosis is supported by studies showing its association with long-term mortality and disease progression. However, cfDNA's diagnostic value can be influenced by numerous factors, such as the presence of other conditions affecting cfDNA levels or its interaction with different biomarkers.[11]

Elevated cfDNA concentrations are associated with increased endogenous thrombin potential and changes in the fibrinolytic system, both of which play critical roles in the development of microvascular thrombosis and organ dysfunction during sepsis.[25] The modulation of fibrinolysis by cfDNA remains an area of ongoing research, with implications for understanding and managing coagulation disorders in septic patients.

***Proenkephalin***

Proenkephalin (PENK) is a small peptide, weighing approximately 4.5 kDa, and is filtered solely by the glomerulus. It is derived from the precursor peptide pre-PENK A, along with enkephalins, which are natural

opioids that primarily bind to delta opioid receptors. These receptors are found in the central nervous system and kidneys.[26] Although the exact function of PENK remains unclear, it is hypothesized to play a role in regulating kidney function, through diuresis, natriuresis, or inhibition of antidiuretic hormone. PENK's stability and correlation with glomerular filtration rate (GFR) make it a promising biomarker for renal health and disease. PENK is characterized by its long in vivo half-life and stability post-collection is unaffected by age, sex, or inflammation and is not protein-bound in plasma, making it a dependable indicator of glomerular filtration injury. This stability and specificity make PENK an attractive biomarker for various renal conditions.[11]

PENK levels are strongly correlated with the severity of sepsis and AKI. In a study of 88 critically ill patients, those who developed AKI had notably higher PENK levels. The biomarker demonstrated a sensitivity of 67.9% and a specificity of 98.3%, with AUC of 0.796.[27] PENK's usefulness in sepsis was demonstrated in three significant trials, A link between PENK levels and the diagnosis and severity of AKI was discovered in research involving 101 septic patients.[28]

AKI diagnosis and the degree of sepsis were linked to higher PENK levels in different research involving 167 patients.[29] In a larger trial involving 978 patients, PENK was identified as a reliable indicator for AKI, the need for RRT, and mortality, with odds ratios of 4.0 (95% CI 3.0-5.4) for AKI and 1.5 (95% CI 1.2-1.8) for mortality.[30]

### *Kidney injury molecule-1*

Kidney injury molecule-1 a type 1 glycoprotein, is expressed on the membranes of proximal renal tubules in response to ischemic or inflammatory damage and serves as an important biomarker for detecting AKI. Although KIM-1 is effective in identifying AKI overall, there is limited evidence on its specific role in S-AKI.[10] Studies in animal models, including zebrafish, have indicated increased transcriptional levels of KIM-1 in nephritic tubules during septic conditions.[31] A cross-sectional study involving 102 patients with different causes of AKI found elevated urinary KIM-1 levels in cases of S-AKI.[32] Additionally, a prospective study with 150 sepsis patients found that urinary KIM-1, measured within the first 24 hours of admission, had an AUC of 0.91 for diagnosing S-AKI.[33]

### *L-FABP*

L-FABP is a biomarker belonging to the lipocalin protein family, which is involved in binding and transporting free fatty acids within the cytoplasm. Despite limited research on L-FABP specifically for S-AKI, it has demonstrated potential in predicting the severity of renal injury.[10] In a study involving 145 septic patients, high urinary L-FABP levels at ICU admission were linked to increased mortality. L-FABP demonstrated a superior predictive performance for mortality compared to traditional scores like APACHE II and SOFA, with an AUC of 0.99, surpassing the AUCs of 0.92 and 0.81 for APACHE II and SOFA, respectively.[34]

### *Tissue inhibitor of metalloproteinase 2 and insulin-like growth factor binding protein 7*

These proteins are involved in regulating the cell cycle and apoptosis. The combination of TIMP2 and IGFBP7 in urine has demonstrated outstanding diagnostic performance for AKI. Discovery and validation studies, including those with large groups of critically ill patients, found that this combination offered higher sensitivity and specificity compared to other biomarkers such as KIM-1, NGAL, L-FABP, and IL-18. Specifically, TIMP2/IGFBP7 had an AUC of 0.80 for detecting any AKI, which increased to 0.84 for S-AKI.[10] Furthermore, elevated levels of TIMP2 and IGFBP7 early in septic shock were found to be independent predictors of progression from mild or moderate AKI to severe AKI within the next 24 hours.[35]

## CONCLUSION

Urinary biomarkers hold significant promise in predicting renal recovery post-sepsis, offering valuable insights into the early detection and prognosis of sepsis-associated AKI. While further research is needed to refine their diagnostic accuracy, the integration of these biomarkers into clinical practice could enhance patient outcomes through more timely and targeted therapeutic interventions.

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