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MINI REVIEW

Risk factors, complications and biomarkers associated with acute kidney injury after craniotomy: a comprehensive mini-review

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Abstract

Acute kidney injury (AKI) is a devastating medical condition that occurs mostly in hospitalized patients after a serious illness or major surgery and imposes a great health and financial burden on patients. Craniotomy is one of the major surgeries in which people who undergo this operation experience critical clinical conditions. Systemic inflammation, hemodynamic variation, and pharmacological agents administered during and after craniotomy can lead to the development of AKI and a poor prognosis. Moreover, AKI itself could cause other significant complications and increase the mortality rate in patients who undergo craniotomy. Determining the pre-, peri-, and postoperative risk factors associated with AKI after craniotomy is important for its prevention. Efficient care of patients after craniotomy and effective treatment approaches should be implemented to maintain normal metabolism, enhance nerve function recovery, and control inflammation. Novel biomarkers have been recognized as reliable indicators for the diagnosis and prognosis of AKI after brain surgery. This study aimed to summarize the most relevant literature regarding the risk factors, postoperative complications, and mortality associated with AKI after craniotomy. Moreover, we reviewed the approaches to postoperative care, followed by an overview of the significant diagnostic or prognostic biomarkers of AKI following craniotomy to provide a basis for preventing and decreasing AKI.

KEYWORDS
acute kidney injury, biomarkers, craniotomy, postoperative care, risk factors

INTRODUCTION

Craniotomy is a surgical procedure involving temporary removal of a part of the skull to access the intracranial space for neurosurgical maneuvers.[1] Subdural hematomas, intracerebral hematomas, brain tumors, and aneurysms are among the medical conditions in which craniotomy is indicated.[2] In 13%–27% of cases, essential complications occur after skull surgery.[3]

Acute kidney injury (AKI) complicates many surgical procedures, markedly affecting both longevity and quality of life.[4] The first widely accepted consensus classification criteria for renal injury (Table 1) were presented in 2004 by the Acute Dialysis Quality Initiative (ADQI), which classifies AKI into five stages: Risk, Injury, Failure, Loss, and End-stage renal disease (RIFLE).[5] Later, expert working groups such as the Acute Kidney Injury Network (AKIN) refined it.[6] The incidence of postoperative AKI is reportedly 7.5% according to the AKIN AKI criteria.[7] The prevalence of AKI in patients undergoing craniotomy is 5.7%, 2.9%, and 1.3% based on the RIFLE class risk, injury, and failure categories, respectively.[8]

Here, we aimed to review the most relevant literature regarding the risk factors, postoperative complications,

TABLE 1 Classification systems for acute kidney injury.

RIFLE classification[5]		
Stage	**Cr/GFR criteria**	**Urine output criteria**
Risk	↑ Cr × 1.5 or ↓ GFR >25%	<0.5 mL/kg/h × 6 h
Injury	↑ Cr × 2 or ↓ GFR >50%	<0.5 mL/kg/h × 12 h
Failure	↑ Cr × 3 or ↓ GFR >75% or if baseline Cr ≥4 mg/dL ↑ Cr >0.5 mg/dL	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss	Complete loss of kidney function >4 weeks	
ESRD	Complete loss of kidney function >3 months	
Acute Kidney Injury Network (AKIN)[6]		
Stage	**Cr Criteria**	**Urine output criteria**
Stage 1	↑ Cr ≥0.3 mg/dL or ↑ Cr × 1.5	<0.5 mL/kg/h × 6 h
Stage 2	↑ Cr × 2	<0.5 mL/kg/h × 12 h
Stage 3[a]	↑ Cr × 3 or if baseline Cr ≥4 mg/dL ↑ Cr ≥0.5 mg/dL	<0.3 mL/kg/h × 24 h or anuria × 12 h

Abbreviations: Cr, Serum creatinine; GFR, Glomerular filtration rate.

[a]Patients who receive RRT are considered to have met stage 3 criteria, irrespective of the stage they are in at the time of RRT.

and mortality related to AKI after craniotomy. Moreover, we present evidence regarding the postoperative care of patients, followed by an overview of the significant diagnostic or prognostic biomarkers of AKI following craniotomy.

METHODS

This comprehensive review on occurrence of AKI after craniotomy, mainly focusing on risk factors, complications and biomarkers, is based on a systematic search, data extraction, and critical analysis of relevant literature. A thorough search was conducted in the PubMed, Scopus, and Web of Science databases for English-language literature on AKI following craniotomy up to April 2023.

A structured search strategy was developed using keywords and Medical Subject Headings (MeSH) terms to identify original studies related to AKI after craniotomy. In this study, the key search terms included "Acute kidney injury," "Acute renal failure," "Acute renal insufficiency," "Craniotomy," and "Craniectomy." Then, the search results were screened based on titles and abstracts, followed by a full-text review of relevant articles. The selected articles were categorized into four groups including risk factors, mortality and complications, post-craniotomy care, and biomarkers. The emphasis was on summarizing and reviewing existing knowledge, presenting it cohesively, and comprehensively providing insight into the topic.

RESULTS

Risk factors

AKI is a devastating medical condition that markedly increases the mortality and morbidity of hospitalized patients.[9] Risk factors related to AKI after craniotomy can be divided into preoperative, perioperative, and postoperative factors (Table 2), their recognition is essential for enlightened surgical decision-making.

A recent study has found that being older (>65 years) is a significant risk factor for AKI among patients in the intensive care unit (ICU). While male patients had a higher incidence of AKI compared to female patients (55.8% vs. 44.2%), there was no statistically significant difference in the occurrence of AKI between male and female patients. However, this study was conducted in a single-center ICU, which limits the generalizability of these findings.[10]

Another study of 50,691 patients by Chung et al. demonstrated that male sex, age ≥ 65 years, body mass index (BMI) ≥ 30, black race, and greater dependence on the activities of daily living might be preoperative risk factors for AKI after craniotomy. Patients with chronic obstructive pulmonary disorder (COPD), venous thromboembolism, hypertension, diabetes, congestive heart failure (CHF), chronic kidney disease (CKD), bleeding disorders, perioperative pneumonia, sepsis, emergent craniotomy, and dyspnea on moderate exertion or at rest are more likely to develop AKI after craniotomy. In contrast, there were no significant differences in superficial surgical site infection, acute infections of the myocardium, stroke, or heart attack between patients with AKI and those without AKI.

Although the sample size in this study was substantial, it was a retrospective analysis with only a 30-day follow-up, which limits the evaluation of long-term outcomes.[11] Overall, higher age (>65 years), male gender, and previous underlying disease could be a preoperative risk factor for AKI after craniotomy. However, the absence of randomization in these studies restricts the generalizability of the findings. Further, large-scale, well-designed studies are needed to confirm these associations.

Post-craniotomy hypertension can be a significant factor that increases the risk of AKI. Past medical characteristics of patients, such as premorbid hypertension and perioperative factors, are believed to be associated with the multifactorial pathophysiology of hypertension after craniotomy.[12,13] AKI was frequently observed in patients with severe hypertension after brain surgery.[14] The multifactorial causes of postcraniotomy hypertension and its influence on AKI development require further investigation, as current studies often rely on retrospective data with limited control over confounding factors.

Liu et al. investigated the effects of antithrombotic therapy on the postoperative clinical results of patients who had spontaneous intracranial hemorrhage and underwent craniotomy. The findings showed that previous anticoagulant therapy, but not antiplatelet treatment, increased the risk

TABLE 2 Studies investigating the risk factors associated with AKI after craniotomy.

Study	*n* (AKI)	Age (mean)	Male (AKI)	T. Of Cran.[a]	Risk factors		
					Preoperative	Perioperative	Postoperative
Gal et al. 1975[13]	60	61	28	—	—	—	HTN
Kim et al. 2018[17]	2363 (43)	50.2	945	Elective craniotomy	HypoAlb ACEI ARB Aspirin CCB Lower eGFR DM pRBC-T Male gender	Mannitol Furosemide	—
Guzel et al. 2019[10]	1190 (437)	62.59	653 (244)	—	Age >65 Male gender	—	—
Liu et al. 2019[15]	18,872	59.7	12,548	—	Anticoagulant therapy	—	—
Oh et al. 2019[20]	726 (39)	51.4	315 (17)	Craniotomy for primary brain tumor resection	HCh	—	HCh BC Nephrotoxic drugs H2-blockers PPIs
Oh et al. 2019[21]	968 (117)	57.0	491	Craniotomy for IH	—	HCh Inhalation anesthesia	HChMA
Chung et al. 2020[11]	344 (172)	64.56	225 (111)	—	Age ≥ 65 Male gender BMI > 30 Black race Dependence on ADL HTN DM COPD CHF CKD VTE Bleeding disorder Sepsis Dyspnea Higher ASA-PS	Pneumonia	—
Perez et al. 2020[14]	282	53.6	122	—	Severe HTN	—	
Wang et al. 2023[22]	305 (129)	56	214	Craniotomy for SCH	Higher SII Higher SUA		

Abbreviations: ACEI, Angiotensin converting enzyme inhibitor; ADL, Activities of daily living; ARB, Angiotensin receptor blocker; ASA-PS, American Society of Anesthesiologists physical status; BC, Balanced crystalloid; CCB, Calcium channel blocker; CHF, Congestive heart failure; CKD, Chronic kidney disease; COPD, Chronic obstructive pulmonary disease; DM, Diabetes Mellitus; eGFR, Estimated glomerular filtration rate; HCh, Hyperchloremia; HChMA, Hyperchloremic metabolic acidosis; HTN, Hypertension; IH, Intracranial hemorrhage, PPIs, Proton pump inhibitors; pRBC-T, Packer red blood cell transfusion; SCH, Spontaneous cerebral hemorrhage; SII, Systemic immune-inflammation index; SUA, Serum uric acid; UTI, Urinary tract infection.

[a]Type of craniotomy.

of mortality and major clinical comorbidities, such as AKI, compared with the control group.[15]

Preoperative anemia and hypoalbuminemia increase the risk of AKI after noncardiac surgery. Surgical burden, including blood loss or transfusion, instability of perioperative hemodynamics, particularly prolonged and severe intraoperative hypotension, and the use of diuretics and vasopressors, are all perioperative risk factors for the incidence of AKI after surgery. Furthermore, the occurrence of postoperative complications, including sepsis, infection, fluid overload, and other organ dysfunctions such as acute respiratory disorders, can increase the likelihood of developing AKI following a craniotomy.[16]

A retrospective study reported that a serum level of albumin less than 3.8 g/dL before operation, packed red blood cell transfusion, lower estimated glomerular filtration rate,

diabetes, and hypotension could increase the risk of AKI after brain tumor surgery. The specific classification of the brain tumor did not have an impact on the likelihood of AKI. A lower perioperative mean arterial pressure was not associated with AKI development. Pre-, peri-, and postoperative anemia were found to increase the risk of AKI after brain surgery.[17]

Consequently, preoperative anemia and hypoalbuminemia may increase the risk of AKI after craniotomy. However, these articles were retrospective with a small sample size, and more prospective trials would provide more definitive evidence.

Multiple studies have indicated that there is no significant association between elevated chloride levels (hyperchloremia) after surgery and the occurrence of AKI in patients with cerebral disorders. However, the results of these studies suffer from a lack of sufficient populations and are not randomized controlled trials.[18,19]

Nevertheless, in a retrospective observational study of 729 patients, Oh et al. demonstrated that patients with primary brain tumors who experienced perioperative hyperchloremia (HCh) were shown to have an increased risk of AKI following craniotomy. The AKI group had higher levels of serum chloride, greater preoperative estimated glomerular filtration rate, higher volume of administered balanced crystalloids, and more use of nephrotoxic medications such as proton pump inhibitors or H2 receptor antagonists until 3 days after the operation than the non-AKI group. However, there was no notable difference in the prevalence of metabolic acidosis between the two groups.[20]

In another retrospective cohort study of 968 patients who underwent craniotomy for traumatic or spontaneous intracranial hemorrhage, the occurrence of AKI after surgery was associated with perioperative hyperchloremic metabolic acidosis (HChMA) until 3 days after surgery. Perioperative HCh was found to be associated with a higher risk of HChMA but was not directly associated with postoperative AKI development.[21] Totally, some research has shown that HCh is not related to the risk of AKI after craniotomy. In contrast, other studies suggest a connection between the two, indicating the need for additional prospective studies with larger sample sizes to clarify this relationship.

A recent retrospective study of 305 patients who underwent craniotomy due to spontaneous cerebral hemorrhage found that higher preoperative systemic immune-inflammation index and serum uric acid concentrations are related to an increased likelihood of developing AKI. The systemic immune-inflammation index (SII) can be determined by multiplying the neutrophil count and monocyte count, and then dividing the result by the lymphocyte count. The optimal SII cutoff value proposed by Wang et al. is 1794.43 with specificity and sensitivity of 41.1% and 65.9%, respectively.[22]

Mortality and complications

AKI significantly affects both the quality and length of life. The requirement for hemodialysis and prolonged hospital stay is increased in patients with AKI, posing an additional financial burden to the healthcare system.[8,10] AKI is one of the five major complications associated with mortality after elective craniotomy.[23]

In a study of 32,695 patients who underwent elective craniotomy, 0.2% of cases were complicated by renal failure, corresponding to 23.9% 30-day mortality. After myocardial infarction, renal failure was the second complication most strongly associated with mortality independent of preoperative risk factors and other complications. AKI remains a complication associated with higher 30-day mortality rates even after preoperative risk stratification for mortality.[23]

AKI was strongly associated with mortality after craniotomy in a retrospective cohort of 1656 craniotomy patients. The overall death rate was 44.7% in patients with AKI, whereas it was 17.7% in individuals without AKI. The cohort study of 282 individuals demonstrated that the RIFLE classification of AKI as risk, injury, or failure significantly predicted 30-day mortality. Furthermore, Acute respiratory failure has emerged as an effective modifier of the association between AKI and mortality. In contrast, sex, age, race, CKD stage, sepsis, Deyo-Charlson Index, trauma, subarachnoid hemorrhage, and intracranial hemorrhage were not effect modifiers of this association. The study found that the odds of hospital readmission were 1.8-, 6.7-, and 13.4-folds greater in AKI patients with RIFLE class risk, injury, and failure, respectively, 30 days after discharge.[8]

Güzel et al. have shown that AKI patients spend more time in the ICU and need more hemodialysis than non-AKI patients. Moreover, the mortality rate in AKI patients was markedly greater than that in those who did not have AKI.[10]

A retrospective cohort of 348 craniotomy patients, 174 of whom developed AKI postoperatively, had a higher number of complications in the AKI group. According to this study, complications most strongly associated with AKI in adult patients undergoing craniotomy were sepsis, urinary tract infection, pneumonia, reintubation, venous thromboembolism, and perioperative transfusions, respectively. However, this study did not find a higher risk of myocardial infarction, cardiac arrest, stroke, or surgical site infection in the AKI group. It should be noted that the study size may not be sufficient to significantly associate low rates of complications with AKI.[11]

Posti et al. conducted a study on 2166 elderly patients (age ≥ 60 years) who had undergone craniotomy and were diagnosed with traumatic brain damage. Renal failure was correlated with a higher risk of both 30-day and 1-year mortality in univariate models. However, this association was not significant in multivariate models.[24]

Altogether, numerous articles indicate that the AKI after craniotomy increases the mortality rate of pateints significently. Additionally, these studies suggest that AKI after craniotomy leads to increased complications. However, more extensive and robust research is necessary to clarify these relationships and accurately determine the morbidity rate associated with AKI in this context.

Post-craniotomy care of patients

Patients with craniocerebral injury are more prone to multiple organ dysfunction due to systemic inflammatory response syndrome (SIRS) and stress-induced organ damage. The administration of treatment measures to maintain normal metabolism, enhance nerve function recovery, and control inflammation is crucial for preventing organ dysfunction. Figure 1 shows a theoretical model of AKI pathophysiology following craniotomy and the inhibitory effect of postoperative treatments on the underlying mechanisms.

Fluid therapy is important in the postoperative care of patients after neurosurgical operations because of dehydration and the use of diuretics to reduce cerebral edema. This randomized clinical trial with 44 patients have shown that normal saline administration in large volumes can cause HChMA. Metabolic status could be maintained more favorably in these patients using a balanced crystalloid (BC) without adversely affecting brain relaxation.[25]

Moreover, a rare case of nephrolithiasis following administration of hypertonic saline after craniotomy due to traumatic brain injury has been reported. Hypertonic saline can cause metabolic acidosis because of its pH of 5, leading to the release of calcium and phosphate into the serum, making calcium phosphate stones potentially due to an underlying non-diagnosed incomplete distal tubular acidosis.[26] While this case report is informative, it emphasizes the need for larger studies to better understand the risks associated with hypertonic saline.

Mild hypothermia treatment after craniotomy is considered an efficient physical therapy to reduce brain metabolism and neuronal cytotoxicity as well as suppress neuronal apoptosis.[27,28] Therefore, the impact of mild hypothermia treatment after craniotomy on the activity of essential organs, including the brain, heart, and kidneys, was investigated. Hypothermia was induced early after the craniotomy, and body temperature was regulated at 33–34°C until the intracranial pressure returned to normal levels for at least 24 h. Patients treated with mild hypothermia had significantly lower serum levels of neutrophil gelatinase-associated lipocalin (NGAL), a known biological marker for AKI diagnosis, at the end of the operation, 24 h after the operation, and 2 weeks after the operation.[29]

Consequently, these meta-analysis and retrospective studies revealed that this method could effectively inhibit organ dysfunction by avoiding SIRS in patients with brain injury, thereby improving their prognosis. However, larger-scale randomized trials are needed to gain a clearer understanding of this relationship.

Biological or biochemical markers

Unfortunately, the early diagnosis of AKI remains a challenge. Blood urea nitrogen (BUN) and serum creatinine (Cr) are frequently employed indicators of renal function. However, they cannot precisely determine whether the renal failure is acute or chronic. The combination of functional

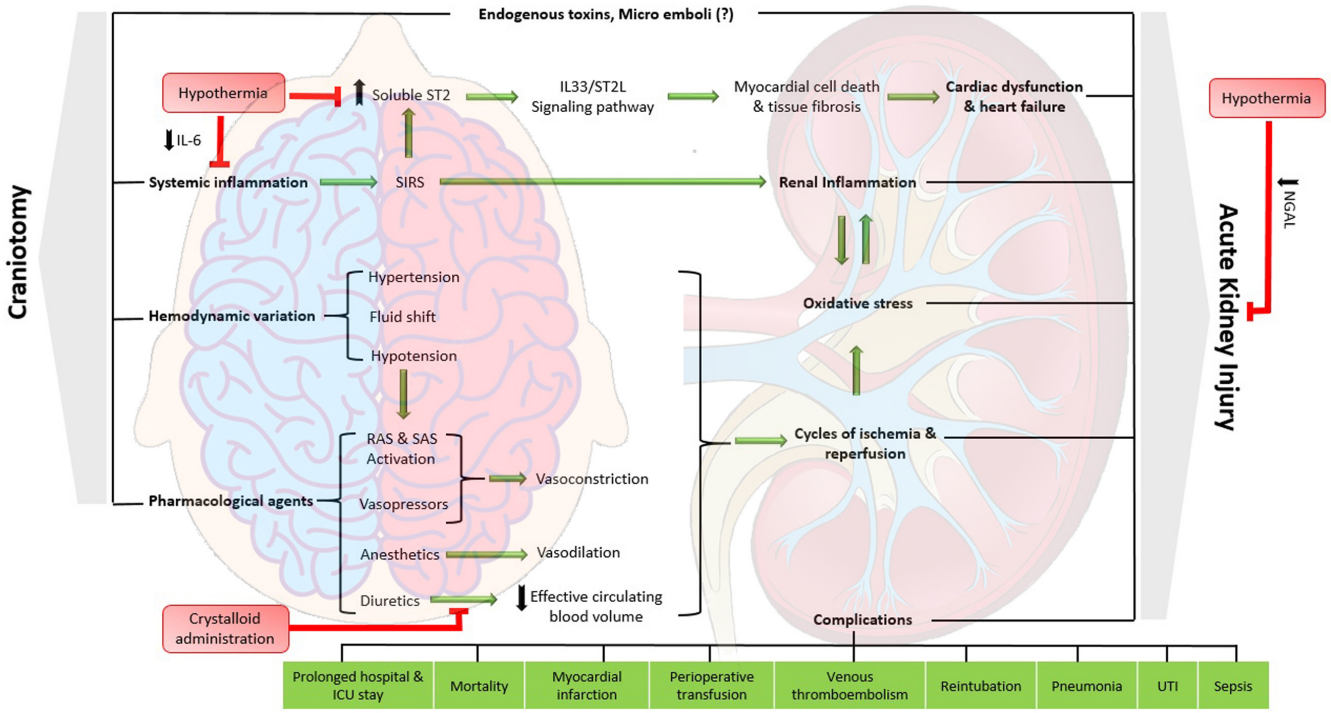

FIGURE 1 A theoretical model illustrating pathophysiology of AKI following craniotomy. Craniotomy can lead to AKI through mechanisms including hemodynamic instability and inflammation. Effect of fluid therapy and induced hypothermia as treatment approaches postoperatively are also demonstrated. Abbreviations: AKI, Acute kidney injury; ICU, Intensive care unit; RAS, Renin-angiotensin system; SAS, Sympathetic adrenomedullary system; SIRS, Systemic inflammatory response syndrome; UTI, Urinary tract infection.

and tubular damage biomarkers may enhance the accuracy of detecting AKI. Nevertheless, the precise ability of this combination to accurately diagnose AKI following neurosurgery is still uncertain.

Serum cystatin C (sCysC) and urine albumin/creatinine ratio (uACR) are classified as functional biomarkers, whereas urinary N-acetyl-β-D-glucosaminidase (uNAG) indicates tubular injury. Deng et al. demonstrated that AKI could be identified accurately and early after the resection of intracranial lesions by comprehensive investigation of all functional and tubular biomarkers including sCysC, uACR, and uNAG.[30]

Recent research indicates that kidney injury molecule-1 (KIM-1) and NGAL are newly discovered biomarkers that can be used to accurately define AKI.[31,32] The roles of KIM-1 and NGAL in the early diagnosis of AKI after mild-to-moderate brain trauma were investigated in an experimental rat model. It was shown that the plasma levels of NGAL and KIM-1 significantly increased in traumatic rats after both 2 and 24 h, which could lead to the early diagnosis of AKI within 24 h after brain trauma, leading to lower mortality and hospitalization.[33] However, translating these findings from animal models to human clinical practice remains a challenge and requires further clinical trials.

The role of procalcitonin (PCT) in predicting AKI after traumatic brain injury was evaluated in 214 patients. PCT levels were higher in the AKI group, which might be a result of oxidative stress or toxicity of PCT to mesangial cells.[34]

Previous studies have shown that hemodynamic parameters of renal arteries, particularly the resistance index (RI), change earlier than the elevation of BUN and Cr levels in early AKI.[35,36] Hemodynamic changes in renal blood flow can be monitored using color Doppler ultrasonography.[37] Hu et al. investigated the relationship between changes in renal hemodynamics and renal function in patients who underwent traumatic brain surgery using color Doppler flow imaging (CDFI). CDFI of the interlobar renal arteries showed that the RI value changed prior to the increase in serum Cr levels. Moreover, the postoperative 1 h and 48 h RI values were significantly higher in AKI patients, showing that RI evaluation may lead to an early diagnosis of AKI.[38]

Totally, several potential biomarkers have been identified that are associated with renal injury. However, their clinical applicability in everyday medical practice must be confirmed through larger-scale studies to ensure their reliability and effectiveness. These studies would help establish standardized protocols for utilizing these biomarkers in the diagnosis and management of AKI after craniotomy.

CONCLUSION

AKI is a significant medical condition that mostly complicates in-hospital patients following a serious illness or major surgery. It is crucial to thoroughly evaluate pre-, peri-, and postoperative risk factors in patients eligible for craniotomy in order to prevent the occurrence of AKI following the procedure. Higher age (>65 years), male gender, previous underlying disease, previous anticoagulant therapy, preoperative anemia and hypoalbuminemia, higher SII index, and serum uric acid concentrations may be related to an increased likelihood of developing AKI after craniotomy. Prolonged hospital stay and mortality are the major complications of AKI, highlighting the immediate use of renal replacement therapy to achieve a good prognosis. The administration of treatment measures including optimum fluid therapy and mild hypothermia after craniotomy to maintain normal metabolism, reduce neuronal cytotoxicity, and control inflammation is considered to effectively inhibit organ dysfunction. Novel biomarkers have been recognized as reliable indicators for the diagnosis and prognosis of AKI, suggesting that functional and tubular biomarkers should be considered together for more precise decision-making.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

Approval of the research protocol: N/A.
Informed consent: N/A.
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