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ORIGINAL RESEARCH

Metabolic Syndrome Is Associated with the Prognosis in Elderly Critically Ill Patients with Acute Kidney Injury

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Purpose: Metabolic syndrome (MetS) is linked to adverse outcomes in chronic diseases, but its impact on acute kidney injury (AKI) in elderly critically ill patients remains unclear. This study aimed to evaluate the association between MetS and 90-day mortality in this population.

Patients and Methods: A retrospective analysis included 774 elderly patients (≥65 years) with AKI admitted to the ICU from January 2022 to December 2023. MetS was defined as the presence of at least three of the following: central obesity, hypertension, dyslipidemia, and hyperglycemia. Propensity score matching (PSM) balanced baseline characteristics between MetS and non-MetS groups. The primary outcome was 90-day all-cause mortality, and the secondary outcome was renal recovery at discharge. Multivariate Cox regression assessed the independent association of MetS with 90-day mortality.

Results: After PSM, 294 patients (147 MetS and 147 non-MetS) were included. The MetS group had a significantly higher 90-day mortality rate compared to the non-MetS group (44.9% vs 31.3%, p=0.016). Multivariate analysis showed that MetS was independently associated with an increased risk of mortality (HR=1.606, 95% CI: 1.080–2.386; p=0.019). A dose-response relationship was observed, with increasing number of MetS components associated with higher mortality risk (HR=1.382, 95% CI: 1.121–1.831; p=0.001). Additionally, patients with MetS had lower rates of full renal recovery compared to those without (74.8% vs 86.4%, p=0.040).

Conclusion: MetS is independently associated with increased 90-day mortality and impaired renal recovery in elderly critically ill patients with AKI.

Keywords: acute kidney injury, elderly patients, metabolic syndrome, mortality, renal recovery

Introduction

Acute kidney injury (AKI) is a significant clinical syndrome characterized by a sudden decline in kidney function, typically induced by ischemic, toxic, or inflammatory insults. Globally, AKI affects approximately 13.3 million individuals annually and contributes to 1.7 million deaths each year, making it a substantial global health burden.[1] AKI occurs in up to 50% of critically ill patients,[2] with mortality rates ranging from 20% to 50%, depending on severity and comorbidities.[3,4] Elderly patients are particularly vulnerable, exhibiting higher incidence and mortality rates due to factors such as "kidney aging" polypharmacy, and comorbidities, underscoring the need for focused attention on AKI progression in this population.[5,6]

Metabolic syndrome (MetS), a well-established cluster of metabolic abnormalities including central obesity, hypertension, dyslipidemia, and insulin resistance, affects approximately 25% of the global adult population and is known to elevate the risk of AKI.[7,8] Several individual components of MetS—such as hypertension, obesity, and insulin resistance —have been independently linked to higher mortality in AKI patients.[9–11] However, data examining the association between MetS as a whole and mortality or renal outcomes in AKI patients remain scarce. In particular, the potential dose-

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dependent relationship between the number of MetS components and adverse outcomes in this population has not been clearly elucidated. Some limited studies suggest that metabolic derangements may exacerbate renal dysfunction through mechanisms such as systemic inflammation, endothelial dysfunction, and oxidative stress.[12]

Supporting these concerns, emerging evidence from prospective cohort studies of kidney donors shows that metabolic abnormalities can lead to long-term adverse renal outcomes. For example, Tarabeih et al found that female kidney donors with prediabetes had significantly higher blood pressure, proteinuria, and lower estimated glomerular filtration rate (eGFR) five years after donation compared to normoglycemic donors.[13] Another study by the same group showed that prediabetic donors with BMI >30 prior to donation experienced worsening diabetes control and kidney function over a seven-year follow-up period.[14] These findings underscore the systemic and renal consequences of metabolic dysregulation, reinforcing the biological plausibility that MetS contributes to poor renal outcomes in critically ill AKI patients.

To our knowledge, no prior study has specifically evaluated the impact of MetS on short-term mortality and renal recovery in elderly critically ill AKI patients. Moreover, the potential dose-response relationship between the number of MetS components and adverse outcomes in this population has not been previously explored. Addressing this knowledge gap is essential for refining risk stratification and identifying targets for intervention. Therefore, this study aims to evaluate the association between MetS and 90-day all-cause mortality and renal recovery in critically ill patients with AKI. The 90-day timeframe was chosen to capture both immediate and delayed effects of AKI, aligning with established research practices and enabling consistent comparisons.[15,16]

Materials and Methods

Patients

This retrospective study included elderly patients admitted to the ICU of our hospital from January 2022 to December 2023 who were diagnosed with acute kidney injury (AKI) based on KDIGO criteria. Patients were eligible for inclusion if they met all of the following: 1) Age ≥ 65 years at ICU admission; 2) Diagnosis of AKI according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines (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 urine output <0.5 mL/kg/h for 6 hours);[17] 3) Availability of complete medical records, including baseline demographics, laboratory data, and clinical outcomes necessary for analysis. Given the study's aim to evaluate the prognostic impact of metabolic syndrome in elderly patients with AKI, we focused on ICU admissions to capture a critically ill population where both conditions are prevalent and clinically impactful. This selection facilitates assessment of outcomes within a high-risk group, enabling clearer detection of associations.

Exclusion criteria were: 1) Patients with end-stage renal disease (ESRD) or on chronic dialysis prior to admission; 2) Patients with incomplete or missing key clinical or laboratory data; 3) Patients who had undergone kidney transplantation; or 4) Patients discharged or deceased within 24 hours of ICU admission (to exclude acute deaths not related to studied factors).

A detailed flowchart of patient selection is presented in Figure 1. A total of 903 patients diagnosed with AKI in our ICU were initially enrolled in this study. After excluding 129 cases, the final analysis included 774 participants, among whom 241 (31.1%) were identified as having MetS.

Definition of MetS

MetS represents a constellation of interrelated metabolic abnormalities, and various diagnostic criteria have been established globally. In this study, we utilized the criteria outlined by the Chinese Diabetes Society (CDS).[18] According to these criteria, MetS is diagnosed when an individual meets three or more of the following conditions: (1) obesity, defined as a body mass index (BMI) ≥25 kg/m^2 for Asian populations; (2) hyperglycemia, indicated by fasting blood glucose ≥6.1 mmol/L, 2-hour plasma glucose ≥7.8 mmol/L, or a prior diagnosis of diabetes; (3) hypertension, characterized by systolic/diastolic blood pressure ≥140/90 mm Hg or ongoing antihypertensive treatment; and (4) dyslipidemia, marked by triglycerides ≥1.7 mmol/L or high-density lipoprotein cholesterol (HDL-C) levels <0.9 mmol/L

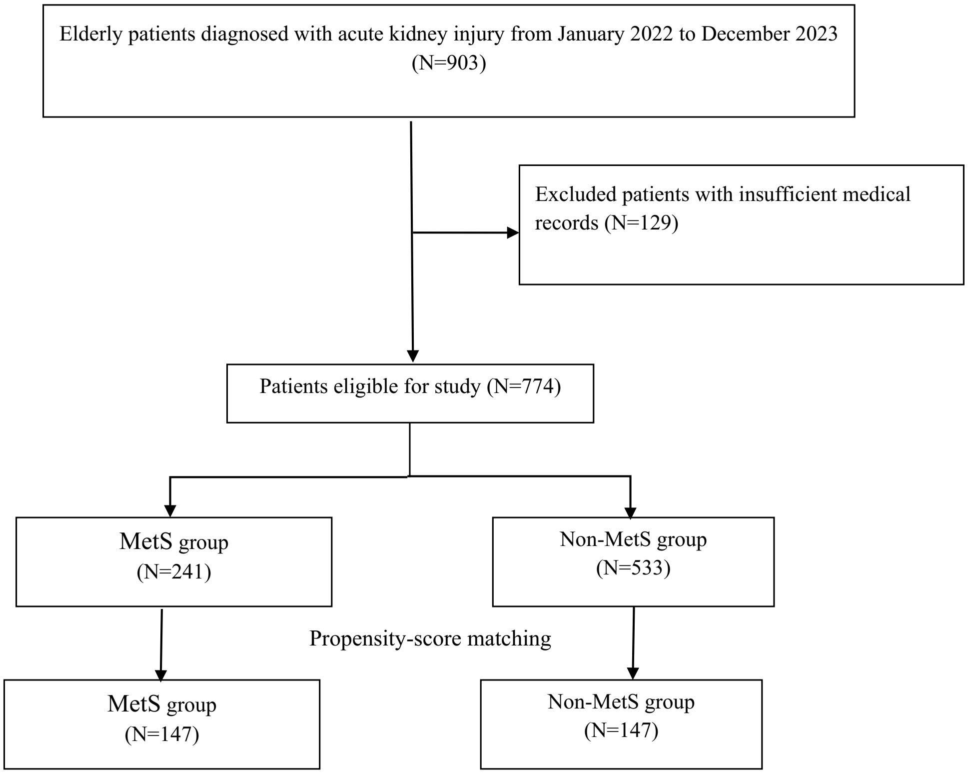

Figure 1 Patients' selection flow.

in men or <1.0 mmol/L in women. Based on these criteria, patients were stratified into two groups: those with MetS and those without, at the time of AKI diagnosis.

Data Collection and Outcomes

Patient data were extracted from electronic medical records using a standardized abstraction form. Baseline characteristics, collected within 24 hours of ICU admission, included demographic information (age, gender, BMI), etiology, CKD stages, primary reasons for ICU admission, comorbidities, severity scores (Acute Physiology and Chronic Health Evaluation II [APACHE II] and Sequential Organ Failure Assessment [SOFA]), Kidney Disease: Improving Global Outcomes (KDIGO) staging, and laboratory parameters such as serum calcium, albumin, creatinine, and parathyroid hormone (PTH) levels. To ensure accuracy, two independent researchers reviewed the medical charts, and any discrepancies were resolved through consensus. All data were anonymized to protect patient confidentiality.

The primary endpoint was 90-day all-cause mortality, with survival data obtained from medical records or, when necessary, through telephone follow-ups. The secondary endpoint focused on renal function recovery, assessed by comparing discharge creatinine levels to baseline values. Recovery was categorized as follows: full recovery (creatinine within 20% of baseline), partial recovery (creatinine 20%–60% above baseline), or poor recovery (creatinine >60% above baseline). Baseline creatinine was defined as the most recent measurement taken between 365 and 7 days prior to hospital admission. If baseline data were unavailable, the creatinine level at hospital admission was used as the reference. Discharge creatinine was recorded as the final measurement before hospital discharge.

Statistical Analysis

To minimize potential bias arising from baseline differences between the MetS and non-MetS groups, propensity score matching (PSM) was applied. Propensity scores were estimated through logistic regression, incorporating covariates such as age, sex, primary reason for admittance to the ICU, sepsis, need of mechanical ventilation, comorbidities (cardiovascular disease [CVD], chronic obstructive pulmonary disease [COPD], and malignant tumor), severity scores, and laboratory tests. These variables were selected based on prior literature demonstrating their relevance to both AKI prognosis and metabolic abnormalities,[19–21] as well as their availability and completeness in our dataset. Missing data for covariates was handled using multiple imputations prior to PSM. A 1:1 matching was conducted between MetS and non-MetS patients using a nearest-neighbor algorithm without replacement, with a caliper width set to 0.2 times the standard deviation of the logit of the propensity score. Previous research has shown that this caliper can effectively reduce more than 90% of the confounding bias while maintaining an optimal balance and minimizing sample loss.[22] In this study, PSM yielded 147 matched pairs, resulting in 294 patients for subsequent comparisons.

Based on an anticipated 13% difference in 90-day mortality between the MetS and non-MetS groups and a significant level of 0.05, a total sample size of 294 patients provided over 80% statistical power to detect a significant difference.

A Log rank test was used to test differences in time to mortality between two groups which were graphically presented by Kaplan-Meier curves. Multivariate Cox regression analysis, using a forward stepwise approach, was used to evaluate the predicting value of MetS for mortality, after controlling for potential cofounding factors including demographic variables, comorbidities, clinical and laboratory data, and severity of illness. These adjustments were made to ensure that the observed associations were not confounded by these factors. All tests were 2-sided and a p value <0.05 was considered statistically significant.

All statistical analyses were performed with the SPSS statistical software program package (SPSS version 22.0 for Windows, Armonk, NY: IBM Corp).

Results

The baseline characteristics of the matched cohorts are summarized in Table 1. The MetS and non-MetS groups were well-balanced in terms of age, gender, primary reasons for ICU admission, comorbidities, APACHE II, SOFA, KDIGO stages, and laboratory parameters (all p>0.05). However, significant differences were observed in MetS-related parameters, including higher BMI, elevated systolic and diastolic blood pressure, and increased fasting glucose, triglyceride,

Table 1 Patients' Baseline Demographics and Clinical Characteristics

	Non-MetS	MetS	*p* Value
N	147	147	
Age (years)	69.0 ± 2.6	69.5 ± 3.1	0.149
Gender			0.723
Male	84 (57.1%)	88 (59.9%)	
Female	63 (42.9%)	59 (40.1%)	
MetS number, n (%)	1.6 ± 0.5	3.3 ± 0.5	<0.001
BMI (kg/m^2)	22.0 ± 2.3	25.8 ± 3.1	<0.001
SBP, mmHg	110.0 ± 5.5	141.6 ± 18.0	<0.001
DBP, mmHg	72.1 ± 4.3	87.4 ± 7.2	<0.001
Fasting glucose, mmol/L	5.8 ± 0.9	8.5 ± 0.9	<0.001
Triglyceride, mmol/L	1.1 ± 0.2	1.7 ± 0.3	<0.001
TC, mmol/L	4.4 ± 0.2	4.8 ± 0.4	<0.001
Etiology			0.968
Pre-renal	78 (53.1%)	80 (54.4%)	
Intra-renal	52 (35.4%)	50 (34.0%)	
Post-renal	17 (11.6%)	17 (11.6%)	
CKD stage			0.716
Stage 1–2 (eGFR ≥60)	68 (46.3%)	66 (44.9%)	
Stage 3a (eGFR 45–59)	39 (26.5%)	37 (25.2%)	
Stage 3b (eGFR 30–44)	28 (19.4%)	26 (17.7%)	
Stage 4–5 (eGFR <30)	12 (8.2%)	18 (12.2%)	

(*Continued*)

Table 1 (Continued).

	Non-MetS	MetS	*p* Value
Primary reason for admittance to the ICU			0.899
Medical	106 (72.1%)	103 (70.1%)	
Surgical	25 (17.0%)	28 (19.0%)	
Trauma	16 (10.9%)	16 (10.9%)	
Sepsis at admission to ICU	91 (61.9%)	98 (66.7%)	0.394
Need of mechanical ventilation (%)	54 (36.7%)	41 (27.9%)	0.105
Comorbidities			
CVD	52 (35.4%)	43 (29.3%)	0.262
COPD	24 (16.3%)	30 (20.4%)	0.366
Malignant tumor	13 (88.8%)	17 (11.6%)	0.441
APACHE II score	18.9 ± 2.8	19.2 ± 2.5	0.241
SOFA score	5.7 ± 1.0	5.5 ± 1.0	0.209
KDIGO stage			0.633
Stage I	42 (28.6%)	48 (32.7%)	
Stage II	67 (45.6%)	67 (45.6%)	
Stage III	38 (25.9%)	32 (21.8%)	
Serum calcium (mg/dl)	9.0 ± 1.2	9.0 ± 1.2	0.631
Serum albumin (g/dl)	3.3 ± 0.4	3.3 ± 0.4	0.528
Serum creatinine (mg/dl)	1.1 ± 0.2	1.1 ± 0.2	0.948
PTH (pg/mL)	49.5 ± 5.4	50.0 ± 5.7	0.368

Abbreviations: MetS, metabolic syndrome; BMI, body mass index; CKD, chronic kidney diseases; CVD, cardiovascular diseases; COPD, Chronic obstruction pulmonary disease; eGFR, estimated Glomerular Filtration Rate; ICU, intensive care unit; APACHE II, Acute Physiology and Chronic Health Evaluation II; SOFA, Sequential Organ Failure Assessment; KDIGO: The Kidney Disease, Improving Global Outcomes; PTH, parathyroid hormone.

and total cholesterol levels (all $p<0.001$). These findings confirm the successful matching of the cohorts while highlighting the distinct metabolic profiles of the MetS group.

The 90-day all-cause mortality rate, including deaths occurring during hospitalization and post-discharge, was significantly higher in the MetS group compared to the non-MetS group (44.9% vs 31.3%, $p=0.016$). As shown in Figure 2A, Kaplan-Meier survival curves showed that patients with MetS had a significantly higher risk of 90-day all-cause mortality compared to those without (unadjusted HR=1.639, 95% CI: 1.125–2.389; $p=0.010$). Multivariate Cox proportional hazards analysis, adjusted for potential confounders, demonstrated that MetS was independently associated with a 1.606-fold increased risk of mortality (95% CI: 1.080–2.386; $p=0.019$) compared to the non-MetS group (Table 2). Additionally, intra-renal (HR=2.867, 95% CI: 1.753–2.545; $p<0.001$), post-renal (HR=2.005, 95% CI: 1.074–3.743; $p=0.029$), CKD stage 4–5 (HR=3.005, 95% CI: 1.687–5.352; $p<0.001$), sepsis (HR=1.656, 95% CI: 1.059–2.591; $p=0.027$), and APACHE II score (HR=1.111, 95% CI: 1.024–1.205; $p=0.011$) were also significantly associated with increased risk of mortality.

Additionally, a dose-response relationship was observed between the number of MetS components and survival outcomes (Figure 2B). Multivariate analysis showed that each additional MetS component was associated and a 1.382-fold increased risk of mortality (95% CI: 1.121–1.831; $p=0.001$). Specifically, compared to patients with MetS component number of 0–1, those with 2, 3, and 4 had 1.211-fold (95% CI: 0.672–1.931, $p=0.372$), 1.769-fold (95% CI: 1.033–2.741, $p=0.031$), and 2.176-fold (95% CI: 1.314–4.622, $p<0.001$) increased risk of mortality, respectively.

To further explore the heterogeneity in the impact of MetS on mortality, we performed stratified multivariate Cox regression analyses based on AKI etiology and baseline CKD stages (Table 3). The association between MetS and 90-day mortality was most pronounced in patients with pre-renal AKI (HR=2.471, 95% CI: 1.732–3.544; $p<0.001$), while no statistically significant associations were observed in intra-renal (HR=1.592, 95% CI: 0.974–2.072; $p=0.082$) or post-renal subgroups (HR=1.233, 95% CI: 0.748–1.873; $p=0.242$). Similarly, when stratified by baseline CKD stage, the

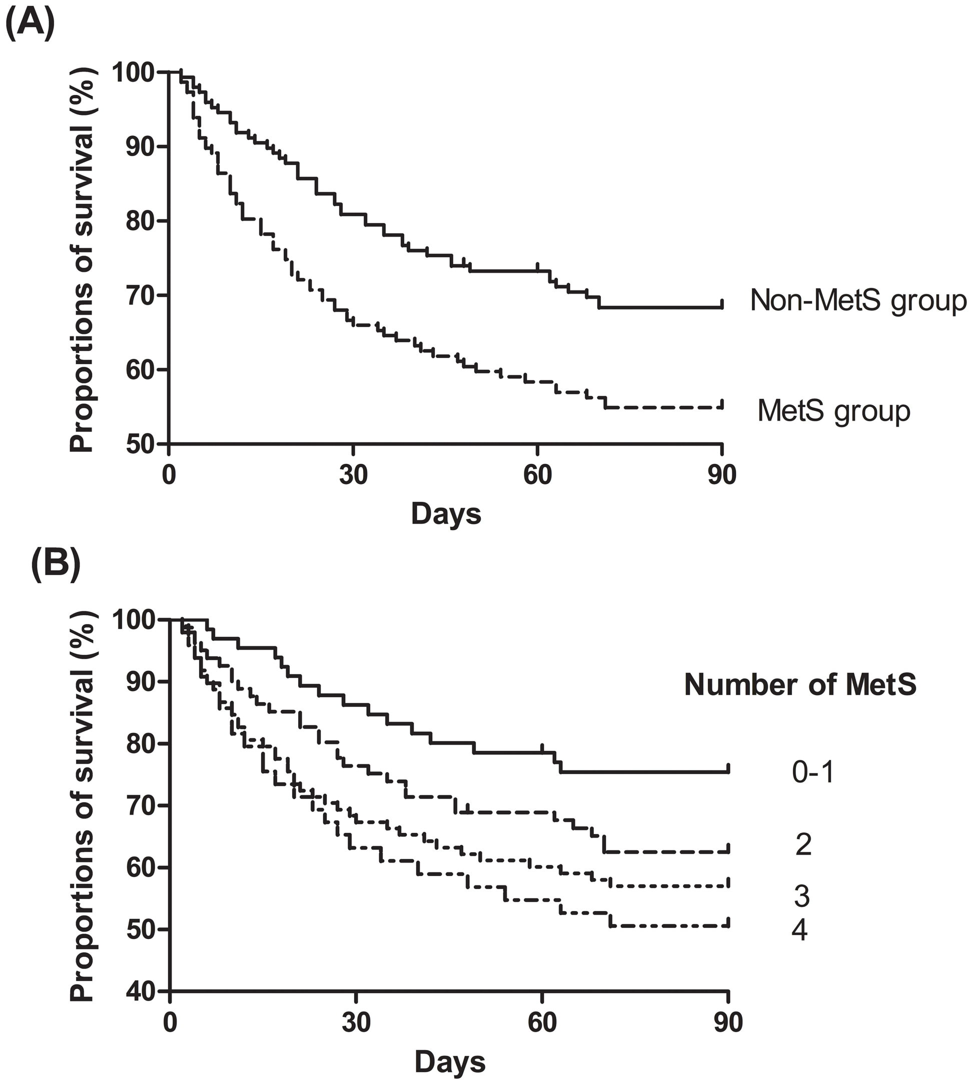

Figure 2 Kaplan-Meier curves by metabolic syndrome (MetS) status (**A**) and by the number of MetS components (**B**).

detrimental effect of MetS on mortality became more evident with declining renal function. Patients with CKD stage 3b and stage 4–5 showed significantly increased risks of mortality (HR=1.964, 95% CI: 1.215–2.964; *p*=0.012 and HR=2.772, 95% CI: 1.971–4.035; *p*<0.001, respectively), whereas no significant association was found in patients with preserved kidney function (CKD stages 1–3a).

For the second endpoint, patients with MetS demonstrated significantly lower rates of full renal recovery compared to non-MetS patients (74.8% vs 86.4%, *p*=0.040), with higher proportions of partial/poor recovery (Table 4).

Discussion

The present study demonstrates that MetS is independently associated with increased 90-day all-cause mortality and impaired renal recovery in elderly critically ill patients with AKI. After propensity score matching to balance baseline characteristics, patients with MetS exhibited a 1.728-fold higher mortality risk compared to non-MetS counterparts, with a dose-response relationship observed between the number of MetS components and mortality. Furthermore, MetS was associated with reduced rates of full renal function recovery at discharge. These findings highlight MetS as a critical prognostic determinant in AKI, emphasizing the need for targeted management strategies in this high-risk population.

While previous studies have linked MetS to adverse outcomes in CKD and cardiovascular populations, evidence in the acute care setting has been limited. For instance, a Japanese cohort of CKD patients reported significantly higher

Table 2 Multivariate Cox Regression Analysis to Evaluate the Association Between MetS Status and 90-Day Mortality in Critically Ill Patients with Acute Kidney Injury

	HR	95% CI	*p* Value
MetS	1.606	1.080–2.386	0.019
Age	0.966	0.903–1.032	0.305
Gender			
Male	Reference		
Female	1.311	0.889–1.935	0.172
Etiology			
Pre-renal	Reference		
Intra-renal	2.867	1.753–4.689	<0.001
Post-renal	2.005	1.074–3.743	0.029
CKD stage			
Stage 1–2 (eGFR ≥60)	Reference		
Stage 3a (eGFR 45–59)	0.750	0.417–1.350	0.338
Stage 3b (eGFR 30–44)	1.665	0.943–2.940	0.079
Stage 4–5 (eGFR <30)	3.005	1.687–5.352	<0.001
Primary reason for admittance to the ICU			
Medical	Reference		
Surgical	0.994	0.610–1.622	0.982
Trauma	0.641	0.322–1.279	0.207
Sepsis at admission to ICU	1.656	1.059–2.591	0.027
Need of mechanical ventilation	0.827	0.537–1.275	0.391
Comorbidities			
CVD	0.938	0.610–1.443	0.771
COPD	1.089	0.644–1.842	0.751
Malignant tumor	0.979	0.511–1.876	0.949
APACHE II score	1.111	1.024–1.205	0.011
SOFA score	1.004	0.830–1.214	0.971
KDIGO stage			
Stage I	Reference		
Stage II	0.641	0.392–1.048	0.076
Stage III	1.238	0.721–2.126	0.438
Serum calcium	0.872	0.733–1.038	0.123
Serum albumin	0.753	0.473–1.200	0.233
Serum creatinine	0.789	0.256–2.430	0.680
PTH	1.023	0.989–1.059	0.190

Abbreviations: MetS, metabolic syndrome; BM, body mass index; CKD, chronic kidney diseases; CVD, cardiovascular diseases; COPD, Chronic obstruction pulmonary disease; eGFR, estimated Glomerular Filtration Rate; ICU, intensive care unit; APACHE II, Acute Physiology and Chronic Health Evaluation II; SOFA, Sequential Organ Failure Assessment; KDIGO, The Kidney Disease: Improving Global Outcomes; PTH, parathyroid hormone.

survival in the non-MetS group ($p = 0.0086$),[23] and Canadian data from kidney transplant recipients indicated shorter time to major cardiovascular events among those with MetS ($p < 0.0001$).[24] A meta-analysis of over 160,000 individuals confirmed the association between MetS and increased risks of mortality, myocardial infarction, and stroke.[25] Our findings extend this knowledge by demonstrating that MetS also confers elevated short-term mortality risk and impairs renal recovery in patients with AKI, a condition with distinct pathophysiology and acute trajectory. The observed dose-response relationship, which has also been reported in other studies,[26] strengthens the plausibility of a causal association and reinforces the clinical significance of metabolic burden in this setting.

Importantly, subgroup analyses further revealed that the prognostic impact of MetS varied depending on the underlying etiology of AKI and baseline renal function. The association between MetS and 90-day mortality was most

Table 3 Multivariate Cox Regression Analysis to Evaluate the Association Between MetS Status and 90-Day Mortality in Critically Ill Patients with Acute Kidney Injury, Stratified by Etiology and Baseline Chronic Kidney Diseases (CKD) Stage

	MetS vs Non-MetS		
	HR	**95% CI**	***p* Value**
Etiology			
Pre-renal	2.471	1.732–3.544	<0.001
Intra-renal	1.592	0.974–2.072	0.082
Post-renal	1.233	0.748–1.873	0.242
CKD stage			
Stage 1–2 (eGFR ≥60)	1.211	0.832–1.672	0.312
Stage 3a (eGFR 45–59)	1.446	0.902–1.833	0.110
Stage 3b (eGFR 30–44)	1.964	1.215–2.964	0.012
Stage 4–5 (eGFR <30)	2.772	1.971–4.035	<0.001

Abbreviations: MetS, metabolic syndrome; CKD, chronic kidney diseases; eGFR, estimated Glomerular Filtration Rate.

Table 4 Comparison of Renal Function Recovery Between Critically Ill AKI Patients with and Without Metabolic Syndrome (MetS)

	Non-MetS	**MetS**	***p* Value**
Recovery of kidney function			0.040
Full recovery	127 (86.4%)	110 (74.8)	
Partial recovery	13 (8.8%)	39 (13.3%)	
Poor recovery	7 (4.8%)	11 (7.5%)	

pronounced in patients with pre-renal AKI and those with advanced CKD (stage 3b–5). In contrast, the relationship was weaker and not statistically significant in intra-renal and post-renal AKI, or in patients with relatively preserved renal function. These findings suggest that the detrimental effects of MetS may be amplified in scenarios where renal perfusion is compromised or renal reserve is severely limited, highlighting a potential interaction between systemic metabolic stress and intrinsic kidney vulnerability. Recognizing this heterogeneity has important clinical implications. MetS may exacerbate hypoperfusion injury in pre-renal AKI through mechanisms such as endothelial dysfunction, vascular stiffness, and impaired autoregulation. Similarly, in patients with severely impaired baseline renal function, MetS may accelerate progression to irreversible injury. Tailored metabolic interventions in these subgroups could be particularly beneficial and warrant further investigation.

MetS may worsen AKI outcomes through multiple pathways. Chronic inflammation and oxidative stress, hallmarks of MetS, likely amplify ischemic or toxic kidney injury by promoting endothelial dysfunction and impairing tissue repair.[27] Insulin resistance, a core feature of MetS, has been implicated in mitochondrial dysfunction and apoptosis in renal tubular cells.[28] Additionally, hypertension and dyslipidemia may exacerbate microvascular injury, while obesity-related adipokine imbalances could further drive systemic inflammation.[29] These mechanisms may collectively delay renal recovery and increase susceptibility to secondary complications such as sepsis, which also emerged as an independent mortality predictor in our cohort. In particular, recognizing high-risk phenotypes such as pre-renal AKI with MetS or AKI superimposed on advanced CKD may guide clinicians in risk stratification, resource allocation, and individualized supportive care.

From a clinical perspective, our findings support the incorporation of routine MetS screening into AKI management protocols. Early identification of metabolically vulnerable patients may inform personalized strategies—such as tighter glycemic and blood pressure control, lipid management, and closer surveillance during ICU stay—to mitigate risk.

This study has several limitations. First, its retrospective design introduces potential unmeasured confounders, such as dietary habits, physical activity, or medication adherence. Second, the exclusive focus on ICU patients may limit the generalizability of findings to less critically ill populations and could introduce selection bias by potentially overestimating the effect of MetS and AKI in this very sick cohort. Third, the use of the CDS criteria for MetS, while appropriate for the study population, limits direct comparability with studies employing other definitions (eg, International Diabetes Federation). Fourth, baseline creatinine estimation using hospital admission values in some cases may have led to AKI staging inaccuracies. Lastly, this was a single-center study, and generalizability to non-Asian populations remains uncertain. Future studies should validate these findings in larger, prospective cohorts across diverse populations. Future studies should validate these findings in larger, prospective cohorts with diverse populations, and explore the interaction between metabolic burden and different AKI subtypes and CKD stages using mechanistic biomarkers. Interventional trials targeting metabolic components may help define best practices for improving outcomes in AKI patients with MetS.

Conclusions

In conclusion, in elderly critically ill patients with AKI, MetS is associated with increased risk of mortality and incomplete renal recovery. This association appears to be particularly pronounced in patients with pre-renal AKI and those with advanced CKD, suggesting that the adverse impact of MetS may be modified by underlying AKI etiology and baseline renal function. These findings advocate for integrating metabolic health assessment into AKI management frameworks and highlight the urgent need for interventions targeting MetS to improve outcomes in this vulnerable population. Tailored metabolic management strategies—especially in high-risk subgroups—may help mitigate the compounded risks of mortality and poor renal prognosis. Implementing structured screening and intervention protocols for MetS in the ICU setting could enhance both survival and renal recovery in critically ill patients with AKI.

Ethics Approval and Informed Consent

The study was approved by Institutional Review Board (IRB) of Wuyi County First People's Hospital. The study was conducted in accordance with the principles of the Declaration of Helsinki. Since all data were fully anonymized before we accessed them, inform consent was waived by Wuyi County First People's Hospital. All data were stored securely, and confidentiality was maintained throughout the study.

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Disclosure

The author reports no conflicts of interest in this work.

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