



Impact of Sarcopenia on Renal Outcomes in Patients with Septic Acute Kidney Injury: A Retrospective Cohort Study

Hyung Jung Oh^{1,2*}, Jung Ho Kim^{3*}, Yun Ho Roh⁴, Jin Young Ahn³,
Su Jin Jeong³, Jun Yong Choi³, Joon-Sup Yeom³, and Nam Su Ku³

¹Department of Nephrology, Sheikh Khalifa Specialty Hospital, Ras Al Khaimah, United Arab Emirates;

²Department of Internal Medicine, Seoul National University College of Medicine, Seoul;

³Department of Internal Medicine and AIDS Research Institute, Yonsei University College of Medicine, Seoul;

⁴Biostatistics Collaboration Unit, Department of Biomedical Systems Informatics, Yonsei University College of Medicine, Seoul, Korea.

Purpose: Despite the high prevalence of sarcopenia and the significant burden of septic acute kidney injury (AKI), the impact of sarcopenia on renal outcomes in patients with septic AKI has not been studied. Therefore, we aimed to evaluate the effect of sarcopenia on the renal prognosis of patients with septic AKI.

Materials and Methods: This retrospective study enrolled patients with septic AKI between July 2008 and March 2019. Sarcopenia was identified by measuring the total abdominal muscle area using abdominal CT. Patients were divided into sarcopenia and non-sarcopenia groups. The effect of sarcopenia on 90-day renal outcomes, including a >50% decrease in the estimated glomerular filtration rate, dialysis dependence, and requirement for continuous renal replacement therapy within 90 days, was analysed. Additionally, the effect of sarcopenia on 90-day mortality was assessed.

Results: Of the 608 enrolled patients with septic AKI, 273 (44.9%) were assigned to the sarcopenia group. At baseline, there were no significant differences in the severity score and distribution of AKI stages between the two groups. There was no significant between-group difference in the 90-day renal outcomes. Moreover, sarcopenia was not associated with 90-day renal outcomes across all AKI stages in stratified multivariable logistic analysis. In contrast, the 90-day mortality rates were significantly higher in the sarcopenia group than in the non-sarcopenia group (30.0% vs. 17.3%, respectively; $p < 0.001$).

Conclusion: Sarcopenia was significantly associated with an increased 90-day mortality rate; however, no significant association with 90-day renal progression was observed in patients with septic AKI.

Key Words: Sarcopenia, septic shock, acute kidney injury, mortality

INTRODUCTION

Acute kidney injury (AKI) is common in critically ill patients.

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Corresponding author: Nam Su Ku, MD, PhD, Department of Internal Medicine, Yonsei University College of Medicine, 50-1 Yonsei-ro, Seodaemun-gu, Seoul 03722, Korea.

E-mail: smileboy9@yuhs.ac

*Hyung Jung Oh and Jung Ho Kim contributed equally to this work.

•The authors have no potential conflicts of interest to disclose.

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Approximately one in three patients in the intensive care unit (ICU) develop AKI.¹ Moreover, its incidence increased from 35.8% to 50.4% over a decade,² and patients with AKI exhibit high mortality rates of up to 60%.^{3,4} Furthermore, emerging evidence indicates that a substantial percentage of patients with AKI do not regain normal renal function.⁵⁻⁹ However, the progression rate of chronic kidney disease (CKD) from AKI differed among studies. Morgera, et al.⁵ reported that 10% of survivors still required chronic renal replacement therapy (RRT), whereas Chertow, et al.⁶ reported that 33% of survivors were still on RRT after 12 months of follow-up, and Bellomo, et al.⁷ showed that only 5.4% of survivors required RRT by day 90.

Sarcopenia is characterised by declines in muscle mass, strength, and physical function.¹⁰ Owing to several adverse fac-

tors such as malnutrition, inflammation, coexisting diseases, and inactivity, the frequency of sarcopenia is higher in critically ill individuals than in other patients (15%–50% in cancer patients and 30%–45% in patients with liver failure), and it is estimated to be 60%–70%.^{11–13} Sarcopenia is a predictor of mortality in critically ill patients.^{14–16} Moreover, there have been several studies on the association between sarcopenia and renal outcomes. Yu, et al.¹⁷ showed the relationship between CKD and sarcopenia, and Bang, et al.¹⁸ reported the impact of sarcopenia on AKI after infrarenal abdominal aortic aneurysmal surgery.

Although sepsis remains the leading cause of AKI in critically ill patients,¹⁹ most studies on sarcopenia in septic patients have focused on mortality.^{14–16} Therefore, in this study, we aimed to evaluate the effect of sarcopenia on renal progression in patients with septic AKI and to show its impact on mortality.

MATERIALS AND METHODS

Study population

We conducted a retrospective analysis of adult patients with septic AKI admitted to the emergency department (ED) of a tertiary care teaching hospital with 2400 beds in South Korea between July 2008 and March 2019. Our definition of septic shock followed the Surviving Sepsis Campaign guidelines; we used the guidelines of Dellinger, et al. until 2018 and the Sepsis-3 guidelines from 2019 onwards to define new cases of septic shock.^{20,21} We included patients with septic shock who underwent abdominal CT within 24 hours of hospitalisation. Patients with septic AKI upon admission were included and analysed. In this study, the AKI definition and staging followed Kidney Disease: Improving Global Outcomes (KDIGO) criteria.²² The definition of AKI is as follows: 1) an increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours; or 2) an increase to ≥ 1.5 times the baseline, known or presumed to have occurred within the prior 7 days. We used two methods to calculate the baseline creatinine levels: 1) we determined the lowest creatinine value recorded between 7 days and 1 year before the patient presented with septic shock; or 2) if no previous creatinine data were available, we assumed a baseline glomerular filtration rate (GFR) of 75 mL/min/1.73 m², and the corresponding serum creatinine was back-calculated using the Modification of Diet in Renal Disease equation, as described by Valette, et al. and the KDIGO clinical practice guideline.^{22–24}

At our institution, we implemented a clinical pathway for patients presenting to the ED with suspected septic shock. The pathway followed the Surviving Sepsis Campaign guidelines, which include early initiation of empiric antibiotic therapy, fluid resuscitation, and administration of vasopressors, if necessary. We previously detailed this pathway for sepsis and septic shock.²⁵ The Institutional Review Board (IRB) of the Yonsei University Health System Clinical Trial Centre approved this study

(IRB no. 4-2021-0765). Written consent from the patients was not required because of the retrospective nature of the study.

Measurement of muscle area

Abdominal CT was used to measure the total abdominal muscle area (TAMA), including the paraspinal and abdominal wall muscles. Cross-sectional TAMA values were calculated at the third lumbar vertebral level as previously described.^{26,27} The image analysis software AquariusNET Server (TeraRecon, Foster City, CA, USA) was used to analyse distinct muscle areas with Hounsfield unit (HU) thresholds. The cross-sectional areas were calculated automatically by summing the muscle tissue pixels and multiplying by the pixel area, with manual checking of the muscle tissue boundaries when necessary. TAMA values were assessed and quantified using thresholds of -29 to 150 HU. Cross-sectional TAMA values were standardised by the square of the subject's height and reported as the muscle index in units (cm²/m²).

Outcomes and variables

The primary outcome of this study was to determine whether sarcopenia affects the 90-day renal outcomes in patients with septic AKI. To achieve this, we investigated cases exhibiting a >50% reduction in the estimated GFR (eGFR) compared with the initial eGFR, dialysis dependence at 90 days, and the need for continuous RRT within 90 days in patients with septic AKI. The secondary outcome was whether sarcopenia had an impact on 90-day mortality in patients with septic AKI. We collected information on mortality using data from the Ministry of the Interior and Safety of South Korea, which handles mortality information for all Korean citizens.

We defined sarcopenia as a muscle index <45.4 cm²/m² in males and <34.4 cm²/m² in females, based on previous findings.²⁸ To assess the patients' overall comorbidities at ED visits, we utilised the Charlson Comorbidity Index (CCI), while the Sequential Organ Failure Assessment (SOFA) scores were applied to stratify disease severity. In addition, we analysed the laboratory test results obtained upon ED admission.

Statistical analysis

We compared the patients with and without sarcopenia using an independent t-test or Wilcoxon rank-sum test for continuous variables, and either the chi-square or Fisher's exact test for categorical variables. Logistic regression analysis was used to identify the risk factors for 90-day renal outcomes. Multi-variable regression models were developed to adjust for confounding variables selected based on clinical significance after checking for multicollinearity. Odds ratios (ORs) and 95% confidence intervals (CIs) were obtained from this analysis, and multicollinearity was defined as a variance inflation factor <5. The risk factors for 90-day mortality were identified using a Cox proportional hazards model, and Kaplan-Meier survival analyses and log-rank tests were conducted to compare the 90-day

mortality between the two groups. Hazard ratios (HRs) and 95% CIs were obtained using a Cox proportional hazards model. Statistical significance was set at $p < 0.05$. The R package, version 4.0.3 (R Foundation for Statistical Computing, Vienna, Austria) was used to perform all statistical analyses.

RESULTS

Baseline characteristics

Of the 608 patients, 273 (44.9%) were diagnosed with sarcopenia (Fig. 1), and we divided the patients into sarcopenia and non-sarcopenia groups. The sarcopenia group was older than the non-sarcopenia group, and there were more male patients with sarcopenia than those without sarcopenia. Moreover, there were more cases of cerebrovascular diseases and dementia in the sarcopenia group than in the non-sarcopenia group. CCI was significantly higher in patients with sarcopenia than in those without sarcopenia.

Serum lactate levels were higher and serum albumin levels were lower in patients with sarcopenia than in those without sarcopenia. Additionally, there were no significant differences in blood urea nitrogen, creatinine, or eGFR between the groups.

Moreover, there were no significant between-group differences in the SOFA score or the Acute Physiology and Chronic Health Evaluation II score; no significant differences in the distribution of AKI stages were observed at baseline between the two groups (Table 1).

Renal outcomes and 90-day mortality

As shown in Table 2, there were no significant differences in 90-day renal outcomes between the two groups. In addition, no significant difference was observed in the incidence of continuous RRT within 30 days between the two groups (21.2% in the sarcopenia group vs. 22.4% in the non-sarcopenia group, $p = 0.768$). However, the 90-day mortality rate was significantly higher in patients with sarcopenia than in those without sarcopenia (30.0% vs. 17.3%, respectively; $p < 0.001$). A separate analysis conducted on 420 patients, excluding 188 cancer patients, yielded similar findings (Supplementary Table 1, only online). Consistent results were also observed in sex-stratified subgroup analyses (Supplementary Tables 2 and 3, only online).

Univariable and multivariable logistic regression analyses of sarcopenia for renal outcomes at 90 days

When we performed logistic regression analysis for renal outcomes at 90 days, greater renal progression was revealed in AKI stage 3 than in AKI stage 1 in the univariable analysis (OR: 3.030, 95% CI: 1.449–6.335; $p = 0.003$), and renal progression was also higher in AKI stage 3 than in AKI stage 1, even after adjusting for the presence of sarcopenia, age, sex, CCI, albumin level, systolic blood pressure on admission, glucose level, and lactate level (OR: 3.495, 95% CI: 1.561–7.822; $p = 0.002$) (Table 3).

In contrast, renal progression did not appear to be affected by sarcopenia. Although the ORs for renal outcomes at 90 days were 1.252 in univariable analysis and 1.723 in multivariable analysis for sarcopenia compared with non-sarcopenia, the results were not statistically significant. Moreover, this result re-

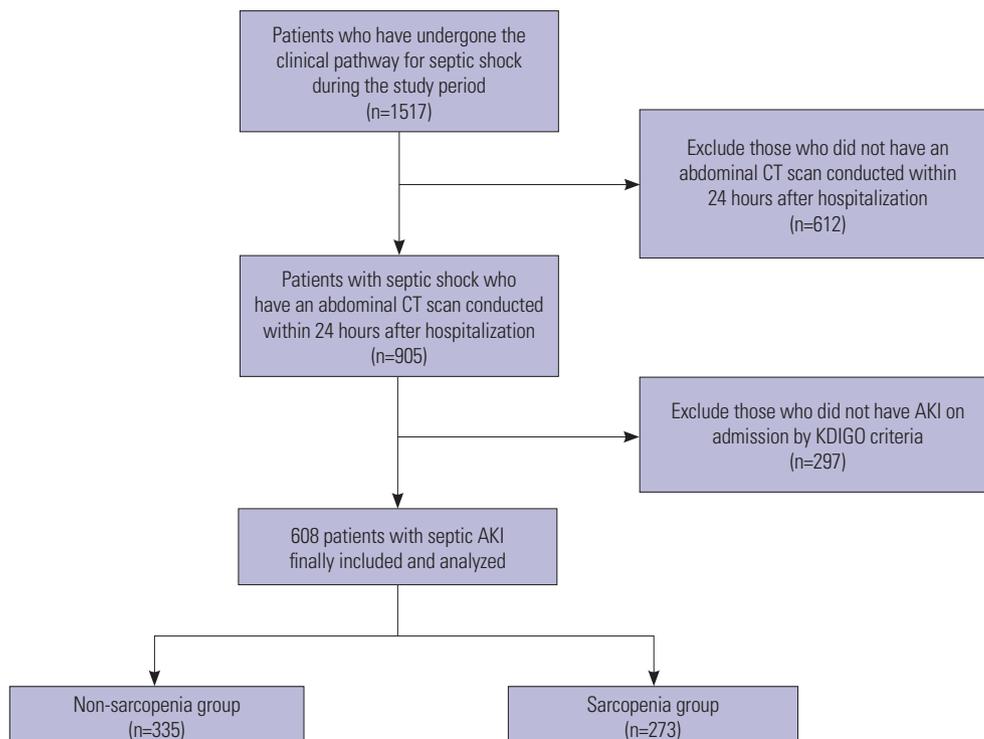


Fig. 1. Flow diagram of the study population. AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes.

mained consistent in the cohort excluding cancer patients (adjusted OR: 1.572, 95% CI: 0.936–2.641; $p=0.087$). Additionally, when the muscle index was analysed as a continuous variable, each 1-unit increase was associated with lower odds of renal

progression by 90 days (adjusted OR: 0.963, 95% CI: 0.925–1.002; $p=0.064$), although this association did not reach statistical significance.

Table 1. Baseline Characteristics of Sarcopenic and Non-Sarcopenic Patients

Variables	Non-sarcopenia (n=335)	Sarcopenia (n=273)	<i>p</i>
Age (yr)	69.0 (57.0, 76.5)	74.0 (62.0, 80.0)	<0.001
Male	112 (33.43)	181 (66.30)	<0.001
Comorbidities			
Hypertension	207 (61.79)	154 (56.41)	0.207
Diabetes mellitus	133 (39.70)	119 (43.59)	0.376
Cerebral vascular disease	55 (16.42)	66 (24.18)	0.023
Chronic liver disease	32 (9.55)	21 (7.69)	0.507
Congestive heart failure	17 (5.07)	14 (5.15)	0.768
Chronic kidney disease	65 (19.40)	45 (16.48)	0.455
Peripheral vascular disease	3 (0.90)	0 (0.0)	0.257
Coronary disease	52 (15.52)	56 (20.51)	0.135
Dementia	18 (5.37)	31 (11.36)	0.011
Chronic pulmonary disease	16 (4.78)	20 (7.33)	0.249
Hemiplegia	22 (6.57)	30 (10.99)	0.073
Cancer	106 (31.64)	82 (30.04)	0.736
Metastatic cancer	23 (6.87)	26 (9.52)	0.295
Solid organ transplantation	2 (0.60)	2 (0.73)	>0.999
AIDS	1 (0.30)	0 (0.0)	>0.999
Charlson Comorbidity Index	4.0 (3.0, 7.0)	5.0 (4.0, 7.0)	0.001
Laboratory findings			
WBC (/mm ³)	12450.0 (6625.0, 18805.0)	13575.0 (7310.0, 20480.0)	0.139
Hemoglobin (g/dL)	11.7 (10.0, 13.0)	11.9 (10.1, 13.5)	0.246
BUN (mg/dL)	32.5 (21.2, 48.0)	33.6 (23.7, 52.9)	0.068
Creatinine (mg/dL)	1.8 (1.3, 2.9)	1.8 (1.3, 2.8)	0.895
eGFR (mL/min/1.73 m ²)	35.0 (19.0, 48.0)	36.0 (21.0, 52.0)	0.184
AST (unit/L)	39.0 (23.5, 100.0)	34.5 (21.0, 86.5)	0.084
ALT (unit/L)	26.0 (15.0, 53.0)	22.0 (14.0, 48.0)	0.089
Total bilirubin (mg/dL)	0.8 (0.5, 1.5)	0.8 (0.5, 1.4)	0.168
Albumin (g/dL)	3.15±0.69	3.00±0.65	0.007
Bicarbonate (mEq/L)	17.0 (14.0, 19.0)	16.0 (13.0, 19.0)	0.584
CRP (mg/L)	132.1 (55.5, 231.2)	160.7 (74.0, 242.8)	0.139
Lactate (mg/dL)	3.3 (1.6, 5.3)	3.8 (2.1, 5.8)	0.018
SOFA score	8.0 (6.0, 10.0)	8.0 (7.0, 11.0)	0.133
APACHE II score	16.0 (12.0, 20.0)	17.0 (12.0, 23.0)	0.074
Use of nephrotoxic agents			
Aminoglycoside	5 (1.49)	3 (1.10)	0.736
Vancomycin	19 (5.67)	24 (8.79)	0.182
NSAIDs	34 (10.15)	16 (5.86)	0.077
AKI stage			0.584
Stage 1	134 (40.00)	98 (35.90)	
Stage 2	90 (26.87)	78 (28.57)	
Stage 3	111 (33.13)	97 (35.53)	
Muscle index (cm ² /m ²)	44.1 (38.8, 50.4)	34.0 (29.6, 40.2)	<0.001

AIDS, acquired immunodeficiency syndrome; WBC, white blood cell; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CRP, C-reactive protein; SOFA, Sequential Organ Failure Assessment; APACHE, Acute Physiology and Chronic Health Evaluation; NSAID, non-steroidal anti-inflammatory drug; AKI, acute kidney injury.

Data are presented as median range or n (%).

Univariable and multivariable Cox regression analyses of sarcopenia for 90-day mortality

In the univariable Cox regression analysis, sarcopenia (vs. non-sarcopenia), age, male sex, CCI, serum lactate level, and SOFA score were risk factors for 90-day mortality. The 90-day mortality rate was 1.337 times higher in AKI stage 3 than in AKI stage 1. However, the serum albumin level was a protective factor against 90-day mortality. After adjusting for age, sex, CCI, serum albumin, lactate, SOFA score, and AKI stage, the 90-day mortality increased by 1.708 (95% CI: 1.343–2.172, $p < 0.001$) in the sarcopenia group compared to the non-sarcopenia group (Supplementary Table 4, only online). The Kaplan–Meier curve also revealed that the 90-day mortality rate was significantly higher in the sarcopenia group than in the non-sarcopenia group (Fig. 2). A similar trend was observed in the cohort excluding patients with cancer (adjusted HR: 1.650, 95% CI: 1.233–2.085; $p = 0.004$). When the muscle index was analysed as a continuous variable, each 1-unit increase was associated with a lower hazard of 90-day mortality (adjusted HR: 0.974, 95% CI: 0.960–0.988; $p < 0.001$).

DISCUSSION

Sarcopenia was a significant risk factor for 90-day mortality

Table 2. Outcomes at 90 Days

Variables	Non-sarcopenia (n=335)	Sarcopenia (n=273)	p
Renal outcomes at 90 days			
>50% decreased eGFR	28 (8.36)	25 (9.16)	0.452
Dialysis dependence	2 (0.60)	1 (0.37)	>0.999
CRRT episode within 90 days	79 (23.58)	60 (21.98)	0.710
Mortality at 90 days	58 (17.31)	82 (30.04)	<0.001

eGFR, estimated glomerular filtration rate; CRRT, continuous renal replacement therapy.

Data are presented as n (%).

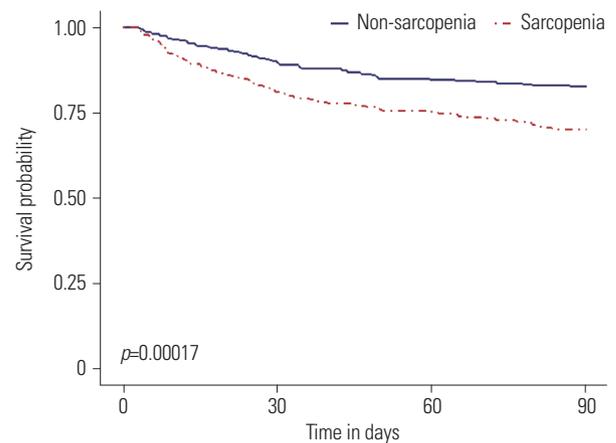
Table 3. Univariable and Multivariable Logistic Analyses of Sarcopenia for 90-Day Renal Outcomes

Variables	Univariable		Multivariable	
	OR (95% CI)	p	OR (95% CI)	p
Sarcopenia vs. none	1.252 (0.696–2.252)	0.453	1.723 (0.871–3.408)	0.118
Age (per 1 year increase)	0.980 (0.960–1.000)	0.050	0.982 (0.956–1.009)	0.190
Male vs. female	1.442 (0.798–2.606)	0.225	1.364 (0.699–2.664)	0.363
CCI (per 1 unit increase)	0.950 (0.826–1.092)	0.470	0.935 (0.816–1.071)	0.333
SBP on admission (per 1 mm Hg increase)	1.001 (0.990–1.013)	0.842	1.008 (0.995–1.022)	0.240
Albumin (per 1 g/dL increase)	0.768 (0.467–1.263)	0.298	0.850 (0.521–1.387)	0.514
Lactate (per 1 mg/dL increase)	0.949 (0.852–1.057)	0.340	0.913 (0.815–1.023)	0.117
Glucose (per 1 mg/dL increase)	0.998 (0.994–1.001)	0.152	0.997 (0.994–1.001)	0.106
AKI stage 1	Reference		Reference	
AKI stage 2	1.973 (0.892–4.362)	0.093	2.033 (0.870–4.751)	0.102
AKI stage 3	3.030 (1.449–6.335)	0.003	3.495 (1.561–7.822)	0.002

OR, odds ratio; CI, confidence interval; CCI, Charlson Comorbidity Index; SBP, systolic blood pressure; AKI, acute kidney injury.

compared to non-sarcopenia among patients with septic AKI. However, it did not affect renal progression after 90 days.

There are two types of sarcopenia, primary and secondary.^{10,29,30} Secondary sarcopenia is related to disease, nutrition, and activity, whereas primary sarcopenia is associated with the physiological aging process. Several studies have attempted to demonstrate the association between secondary sarcopenia and renal outcomes. Moreover, they found that sarcopenia was more prevalent in patients with CKD than in those with healthy kidneys and suggested that gradually decreased protein intake, increased metabolic acidosis, increased pro-inflammatory factors, and protein-energy wasting in CKD might lead to sarcopenia.^{17,18,31,32} Yu, et al.¹⁷ showed that CKD progression was independently associated with an increased risk of sarcopenia. Bang, et al.¹⁸ revealed that the incidence of postoperative AKI was higher in the sarcopenia group than in the non-



Number at risk	0	30	60	90
Non-sarcopenia	335	301	284	277
Sarcopenia	273	221	206	191

Fig. 2. Kaplan–Meier curves of the 90-day survival rates of patients with septic acute kidney injury with and without sarcopenia. The p-value was obtained through the log-rank test.

sarcopenia group; however, they did not clarify the association between the development of AKI and sarcopenia. Nevertheless, they surmised that postoperative activation of inflammation following chronic inflammation in sarcopenia may influence the incidence of AKI.

The incidence of AKI has been increasing in critically ill patients, and over 40% of these AKI cases are associated with sepsis, which largely increases medical costs and mortality.¹⁻⁴ Recently, it has been recognised that AKI itself may be a major risk factor for CKD progression, even after complete recovery. Another issue with AKI is that a substantial percentage of patients do not return to normal renal function, but the progression rate from AKI to CKD differed among studies.⁵⁻⁹ In the current study, we hypothesised that sarcopenia might affect renal progression in patients with septic AKI and attempted to evaluate the association between sarcopenia and renal outcomes at 90 days. However, we did not observe a significant association between these two factors.

Although the reason for the insignificant association between the two is not clearly understood, we make certain assumptions. First, acute sarcopenia in patients with sepsis may affect disease progression. Welch, et al.³³ used the term “acute sarcopenia” to refer to the acute loss of muscle mass and function associated with hospitalisation.^{34,35} They described that systemic inflammatory markers due to acute illness and stressor events may potentiate the acute loss of muscle mass and function via an endocrinological mechanism: hypercortisolaemia, which usually occurs in sepsis, may exacerbate the loss of muscle mass and strength during acute illness.³⁶ Moreover, sepsis is a known risk factor for ICU-acquired weakness, including critical illness myopathy, critical illness polyneuropathy, and critical illness neuromyopathy.^{37,38} Therefore, the loss of muscle mass and function might have progressed in the non-sarcopenia group during the follow-up period, suggesting that sarcopenia at baseline may be insufficient to reflect renal outcomes at 90 days. We surmise that serial measurements of sarcopenia may be useful for exploring the impact of sarcopenia on 90-day renal outcomes in these patients. Second, there were more cases of mortality in the sarcopenia group than in the non-sarcopenia group, which may have affected the investigation of the impact of sarcopenia on 90-day renal outcomes. Therefore, we explored 90-day renal outcomes in the same way using 90-day survivors who still had data at that point. However, there were still no significant differences in the 90-day renal outcomes between the two groups. Third, initial renal injury may be more strongly associated with 90-day renal progression than baseline sarcopenia. As shown in Table 3, AKI stage 3 was independently associated with increased 90-day renal progression (OR: 3.190, 95% CI: 1.449–7.019; $p=0.004$) compared to AKI stage 1, even after adjusting for confounding factors. However, sarcopenia was not significantly associated with the 90-day renal progression, even in patients with AKI stage 3.

In contrast, sarcopenia was independently associated with

increased 90-day mortality even after adjusting for several confounding factors, which is consistent with other studies.¹⁴⁻¹⁶ Taken together, we surmise that the significant association between sarcopenia and 90-day mortality may not be related to renal disease progression in patients with septic AKI.

This study has several limitations. First, this was a retrospective cohort study, which means that selection bias was inevitable. Second, we conducted the study using data from a single centre in Korea, suggesting that generalizability to other ethnicities should be interpreted cautiously. Third, we used TAMA to define sarcopenia,^{26,27} although there are different methods to measure it. Therefore, new studies with various methods of measuring sarcopenia are required. Despite these limitations, a key strength of our study is its investigation of the association between sarcopenia and 90-day renal outcomes in patients with septic AKI. While a previous study demonstrated that sarcopenia negatively affects 28-day mortality and the absence of RRT in patients with septic AKI,³⁹ there is a paucity of research examining 90-day mortality or renal outcomes. By addressing this gap, our study provides a meaningful contribution to the existing literature.

In conclusion, sarcopenia was significantly associated with an increase in the 90-day mortality rate; however, no significant association with 90-day renal progression was observed among patients with septic AKI in this study. Since sepsis is the leading cause of AKI, and whether AKI progresses to CKD has clinical significance for patient prognosis, the results of this study are worth considering. Although sarcopenia was not shown to impact 90-day renal outcomes in patients with AKI, more studies are required to explore the impact of sarcopenia on renal progression in patients with sepsis without renal injury.

AVAILABILITY OF DATA AND MATERIALS

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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AUTHOR CONTRIBUTIONS

Conceptualization: Nam Su Ku. **Data curation:** Jung Ho Kim, Jin

Young Ahn, Su Jin Jeong, Jun Yong Choi, Joon-Sup Yeom, and Nam Su Ku. **Formal analysis:** Hyung Jung Oh, Jung Ho Kim, and Yun Ho Roh. **Funding acquisition:** Hyung Jung Oh and Nam Su Ku. **Investigation:** Hyung Jung Oh and Jung Ho Kim. **Methodology:** Jung Ho Kim and Yun Ho Roh. **Project administration:** Nam Su Ku. **Resources:** Jung Ho Kim, Jin Young Ahn, Su Jin Jeong, Jun Yong Choi, Joon-Sup Yeom, and Nam Su Ku. **Software:** Jung Ho Kim and Yun Ho Roh. **Supervision:** Hyung Jung Oh and Nam Su Ku. **Validation:** Hyung Jung Oh, Jung Ho Kim, Jin Young Ahn, Su Jin Jeong, Jun Yong Choi, Joon-Sup Yeom, and Nam Su Ku. **Visualization:** Hyung Jung Oh and Jung Ho Kim. **Writing—original draft:** Hyung Jung Oh and Jung Ho Kim. **Writing—review & editing:** all authors. **Approval of final manuscript:** all authors.

ORCID iDs

Hyung Jung Oh <https://orcid.org/0000-0002-4281-696X>
 Jung Ho Kim <https://orcid.org/0000-0002-5033-3482>
 Yun Ho Roh <https://orcid.org/0000-0003-0841-7161>
 Jin Young Ahn <https://orcid.org/0000-0002-3740-2826>
 Su Jin Jeong <https://orcid.org/0000-0003-4025-4542>
 Jun Yong Choi <https://orcid.org/0000-0002-2775-3315>
 Joon-Sup Yeom <https://orcid.org/0000-0001-8940-7170>
 Nam Su Ku <https://orcid.org/0000-0002-9717-4327>

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