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Nezih KAVAK¹
Rifat BOZKUŞ²
İhsaniye SÜER DOĞAN³
Rasime Pelin KAVAK³
Berna TURHAN³
Mehmet Okan KAYHAN³

¹ Ministry of Health Etlik City Hospital , Emergency Department, Ankara, Türkiye

² Ministry of Health Etlik City Hospital , Internal Medicine Department, Ankara, Türkiye

³ Ministry of Health Etlik City Hospital , Radiology Department, Ankara, Türkiye

Correspondence

Nezih KAVAK
Phone : +905322551179
e-mail : nezih_kavak@hotmail.com

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ORIGINAL ARTICLE

THE IMPACT OF SARCOPENIA AND VISCERAL OBESITY ON IN-HOSPITAL MORTALITY IN ELDERLY PATIENTS PRESENTING TO THE EMERGENCY DEPARTMENT WITH SEPSIS

ABSTRACT

Introduction: Sepsis remains a leading cause of morbidity and mortality in elderly adults, with early risk stratification posing a significant challenge. This study aims to investigate the association between sarcopenia and visceral obesity and in-hospital mortality in elderly septic patients.

Materials and Method: This retrospective single-centre study included patients aged ≥ 65 years who presented to the emergency department with suspected infection and met the Sepsis-3 criteria. Data collected included demographics, comorbidities, infection site, body mass index, lactate and procalcitonin levels, Acute Physiology and Chronic Health Evaluation II scores, and Sequential Organ Failure Assessment scores. Abdominal computed tomography scans at admission were analysed to assess sarcopenia, defined by the Skeletal Muscle Index, and visceral obesity, determined by an elevated visceral-to-subcutaneous adipose tissue ratio.

Results: 453 patients were included (mean age, 67.8 ± 1.8 years; 55.2% female), with an in-hospital mortality rate of 42.9%. Non-survivors had significantly elevated Acute Physiology and Chronic Health Evaluation II scores, Sequential Organ Failure Assessment scores, serum lactate levels, and procalcitonin levels (all $p < 0.001$). Non-survivors also had significantly lower Skeletal Muscle Index and higher visceral-to-subcutaneous adipose tissue ratio values than survivors ($p < 0.001$). The optimal cut-off values for predicting mortality were $< 32.65 \text{ cm}^2/\text{m}^2$ for Skeletal Muscle Index and > 2.15 for visceral-to-subcutaneous adipose tissue ratio. Logistic regression showed that diabetes mellitus and malignancy were independent predictors of mortality.

Conclusion: Computed tomography-derived sarcopenia and visceral obesity are independent predictors of in-hospital mortality in elderly septic patients. These parameters may enhance early risk stratification in the emergency department.

Keywords: Sepsis; Sarcopenia; Intra-Abdominal Fat; Tomography; Aged; Mortality.

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INTRODUCTION

The word sepsis is derived from the Greek term *sepo*, meaning “to rot.” It is a life-threatening clinical condition characterised by an aberrant and overwhelming host response to infection, resulting in acute dysfunction of one or more vital organs (1,2).

Its global burden is escalating, largely driven by demographic ageing and the associated increase in frailty and chronic illnesses, which enhance infection susceptibility among elderly individuals. Epidemiological data indicate that over 60% of sepsis cases occur in persons aged 65 years and above (3,4). Mortality associated with sepsis has increased more significantly in this age group than in younger populations. The heightened vulnerability of older adults to sepsis is attributed to age-related immunosenescence, chronic low-grade inflammation, and malnutrition, all of which impair immunity and increase infection risk (1).

Sarcopenia, defined as the progressive loss of skeletal muscle mass and strength, is increasingly recognized as a major contributor to a range of adverse health outcomes. These include frailty, increased risk of falls and fractures, functional impairment, cognitive decline, and a markedly elevated risk of both morbidity and mortality (5).

Sarcopenia is commonly evaluated using the Skeletal Muscle Index (SMI), typically measured at the level of the third lumbar vertebra (L3) via computed tomography (CT) and calculated by dividing the cross-sectional area of skeletal muscle identified on CT images by the square of the individual’s height (5).

Visceral adipose tissue (VAT), a metabolically active fat depot located within the abdominal cavity surrounding internal organs, plays a key role in systemic metabolic regulation. It secretes various adipokines that influence inflammatory pathways, insulin sensitivity, and cardiovascular function (6). In contrast, subcutaneous adipose tissue (SAT), predominantly located beneath the

dermis, is primarily involved in energy storage, thermoregulation, and endocrine activity (7). The VAT-to-SAT ratio is a clinically relevant indicator of adipose tissue distribution, with visceral obesity defined by an elevated ratio. CT imaging has become a standard method for assessing this distribution, particularly in diagnosing visceral obesity based on the VAT/SAT ratio.

Identifying risk factors associated with mortality in patients with sepsis is essential for early risk stratification and the formulation of targeted therapeutic strategies (8).

This study aims to investigate the impact of CT-measured sarcopenia and visceral obesity on in-hospital mortality among elderly patients presenting to the emergency department (ED) with sepsis.

MATERIALS AND METHODS

Study Design and Participants

Following approval from the local ethics committee of Ankara Etlik City Hospital (Approval No: 2025-0195), a retrospective analysis was conducted on patients aged ≥ 65 years who presented to the ED with suspected infection between 1 January 2023 and 30 December 2023. Patients were eligible for inclusion if they fulfilled the Sepsis-3 diagnostic criteria and had undergone abdominal CT imaging at the time of ED admission (9).

Exclusion criteria encompassed patients whose CT scans failed quality assurance checks due to factors such as motion artefacts, incomplete imaging of relevant muscle or adipose tissue, or inadequate tissue differentiation, as well as those who were discharged or referred to another healthcare facility.

Data collected included demographic information (age, gender, body mass index), comorbid conditions, site of infection, initial lactate and procalcitonin levels in the ED, Acute Physiology



and Chronic Health Evaluation II (APACHE II) scores, Sequential Organ Failure Assessment (SOFA) scores, and in-hospital mortality outcomes.

Computed Tomography Measurements

Abdominal CT scans were performed using multislice CT scanner systems (GE Revolution EVO, GE Medical Systems, Milwaukee, WI, USA) at the time of admission. CT images were obtained from the institutional Picture Archiving and Communication System (PACS) and analysed using AW Volume Server 7 3.2 software (GE Medical Systems S.C.S.). For each patient, a single axial slice was selected at the level of the L3 vertebra, which serves as a validated anatomical landmark for body composition analysis (10,11).

The total cross-sectional area of skeletal muscle (SMA) was quantified (cm^2) using established Hounsfield Unit (HU) thresholds ranging from -29 to $+150$ HU (11). The skeletal muscles evaluated in the

region of interest included the m. obliquus internus abdominis, m. obliquus externus abdominis, m. transversus abdominis, m. rectus abdominis, m. psoas, m. quadratus lumborum, and m. erector spinae. The SMI was calculated by dividing the SMA (cm^2) by the square of the patient's height in metres (m^2) (11).

VAT (cm^2) and SAT (cm^2) areas were quantified from axial abdominal CT images acquired at the level of the umbilicus. Adipose tissue was segmented using a predefined HU range of -190 to -30 to identify relevant pixels within the region of interest (12).

VAT was delineated by manually tracing the intra-abdominal compartment bounded by the internal aspect of the abdominal musculature, whereas SAT was defined by tracing the area between the skin surface and the outer margin of the abdominal wall musculature on the same axial slice (Figure 1 a,b,c,d).

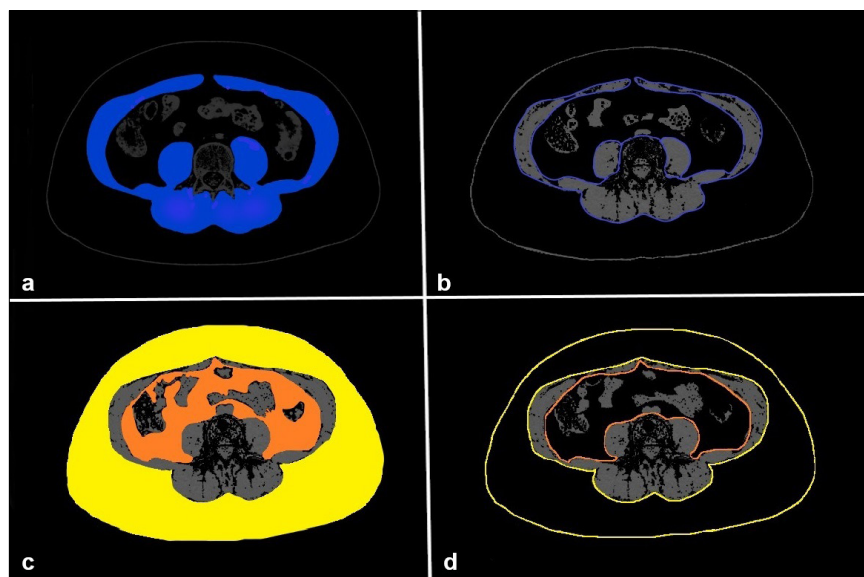


Figure 1. Computed tomography illustrations at the L3 vertebral level demonstrate the skeletal muscles highlighted in blue (a), at the same level, after applying a threshold range of -29 to $+150$ Hounsfield units, the muscle area was delineated with a blue contour and measured (b), computed tomography illustrations at the umbilical level show visceral adipose tissue in orange and subcutaneous adipose tissue in yellow (c), at this level, after applying a threshold range of -190 to -30 Hounsfield units, visceral adipose tissue was marked in orange and subcutaneous adipose tissue was outlined in yellow for area measurement (d)

All CT analyses were performed in consensus by two experienced radiologists (R.P.K. and İ.S.D.) who were blinded to all clinical data and outcomes.

Statistical Analysis

Descriptive statistics for continuous variables were expressed as mean \pm standard deviation or median with interquartile range (IQR; 25th–75th percentiles), as appropriate. Discrete variables were summarised as frequencies and percentages. The Kolmogorov–Smirnov test was employed to assess the normality of data distribution. Comparisons of continuous variables between survivors and non-survivors were conducted using the Mann–Whitney U test due to non-normal distribution. For comparisons involving nominal variables, the Chi-square test was applied. The diagnostic performance of SMA and the VAT/SAT ratio was evaluated using the area under the receiver operating characteristic (ROC) curve (AUC). Optimal cut-off values were determined using Youden's Index. The diagnostic accuracy of SMA and VAT/SAT values was further assessed by calculating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). In addition, ROC analyses were performed for the SMI within subgroups stratified by the presence or absence of comorbidities. AUC values were compared descriptively to assess whether the predictive performance of SMI varied across these subgroups. Furthermore, multivariate logistic regression analyses were conducted to evaluate the independent effects of comorbidities on in-hospital mortality. Risk factors associated with in-hospital mortality were examined using univariate logistic regression analysis. All statistical analyses were performed using IBM SPSS for Windows version 20.0 (SPSS Inc., Chicago, IL, USA), and a p-value <0.05 was considered statistically significant.

RESULTS

The study included 453 patients, with a mean age of 67.82 ± 1.84 years (range: 65–75). Of these, 250 (55.2%) were female. The overall in-hospital mortality rate was 42.9%. Compared with survivors, non-survivors had significantly higher rates of comorbid diabetes mellitus (DM), hypertension, and malignancy (all $p < 0.01$). Mortality was also significantly elevated among patients with abdominal infections as the primary source of sepsis ($p < 0.001$). Non-survivors exhibited significantly higher APACHE II and SOFA scores upon admission compared to survivors (both $p < 0.001$). Additionally, serum lactate and procalcitonin levels were markedly elevated in deceased patients (both $p < 0.001$). Body composition analysis revealed that non-survivors had significantly lower SMA and SMI values, while their VAT/SAT ratios were significantly higher than those of survivors (all $p < 0.001$) (Table 1).

In gender-stratified comparisons among non-survivors, both male and female patients exhibited significantly lower SMI values and higher VAT/SAT ratios compared to survivors within their respective gender groups (all $p < 0.001$) (Table 2).

The optimal cut-off value for predicting in-hospital mortality was <32.6 for SMI and >2.15 for the VAT/SAT ratio (Table 3).

Both decreased SMI and elevated VAT/SAT values were found to be significant predictors of in-hospital mortality ($p < 0.001$ for both) (Table 4).

ROC analyses using the SMI demonstrated reduced discriminative performance for predicting in-hospital mortality across comorbidity subgroups. In patients with DM, hypertension, chronic obstructive pulmonary disease, stroke, cancer, and dementia, AUC values were 0.825, 0.809, 0.788, 0.808, 0.804, and 0.804, respectively, compared to 0.810, 0.830, 0.836, 0.815, 0.820, and 0.820 in those without these (Table 5) conditions (Table 2) (Figure 2 a,b,c,d,e,f).



Table 1. Comparison of demographic and clinical characteristics between survival and non-survival patients

| | Overall Patients (n=453) | Survivor (n=230) | Non-survivor (n=223) | p value |
|--|-----------------------------|---------------------|-------------------------|-------------------------------|
| Age (years), median (IQR) | 68 (67-69) | 67 (66-69) | 68 (67-69) | 0.052 ^b |
| Body mass index (kg/m2) median (IQR) | 21.9 (20.2-22.2) | 22.3 (21.1-25.6) | 22.1 (20.2-22.7) | 0.853 ^b |
| Gender n (%) | | | | |
| Female | 250 (55.2) | 130 (56.5) | 120 (53.8) | 0.562 ^c |
| Male | 203 (44.8) | 100 (43.5) | 103 (46.2) | |
| Comorbidities, n (%) | | | | |
| Diabetes mellitus | 320 (70.6) | 149 (64.8) | 171 (76.7) | 0.005 ^c |
| Hypertension | 304 (67.1) | 34 (58.3) | 170 (76.2) | <0.001 ^c |
| Chronic obstructive pulmonary disease | 184 (40.6) | 90 (39.1) | 94 (42.2) | 0.513 ^c |
| Stroke | 18 (4.0) | 7 (3.0) | 11 (4.9) | 0.303 ^c |
| Chronic liver disease | 26 (5.7) | 6 (2.6) | 20 (9.0) | 0.004 ^c |
| Malignancy | 40 (8.8) | 14 (6.1) | 26 (11.7) | 0.037 ^c |
| Dementia | 31 (6.8) | 15 (6.7) | 16 (7.0) | 0.923 ^c |
| Sites of Infection, n (%) | | | | |
| Respiratory | 167 (36.9) | 92 (40.0) | 75 (33.6) | <0.001 ^c |
| Urinary | 209 (46.1) | 120 (52.2) | 89 (39.9) | |
| Abdomen | 59 (13.0) | 14 (6.1) | 45 (20.2) | |
| Other | 18 (4.0) | 4 (1.7) | 14 (6.3) | |
| APACHE II score median (IQR) | 17 (11-18) | 11 (10-12) | 18 (17-19) | <0.001 ^b |
| SOFA score median (IQR) | 5 (4-7) | 4 (3-4) | 7 (7-8) | <0.001 ^b |
| Lactate level (mmol/L) median (IQR) | 1.55 (1.43-1.76) | 1.43 (1.33-1.48) | 1.77 (1.69-1.80) | <0.001 ^b |
| Procalcitonin level (ng/mL) median (IQR) | 1.0 (0.8-1.3) | 0.8 (0.8-0.9) | 1.3 (1.2-1.4) | <0.001 ^b |
| Skeletal Muscle Area median (cm²) (IQR) | 88 (73-92) | 88 (85-107) | 73 (71-92) | <0.001 ^b |
| Skeletal Muscle Index median (cm²) (IQR) | 32.7 (28.3-36.0) | 39.3 (31.3-52.4) | 34.5 (28.3-45.8) | <0.001 ^b |
| VAT/SAT median (IQR) | 2.20 (1.77-2.60) | 1.77 (1.24-1.89) | 2.56 (2.40-2.65) | <0.001 ^b |

APACHE II: Acute Physiology and Chronic Health Evaluation II score, SOFA: Sequential Organ Failure Assessment score, VAT/SAT: visceral-to-subcutaneous adipose tissue ratio

IQR: Interquartile Range, ^b Data are presented as median (25th–75th percentile); comparisons were made using the Mann–Whitney U test, ^c Data are presented as number (percentage); comparisons were made using the Chi-square test.

Table 2. Comparison of Skeletal Muscle Index and Visceral-to-Subcutaneous Adipose Tissue Ratio between survivors and non-survivors in female and male patients

| | Female | | p value | Male | | p value |
|---|------------------|------------------|-------------------------------|------------------|------------------|-------------------------------|
| | Survivor | Non-survivor | | Survivor | Non-survivor | |
| SMI (cm²/m²) | 37.1 (31.3-51.8) | 33.5 (28.3-48.9) | <0.001 ^b | 41.4 (31.9-52.4) | 35.8 (29.8-45.8) | <0.001 ^b |
| VAT/SAT | 1.38 (1.23-1.89) | 2.56 (2.41-2.65) | <0.001 ^b | 1.87 (1.36-1.89) | 2.60 (2.40-2.65) | <0.001 ^b |

SMI: Skeletal Muscle Index, VAT/SAT: visceral-to-subcutaneous adipose tissue ratio, ^b Data are presented as median (25th–75th percentile); comparisons were performed using the Mann–Whitney U test

Table 3. Prognostic Performance of Skeletal Muscle Area, Skeletal Muscle Index, and Visceral-to-Subcutaneous Adipose Tissue Ratio in Predicting in-hospital mortality

| | AUC 95% CI | p | Cut off | Sensitivity 95% CI | Specificity 95% CI | PPV | NPV |
|--------------|----------------------|--------|------------------|--------------------|--------------------|-------------------|-------------------|
| SMI (cm2/m2) | 0.818 0.795-1.000 | <0.001 | <36.65 | 0.98 0.95-0.99 | 0.97 0.94-0.98 | 0.97 0.95-0.98 | 0.98 0.96-0.99 |
| VAT/SAT | 0.968 0.949-0.988 | <0.001 | >2.15 | 1.00 0.98-1.00 | 0.93 0.88-0.95 | 0.93 0.90-0.95 | 1.00 0.99-1.00 |

SMI: Skeletal Muscle Index, VAT/SAT:visceral-to-subcutaneous adipose tissue ratio, AUC: Area Under the Curve, PPV: Positive Predictive Value, NPV: Negative Predictive Value

Table 4. Univariate Logistic regression analysis of predictors of in-hospital mortality

| Variable | Regression Coefficient (SE) | OR | 95 % CI | | p value |
|--|-----------------------------|----------|---------|----------|------------------|
| SMI (cm ² /m ²) | -0.331 | 0.718 | 0.672 | 0.768 | <0.001 |
| VAT/SAT | 9.220 (0.917) | 10094.07 | 1672.43 | 60923.43 | <0.001 |

SMI: Skeletal Muscle Index, VAT/SAT:visceral-to-subcutaneous adipose tissue ratio

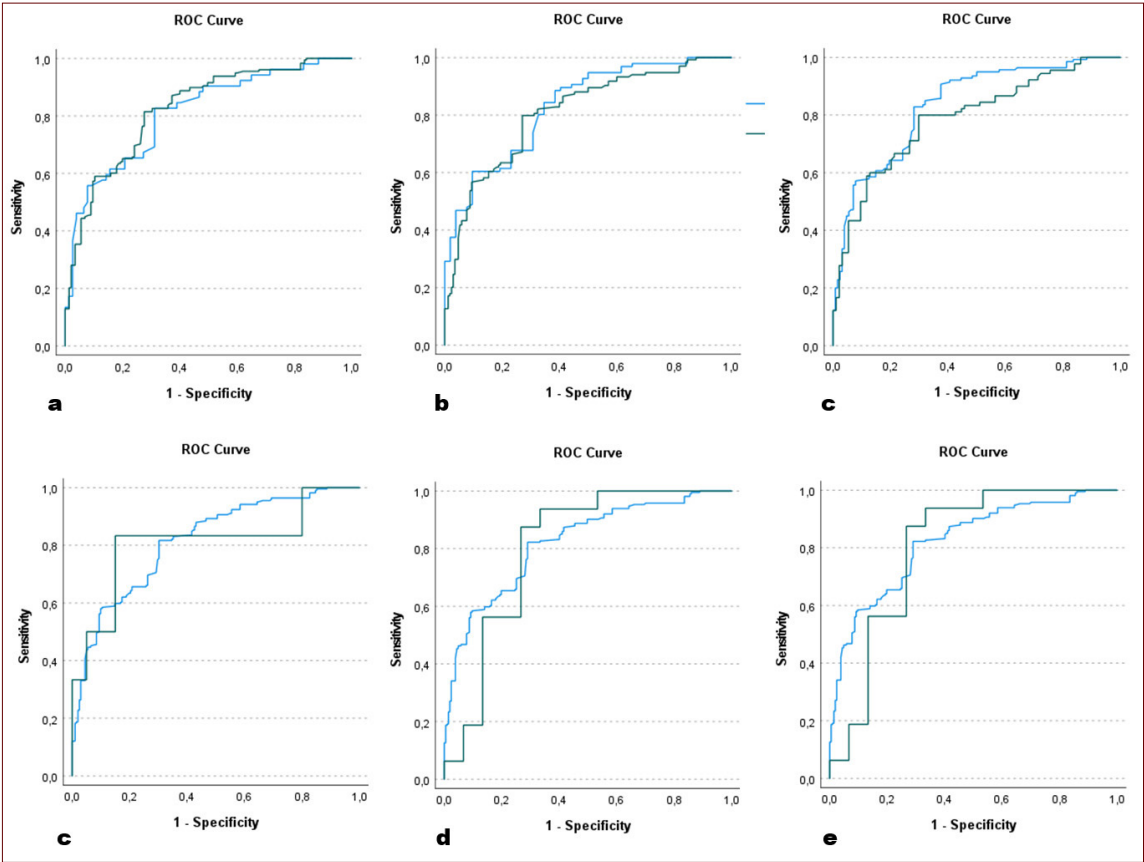


Figure 2. Comparative Receiver Operating Characteristic curves for Skeletal Muscle Index (SMI) by comorbidity status: diabetes mellitus (a), hypertension (b), chronic obstructive pulmonary disease (c), stroke (d), malignancy (e), and dementia (f).



Table 5. Discriminative performance of Skeletal Muscle Index for in-hospital mortality by comorbidity subgroups

| Comorbidity | AUC (Present) | AUC (Absent) |
|---------------------------------------|---------------|--------------|
| Diabetes mellitus | 0.825 | 0.81 |
| Hypertension | 0.809 | 0.83 |
| Chronic obstructive pulmonary disease | 0.788 | 0.836 |
| Stroke | 0.808 | 0.815 |
| Malignancy | 0.804 | 0.82 |
| Dementia | 0.804 | 0.82 |

Multivariate logistic regression analysis was conducted to evaluate the independent association between comorbidities and in-hospital mortality. The presence of malignancy was found to be the strongest independent predictor of mortality (OR = 1.96, $p = 0.014$), followed by DM (OR = 1.48, $p = 0.029$).

DISCUSSION

Identifying prognostic parameters that influence mortality and incorporating them into clinical decision-making enables timely and targeted interventions, thereby improving patient outcomes.

Although current literature offers limited insight into the prognostic significance of sarcopenia and visceral obesity in elderly patients with sepsis, this study is noteworthy as the first to concurrently evaluate both parameters in the ED setting. The study also establishes threshold values for SMI and the VAT/SAT ratio associated with an increased risk of in-hospital mortality.

There is currently no universally accepted cut-off value for defining low muscle mass or sarcopenia. Van der Werf et al. proposed gender-specific thresholds for the skeletal muscle index at the L3-SMI based on a healthy Caucasian population, defining sarcopenia as $<41.6 \text{ cm}^2/\text{m}^2$ in men and $<32.0 \text{ cm}^2/\text{m}^2$ in women (13). Another study conducted in a healthy Korean liver donor cohort also established gender specific L3-SMI

reference values. When defined using the standard deviation method, the sarcopenia thresholds were $39.33 \text{ cm}^2/\text{m}^2$ for men and $27.77 \text{ cm}^2/\text{m}^2$ for women; when using the fifth percentile approach, the corresponding values were $40.96 \text{ cm}^2/\text{m}^2$ and $30.60 \text{ cm}^2/\text{m}^2$, respectively (14). In contrast, a study involving hospitalised internal medicine patients adopted higher thresholds, identifying sarcopenia as an L3-SMI $<53 \text{ cm}^2/\text{m}^2$ in men and $<41 \text{ cm}^2/\text{m}^2$ in women (15). These discrepancies likely reflect underlying racial and ethnic differences in muscle mass and quality. Genetic factors may contribute to the greater muscle mass typically observed in Black individuals, while Asian populations generally have lower muscle mass, which may influence population-specific sarcopenia thresholds (16,17).

The association between sarcopenia and increased mortality in septic patients is multifactorial and remains incompletely understood. One contributing factor is the frequent presence of poor nutritional status among sarcopenic individuals, which predisposes them to sepsis and impairs recovery. Sarcopenia is also closely associated with immunosenescence, the age-related decline in immune function, which compromises host defences against infection. This immunological deterioration is particularly consequential in the context of sepsis, where an effective immune response is vital for pathogen clearance (18). Additionally, the ageing immune system fosters a pro-inflammatory state that accelerates muscle catabolism, potentially exacerbated by acute

physiological stressors such as infection. Sepsis-induced systemic inflammation further promotes muscle wasting through activation of proteolytic pathways, including the ubiquitin–proteasome and autophagy–lysosome systems. These pathways upregulate genes associated with muscle atrophy, thereby perpetuating the loss of muscle mass. This progressive muscular degeneration may impair essential physiological functions, such as swallowing and respiratory mechanics, predisposing patients to complications like aspiration pneumonia, ultimately contributing to increased mortality risk (18).

Sarcopenia may impair recovery in septic patients due to diminished skeletal muscle mass, which reduces the availability of amino acids required for acute-phase protein synthesis and immune cell activation (19). Furthermore, sarcopenic individuals often exhibit compromised antioxidant capacity, resulting in decreased clearance of reactive oxygen species. This oxidative imbalance can exacerbate tissue injury and further hinder recovery from sepsis (19).

The risk of developing sarcopenia is elevated in individuals with multiple coexisting chronic diseases. Chronic conditions such as malignancy and DM contribute to sarcopenia through catabolic stress, inflammation, and reduced mobility. In this study, subgroup ROC analyses showed that comorbidities diminished the discriminative ability of SMI for predicting in-hospital mortality. This suggests that overlapping pathophysiological mechanisms may reduce the prognostic utility of sarcopenia indices. Supporting this, multivariate regression analysis confirmed that comorbidities were independently associated with increased mortality, highlighting the need for comorbidity-adjusted risk models in sepsis care.

Additionally, a high VAT/SAT ratio has been associated with increased mortality in patients with sepsis. This pattern of fat distribution reflects greater metabolic activity and systemic inflammation, which

may negatively influence sepsis outcomes (12,20). In the current study, a VAT/SAT ratio exceeding 2.15 was identified as the optimal threshold for predicting in-hospital mortality among elderly patients with sepsis.

VAT, located within the abdominal cavity and surrounding internal organs, exhibits high endocrine and immunological activity. It functions as a prolific source of pro-inflammatory mediators such as tumour necrosis factor- α and plasminogen activator inhibitor-1, both of which are elevated in septic states and have demonstrated associations with increased disease severity and mortality (21,22). In contrast, SAT, located beneath the dermis, is relatively metabolically inert and secretes lower levels of inflammatory cytokines. This difference may contribute to its proposed protective role in systemic inflammatory conditions such as sepsis (12). Moreover, during sepsis, VAT undergoes pronounced immunological activation characterised by increased macrophage infiltration and upregulation of pro-inflammatory cytokines. These changes potentiate the systemic inflammatory response, in contrast to SAT, which remains comparatively immunologically quiescent (23). Thus, an elevated VAT/SAT ratio may serve as an indicator of an amplified inflammatory milieu, offering a plausible mechanistic explanation for its association with poorer clinical outcomes in septic patients.

An elevated VAT/SAT ratio has been correlated with increased circulating levels of inflammatory biomarkers such as C-reactive protein and leptin, both of which are implicated in adverse clinical outcomes in sepsis (24).

Conversely, a lower VAT/SAT ratio has been associated with more favourable prognostic indicators, including the relative preservation of low-density lipoprotein (LDL) levels during the septic response, a factor linked to improved survival outcomes. Notably, the prognostic utility of the VAT/SAT ratio in sepsis appears to be modulated by lipid



metabolism, particularly LDL dynamics. While a low VAT/SAT ratio may confer a survival advantage via preserved LDL concentrations, this benefit can be attenuated by statin therapy, which suppresses endogenous LDL synthesis. In contrast, individuals carrying a proprotein convertase subtilisin/kexin type 9 loss-of-function genotype, which facilitates increased LDL clearance, demonstrate enhanced survival in the context of low VAT/SAT ratios (20).

These findings suggest a complex interplay between adipose tissue distribution, lipid homeostasis, and immune response regulation in pathophysiology of sepsis.

A key strength of the present study is its relatively large sample size, which enhances the statistical power and reliability of the observed associations.

Limitations

This study has several limitations that warrant consideration. First, its retrospective design and single-centre setting may limit the generalisability of the findings. As data were extracted from existing medical records, variability in documentation and potential missing data could have introduced bias. Second, inclusion was limited to patients who underwent abdominal CT imaging, which may have led to selection bias, as these individuals could differ systematically from those who did not undergo imaging. Third, due to the retrospective nature of the study, direct assessments of physical performance and muscle strength could not be performed, restricting the evaluation to imaging-based parameters alone. Fourth, while sarcopenia is intrinsically linked to nutritional status, our study did not include validated nutritional risk assessment tools such as the Nutritional Risk Screening 2002 and the Nutrition Risk in the Critically Ill score due to the retrospective nature of the data. These tools typically require real-time clinical evaluation or comprehensive dietary intake information, which were not systematically recorded in our dataset. This represents a methodological limitation that

may have influenced the full characterization of the patients' nutritional risk profiles.

CONCLUSION

To our knowledge, this is the first study to concurrently evaluate the prognostic significance of sarcopenia and visceral obesity, as measured by CT-derived SMI and VAT/SAT ratio, in elderly patients presenting with sepsis to the ED. Our findings demonstrate that both reduced muscle mass and increased visceral fat distribution are independently associated with in-hospital mortality. Given the routine use of abdominal CT imaging in emergency settings, these parameters offer practical and objective tools for early risk stratification. In addition, logistic regression analysis showed that DM and malignancy were independent predictors of in-hospital mortality, further reinforcing the need to integrate comorbidity assessment into prognostic models. Moreover, identifying sarcopenia and visceral obesity as modifiable risk factors supports the rationale for targeted interventions, such as nutritional support and inflammation-reducing strategies, to improve outcomes in this high-risk population. Future prospective multicentre studies are warranted to validate these cut-off values and explore the integration of body composition metrics with inflammatory and metabolic biomarkers to inform personalised management approaches in geriatric sepsis care.

Statement of ethics: This study was approved by the Ankara Etlik City Hospital, Ankara, Turkey (approval no. 2025-0195).

Conflict of interest statement: The authors declare no competing interests.

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