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Article

Sarcopenic Obesity in Older Adults: A More Dependent, Frail, and Even Fatal Condition than Sarcopenia or Obesity Alone. A Retrospective Cross-Sectional Study

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Abstract: Background: Sarcopenia, characterized by age-related declines in muscle mass and strength, and obesity, marked by excessive body fat accumulation, often manifest concurrently, leading to a new entity known as sarcopenic obesity (SO). Although there are many studies on SO in older adults, the number of studies with new definition criteria is limited. **Methods:** We conducted a cross-sectional retrospective study including 364 patients aged 65 and older who underwent Bioelectrical Impedance Analysis (BIA) to assess body composition. We applied geriatric assessments (Katz Index of Activities of Daily Living, Lawton Instrumental Activities of Daily Living Scale), mini nutritional assessment, geriatric depression scale, and mental status examination). SO was defined using ESPEN (European Society for Clinical Nutrition and Metabolism) and EASO (European Association for the Study of Obesity) criteria, and frailty was graded with the clinical frailty score. Mortality data were obtained. We analyzed the associations of SO with geriatric tests, frailty, and mortality using univariate and multivariate analyses. **Results:** The mean age of the participants was 77.11 years (SD: 6.97). The prevalence rates for the groups were as follows: 39.6% classified as normal, 16.5% as obese (O), 19.5% as sarcopenic (S), and 24.5% as sarcopenic obese (SO). Patients in the SO group demonstrated significantly lower scores in functional and cognitive assessments, including ADL, IADL, MMSE, and MNA (p-values: 0.002, <0.001, <0.001, and <0.001, respectively). Additionally, this group exhibited reduced handgrip strength and elevated mortality rates (p = 0.002). SO patients showed the highest rates of cognitive impairment, S patients had the most elevated depression scores, and O patients displayed the slowest walking speeds. Both hypertension ($\beta = 0.396$, p = 0.001) and diabetes mellitus ($\beta = 3.074$, p < 0.001) were identified as significant risk factors for SO, with diabetes increasing the risk approximately threefold. **Conclusion:** SO exhibited greater physical dependence, mortality, and frailty. The S group showed a higher tendency toward depression. Significant risk factors for SO included poor nutrition, cognitive decline, low muscle strength, hypertension, and diabetes.

Keywords: sarcopenic obesity definition; espen- easo criteria; functionality; frailty; mortality

1. Introduction

Sarcopenia, characterized by the age-related decline in muscle mass and strength, and obesity, marked by the excessive accumulation of body fat, are two distinct medical conditions that often manifest concurrently, leading to the emergence of a novel entity known as sarcopenic obesity (SO) [1,2]. The relationship between sarcopenia and obesity is particularly concerning, as the two conditions can exacerbate each other and lead to even more severe functional impairments. SO has been linked to an increased risk of physical disability, metabolic disorders, cardiovascular disease,

and mortality in older people [1,3,4]. Studies suggest that SO affects functionality more than sarcopenia or obesity alone. Many different definitions of SO have been proposed in the literature. This leads to significant differences in reported prevalence and associations with functional problems. However, after the SO definition proposal was made in 2022, studies focusing on the difference between SO according to its definition components remain limited in studies conducted following these criteria [2]. SO is a complex condition that requires tailored interventions to address the dual challenges of preserving muscle mass while reducing the health risks associated with excess body fat. Targeted interventions, including the incorporation of functional foods and sustainable physical activity, have been identified as potential strategies to mitigate the burden of sarcopenia and obesity. As the global population ages and the prevalence of these conditions rises, a comprehensive understanding of their multifactorial causes and the development of effective, multidisciplinary approaches to prevention and management will be crucial in addressing this growing public health challenge.

The causes of these complex conditions are multifactorial, involving a combination of lifestyle, genetic, and environmental factors. Factors such as disuse, changing endocrine function, chronic diseases, inflammation, insulin resistance, and nutritional deficiencies have all been identified as potential contributors to the development of sarcopenia. Similarly, poor diet, sedentary lifestyle, and aging have been linked to the rising prevalence of obesity, with the coexistence of sarcopenia and obesity, known as SO, presenting a particularly concerning challenge [1,3,5,6].

Previous studies have highlighted SO's impact on activities of daily living, mortality, and frailty in older adults. However, research comparing SO with sarcopenia and obesity alone, using the 2022 criteria, is sparse. The study aims to examine the associations of sarcopenic obesity (SO) with physical dependence, mortality, frailty, and cognitive status in older adults and to demonstrate that SO has more significant negative effects on older adults' functionality than sarcopenia or obesity alone, underscoring its importance as a public health concern in an aging population.

2. Materials and Methods

2.1. Study Design and Patient Selection

In this cross-sectional retrospective study, 364 patients with appropriate files and data were included among 476 patients aged 65 and over who underwent Bioelectrical Impedance Analysis (BIA) in two different hospitals between July 2015 and September 2023. Those who gave informed consent for the tests underwent a comprehensive geriatric assessment, and those with complete data and cognitive and other functional measurements were included in the study. Patients whose data were incomplete in the file (those not suitable for BIA, laboratory values were missing, comprehensive geriatric assessment tests could not be performed or were missing) were excluded from the study. The flow chart (Figure 1) shows the exclusion criteria and details of patient selection.

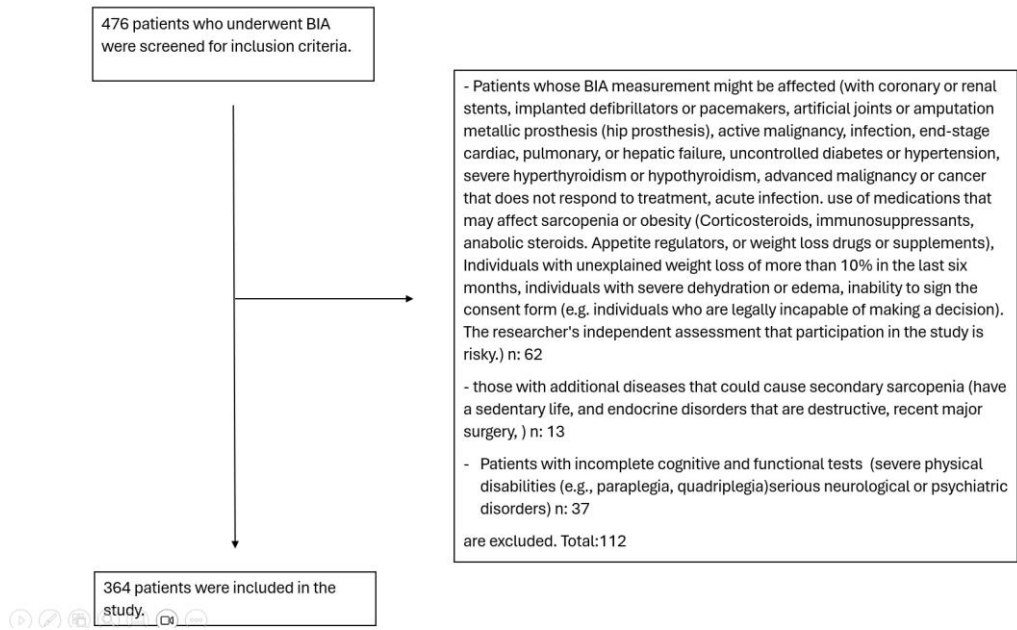


Figure 1. Patient selection flow chart.

Patients were divided into 4 groups following the definition of SO; Group 1: normal; Group 2: Obese only group (O); Group 3: Sarcopenic only group (S); Group 4: Sarcopenic Obesity group (SO)

2.2. Comprehensive Geriatric Assessment

Geriatric assessments included the Katz Activities of Daily Living Index (ADL), Lawton Instrumental Activities of Daily Living Scale (IADL), Mini-Nutritional Assessment Short-Form (MNA-SF), Geriatric Depression Scale (GDS) and Mini-Mental Status Examination (MMSE). See details in Table 1.

Table 1. Comprehensive Overview of Geriatric Assessments, Biochemical Parameters, and Mortality Detection Methods Used in the Study.

COMPREHENSIVE GERIATRIC ASSESSMENT TESTS			
Test	Measurement Purpose	Interpretation	Reference
Katz	Activities of Daily Living Index (ADL)	The functions of dressing, bathing, going to the toilet, getting out of bed, eating, and continence are over six points. A total score means complete independence. A decrease in score suggests a decrease in functionality.	[7]
Lawton- Brody	Instrumental Activities of Daily Living Scale (IADL)	Activities such as telephone use, shopping, food preparation, household chores, laundry, urban transportation, and proper use	[8]

		of drugs are evaluated at over eight points. A full score means complete independence. A decrease in score suggests a decrease in functionality.	
Mini-Nutritional Assessment Short-Form (MNA-SF)	Malnutrition Screening	In the short screening form with 14 points; 0-7 points means malnutrition, 8-11 points means malnutrition risk and 12-14 points means normal nutrition.	[9]
Geriatric Depression Scale (GDS)	Depression Screening	A score of 5 or above on the 15-item short form of the geriatric depression scale is considered consistent with a depressive mood.	[10]
Mini-Mental Status Examination (MMSE)	Cognitive Screening	Low scores on this test, which is evaluated over 30 points, indicate impairment in cognitive functions	[11]
Hand grip strength	Muscle Strength Screening A component of the diagnosis of sarcopenia	Hand grip strength is measured by an electronic hand dynamometer (GRIP-D, influenza strength dynamometer produced by Takei, made in Japan). The unit of results is kilograms, with <22 kg for women and <32 kg for men indicating reduced muscle strength	[12]
Gait Speed over a 4-meter	Muscle performance A component of the diagnosis of sarcopenia	After walking time was measured with an electronic stopwatch, the walking speed was calculated with the formula 4 meter/walking time (seconds) in m/s., with ≤ 0.8 m/s indicating decreased performance	[12]
Clinical Frailty Score	Frailty Screening	Clinical frailty scores were used to assess frailty. In this	[13]

		<p>scoring, high values are associated with frailty. There are nine categories: 1: Very fit-robust, active, energetic, well-motivated, and fit; these people commonly exercise regularly and are in the fittest group for their age. 2: Fit-without active disease, but less fit than people in category 1. 3: Well, with treated comorbid disease- disease symptoms are well controlled compared with those in category 4. 4: Vulnerable although not frankly dependent, these people commonly complain of being “slowed up” or having disease symptoms. 5: Mildly frail- with limited dependence on others for instrumental activities of daily living. 6: Moderately frail- help is needed with instrumental and non-instrumental daily living activities. 7: Severely frail- completely dependent on others for activities of daily living, but not at high risk of dying within six months. 8: Very severely frail- completely dependent on others for activities of daily living and approaching end of life. 9: Terminally ill- approaching end of life with life expectancy</p>	
<p>BIA (bioelectrical impedance analysis)</p>	<p>Muscle Mass A component of the diagnosis of sarcopenia</p>	<p>Portable BIA analyzer in the supine position. Quadscan 4000 (Bodystat, Douglas, Isle of Man, UK) obtained the BIA resistance in ohms (Ω). The device was set to the</p>	<p>[14]</p>

		participant's age, gender, height, and body weight. Skeletal muscle mass (SMM) was calculated.	
BIOCHEMICAL PARAMETERS			
Laboratory Values	(Unit-Normal Range)	Method	
Fasting blood glucose	(mg/dL 74-100)	Enzymatic Methods	.
Calculated Glomerular Filtration Rate	(mL/ min/1.73 m ² >60)	Calculated From Serum Creatinine Levels	
Calcium	(mg/dL 8.8-10.6)	Spectrophotometric	The spectrophotometric method measures the amount of light absorbed by a substance at specific wavelengths. It is widely used in biochemistry and chemistry to analyze concentrations and monitor reactions
Total protein	(g)/L 66-83)	Spectrophotometric	
Albumin	(g/L 35-52)	Spectrophotometric	
Leukocyte (white blood cell)	(x10 ⁹ /L 4.5-11)	counted using hematology analyzers	
Hemoglobin	(g/dL 11.7-16.1)	cyanmethemoglobin method	
Vitamin B12	(pg/mL 126.5-505),	Spectrophotometric	
Thyroid-stimulating hormone	(μIU/mL 0.38-5.33)	ECLIA method	ECLIA (Electrochemiluminescence Immunoassay) is a sensitive immunoassay method that uses electrochemiluminescence to measure the concentration of specific analytes, such as hormones and proteins, in clinical laboratories.
C-reactive protein (CRP)	(mg/L 0.0-5.0)	Turbidimetric	Turbidimetry is an analytical technique for determining the concentration of suspended particles in a solution by measuring the amount of light scattered by these particles.

25-hydroxy vitamin D	(µg/L 10-60)	HPLC method	HPLC (High-Performance Liquid Chromatography) is an analytical technique for separating, identifying, and quantifying components in a mixture by passing a liquid sample through a column packed with solid adsorbent material.
MORTALITY DETECTION			
“TC Turkey Ministry of Health Public Health Agency of Death Reporting System”			[15]

2.3. SO Definition

We defined SO and its components according to ESPEN and EASO's definition and diagnostic criteria [2]. We used BMI and waist circumference to screen for obesity and handgrip strength to screen for sarcopenia. For details on BIA use, see Table 1.

2.4. Frailty Definition and Mortality

We used the clinical frailty score to define and grade frailty. We determined mortality using the 'Public Health Institution Death Notification System'. We identified those who died at least one year after their application as mortality. We determined the mortality status as a percentage on a case-by-case basis.

2.5. Laboratory Values

Biochemical laboratory results presented and compared in Table 2 were utilized. Refer to Table 1 for measurement methods and comments.

Table 2. Comparative Analysis of Demographic Data, Comprehensive Geriatric Assessments, and Laboratory Parameters Across Study Groups.

P*	All	SO	S	O	Normal	Parameter
	364(100)	89(24.5)	71(19.5)	60(16.5)	144(39.6)	n (%)
<0.001	77.11±6.97	79.40 ± 7.15 ^a	75.86 ± 5.54	75.18 ± 6.80	75.28 ± 7.13 ^d	Age
<0.001	235(64.6)	74(20.3)	54(14.8)	28(8.0)	78(21.4)	Female n (%)
Comprehensive Geriatric Assessment						

<0.001	24(0-30)(7,50)	17(0-30)(14.50) ^a	21(0-30)(11)	20(0-30)(8.75)	23(0-30)(6) ^d	MMSE
<0.001	187(51.4) 177(48.6)	30(8.2) 59(16.2)	37(10.2) 34(9.3)	27 (7.4) 33(9.1)	2(25.5) 51(14.0)	MMSE group n(%) (24-30; normal cognition) (<24; poor cognition)
.002	6 (0-6)(0)	3 (0-6)(3) ^a	4 (0-6)(2)	4 (0-6)(3)	5(0-6)(0) ^d	Katz ADL
<0.001	7 (0-8)(2)	4 (0-8)(6) ^a	5 (0-8)(3)	5 (0-8)(4)	6 (0-8)(8) ^d	Lawton-Brody IADL
<0.001	12(2-14)(1)	9 (2-14)(4) ^{abc}	12 (3-14)(3) ^{ad}	12 (3-14)(2) ^d	12(7-14)(1.8) ^{cd}	MNA-SF
.010	3(0-15)(6)	4(0-15)(6,25)	3(0-15)(8) ^a	5(0-15)(12)	6(0-15)(4) ^c	GDS
<0.001	4.54 ± 1.58	4.98 ± 1.50 ^a	4.91 ± 1.47 ^a	4.65 ± 1.65	4.04 ± 1.51 ^{cd}	Clinical Frailty Score
.004	0.57(0-2.25)(,36)	0.50(0-1.29)(,37)	0.57(0-2.25)(,32)	0.47(0-1.05)(,39) ^a	0.57(0-1.60)(,4) ^b	4 m Walking Speed (m/sn)
<0.001	16.5(0-43.1)(7,3)	12.6(0-26.5)(9,2) ^{abc}	16.5(5.5-34)(11,3) ^{ad}	16.45(5-38.1)(10,1) ^{ad}	18.3(5.9-43.1)(9,4) ^{bc} ^d	Handgrip strength (kg)
.002	87(23.9)	34(9.3)	12(3.3)	15(4.1)	26(7.1)	Mortality n(%)
Comorbidities						
.001	250(68.7)	47(12.9)	48(13.2)	44(12.1)	111(30.5)	HT (n, %)
<0.001	134(36.8)	15(4.1)	31(8.5)	24(6.6)	64(17.6)	DM (n, %)
.078	67(18.5)	16(4.4)	10(2.8)	18(5)	23(6.3)	HF (n, %)
.371	77(21.9)	13(3.7)	17(4.8)	16(4.5)	31(8.8)	Hypothyroidism (n, %)
.326	36(10.2)	9(2.6)	8(2.3)	9(2.6)	10(2.8)	Cerebrovascular Diseases (n, %)

Laboratory Values						
.159	98(51-442)(5,21)	95(53-196)(16)	102(69-442)(13)	90(52-293)(21)	105(57-381)(43)	Fasting blood glucose (mg/dL)
.174	0.86(0.17-3.45)(,28)	0.78(0-5,3)(,54)	0.91(0.42-1.9)(,33)	0.84(0.59-2.45)(,26)	0.81(0.5-2.86)(,27)	Creatinine (mg/dL)
.218	69(11-90)(26)	59(24-90)(31,50)	67(25-90)(27,75)	69(11-90)(22)	73(15-90)(21,25)	Calculated glomerular filtration rate (mL/min/1.73 m ²)
.108	9.5(8.20-11.7)(,50)	9.6(8.2-11.7)(,50)	9.1(8.3-10.9)(,90)	9.6(8.7-10.8)(,30)	9.6(8.2-11.6)(,60)	Calcium (mg/dL)
.310	7.10(5.20-8.20)(,70)	7(6.10-8.10)(,56)	6.9(5.40-7.84)(,60)	7.3 (6.2-8.1)(,55)	7.2(5.3-8.3)(,70)	Total protein (g)/L
.232	4(2-4.90)(,40)	3.80(2.2-4.9)(,51)	4.05(2.3-4.8)(,80)	3.95(2-4.7)(,50)	4.1(2.4-4.8)(,30)	Albumin (g/L)
.883	13(2-88)(15,25)	13(5-87)(11,5)	13(5-88)(9)	14(3-49)(20)	13(2-87)(16,75)	Sedimentation Rate (mm/hour)
.063	6.78(2.62-35.63)(2,54)	6.13(2.8-12.47)(2,21)	6.75(2.62-11.77)(3,84)	7.04(3.18-39.63)(2,12)	6.8(2.63-34)(2,76)	Leukocyte (WBC) (×10 ⁹ /L)
.128	12.41±1.88	12.31±1.6	12.17 ±1.83	12.161.95	12.7±2.02	Hemoglobin (Hb) (g/dL)
.492	6.60(5.10-14.90)(1,70)	6.60(5.6-10.49)(2,40)	6.60(5.10-9.20)(1,55)	5.95(5.20-8.60)(1,40)	6.8(5.5-14.9)(2,18)	HbA1C (%)
.224	342(50-1500)(312,25)	329(50-1500)(306,25)	397(102-1500)(643)	372(169-1111)(230)	317.5(77-2000)(195,5)	Vitamin B12 (pg/mL)
.218	1.49(0.02-30.59)(1,73)	1.36(0.02-6.76)(2,35)	1.16(0.02-21.58)(3,14)	1.47(0.02-30.59)(,71)	1.80(0.02-7.15)(2,30)	Thyroid-stimulating hormone (TSH) (μIU/mL)
.945	4.70(.10-147.9)(5,21)	19.35(10-100)(5,73)	5.8(0.10-105)(9,45)	4.5(0.80-139.90)(3,30)	4.1(0.2-147.9)(5,48)	CRP(mg/L)

.200	17.75(4.5-299.0)(20,2)	14.3(4.5-58.1)(20,4)	19.0(4.9-47.4)(17,9)	19.5(5.2-125.7)(18.8)	19.35(5-299)(16)	25-hydroxy vitamin D (µg/L)
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Details of all tests used in the evaluation are presented in Table 1.

2.6. Statistical Analysis

The sample size was calculated based on an SO prevalence of approximately 34%, requiring at least 102 patients for a power of 95% and alpha of 0.05. Statistical analysis was performed using SPSS 26. The normality of variables was assessed through visual (histograms and probability graphs) and analytical methods (Kolmogorov-Smirnov/Shapiro-Wilk tests). Descriptive statistics included mean and standard deviation for normally distributed variables and median with maximum-minimum values for non-normally distributed variables. Frequencies of categorical variables were expressed as percentages. Chi-square and ANOVA tests were used for group comparisons, followed by Bonferroni post hoc tests. Variables found significant in the ANOVA analysis were further analyzed using regression analyses. Multicollinearity among independent variables was checked using Pearson, Spearman, or Kendall's tau-b correlation analyses, and variables with multicollinearity were not included in the same regression models. Logistic regression results for SO were reported as odds ratios (OR) with a 95% confidence interval (CI). A p-value of less than 0.05 was considered significant.

3. Results

The mean age of 364 patients was 77.11±6.97 years. 235 (64.6%) were women. We determined the prevalence of the groups as follows: normal (non-obese, non-sarcopenic) 39.6% (n:144), obese 16.5% (n:60), sarcopenic 19.5% (n:71) and sarcopenic obese 24.5% (n:71). The mean age in the SO group was significantly higher than in the other groups. Katz ADL, Lawton-Brody IADL, MMSE, MNA scores, and handgrip strength were significantly lower in the SO group. The percentage of the MMSE group associated with poor cognition was highest in SO patients. We found that the mortality rate was highest in the SO group. GDS scores averages were found to be higher in the S group than in the normal group, consistent with depression. Walking speed was the worst in the O group. When the clinical frailty score means were compared, we found that the highest mean, considered the most frail, was in the SO group. In subgroup analyses affecting the clinical frailty score, S and SO status have an impact. The difference between the O group is not significant. Other demographic data, comprehensive geriatric assessment, and laboratory data comparisons between the groups are shown in detail in Table 2.

We have summarized the study's main findings below with tables and Figures 2–4.

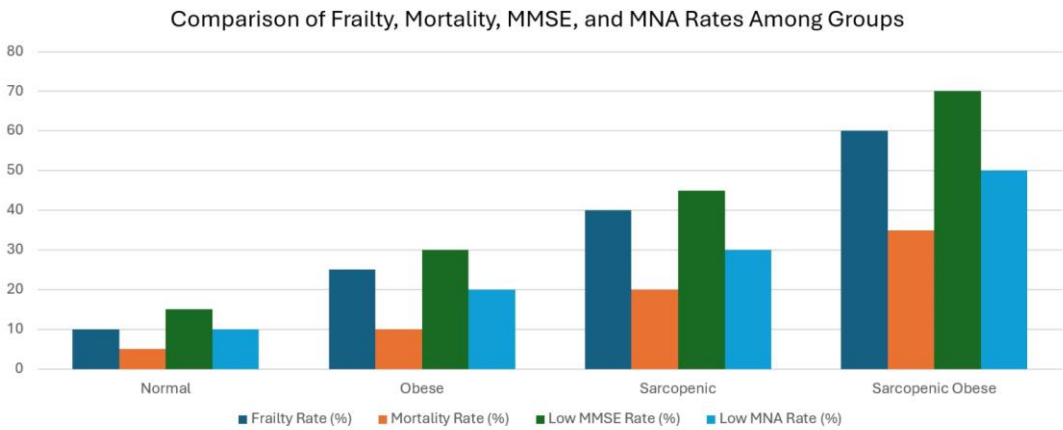


Figure 2. Comparison of Frailty, Mortality, Cognitive Impairment, and Nutritional Deficiency Rates Across Groups.

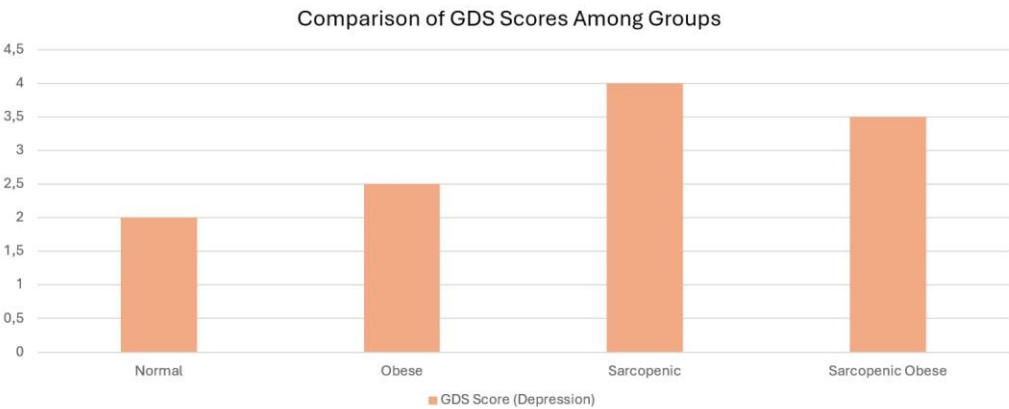


Figure 3. Comparison of GDS Scores Among Groups Reflecting Depression Levels. This bar graph illustrates the Geriatric Depression Scale (GDS) scores across four groups: Normal, Obese, Sarcopenic, and Sarcopenic Obese. The Sarcopenic group exhibits the highest GDS scores, indicating the most severe depressive symptoms among the groups. The Sarcopenic Obese group also shows elevated scores, while the Normal group has the lowest depression levels, reflecting better mental health outcomes. These results highlight the association between sarcopenia and increased depression risk in older adults.

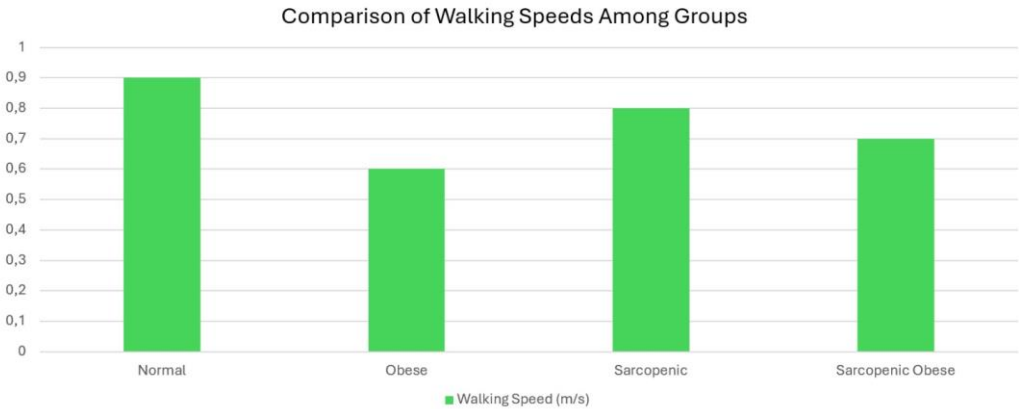


Figure 4. Comparison of Walking Speeds Among Groups. This bar graph compares the walking speeds (measured in meters per second) among the Normal, Obese, Sarcopenic, and Sarcopenic Obese groups. The Normal group demonstrated the fastest walking speed, while the Obese group exhibited the slowest. These findings highlight the significant impact of obesity on physical mobility, whereas sarcopenia and sarcopenic obesity also result in reduced walking speeds compared to the Normal group.

This bar graph compares the frailty rate, mortality rate, and prevalence of low MMSE and MNA scores across different groups: Normal, Obese, Sarcopenic, and Sarcopenic Obese. The Sarcopenic Obese group exhibits the highest rates of frailty, mortality, and low cognitive (MMSE) and nutritional (MNA) scores, indicating a more severe overall health decline. In contrast, the Normal group consistently shows the lowest rates across all measures, reflecting better functional and health outcomes. These results highlight the compounded impact of sarcopenic obesity on geriatric health.

Logistic regression analysis was conducted to identify factors influencing sarcopenic obesity (SO). Variables with significant differences between groups, as presented in Table 2, were included in the initial analysis. These significant variables were first analyzed in an unadjusted model, followed by adjustments for potential confounders such as age and gender to ensure robust and clinically meaningful results. When the significant data were adjusted for age and gender, the statistically significant data obtained were summarized in Figure 5. Low MNA, MMSE, Lawton-Brody scores, and low handgrip strength are likely related to SO. In addition, hypertension and diabetes also increase the risk of SO. We observed that diabetes mellitus increased the likelihood of SO by 3 times.

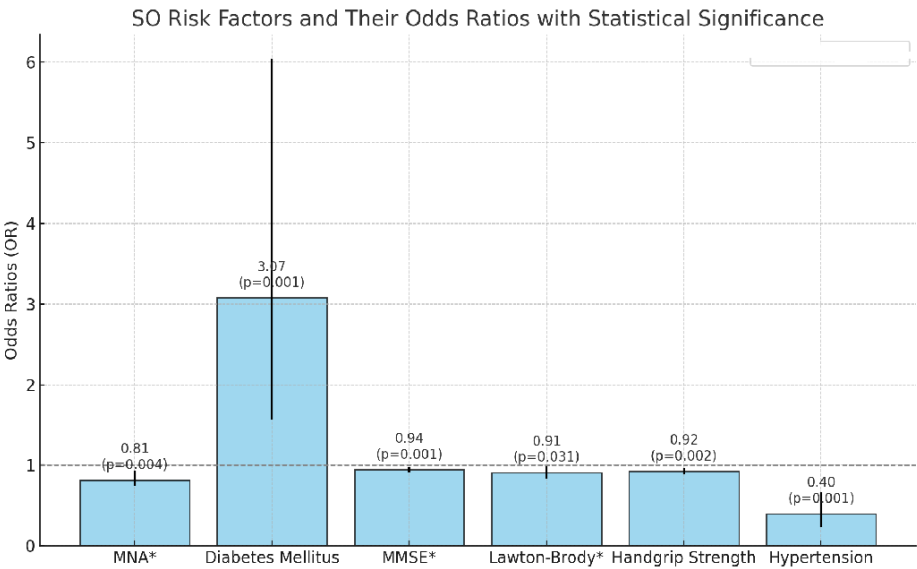


Figure 5. Odds Ratios of Risk Factors Associated with Sarcopenic Obesity (SO). This bar graph presents the odds ratios (OR) of key risk factors associated with sarcopenic obesity (SO) and their statistical significance. Diabetes mellitus demonstrates the highest odds ratio (OR = 3.37, $p = 0.001$), indicating a strong association with SO. Other significant factors include low MNA (OR = 0.81, $p = 0.004$), low MMSE (OR = 0.94, $p = 0.001$), low Lawton-Brody IADL (OR = 0.91, $p = 0.031$), reduced handgrip strength (OR = 0.92, $p = 0.002$), and hypertension (OR = 0.40, $p = 0.001$). These findings highlight the multifactorial nature of SO, emphasizing the importance of addressing cognitive and physical health parameters and managing chronic conditions like diabetes and hypertension.

4. Discussion

Our study demonstrated that patients with sarcopenic obesity (SO) exhibited significant impairments in key functional and cognitive measures, including ADL, IADL, MMSE, MNA, and handgrip strength. Frailty and mortality rates were notably higher in the SO group compared to other groups, highlighting the compounded burden of sarcopenia and obesity. These findings underscore that the coexistence of sarcopenia and obesity leads to greater physical dependence and cognitive decline than either condition alone. Sarcopenia appears to be more strongly associated with depression, whereas obesity primarily affects walking speed. Factors associated with an increased likelihood of SO include poor nutritional and cognitive status, low muscle strength, hypertension, and diabetes mellitus. The prevalence of SO in older adults varies across studies and regions. While some studies report a prevalence of up to 20% [2,16,17], our study found a rate of 24.5%. This higher prevalence may be attributed to age-related changes, such as decreased muscle mass and increased body fat, which deteriorate physical function and quality of life. These results align with existing literature, emphasizing the pressing need for targeted interventions.

4.1. Functional Limitations in Sarcopenic Obesity

Previous studies have demonstrated that SO significantly impairs older adults' ability to perform basic and instrumental daily living activities [4,5,18]. This is primarily due to reduced muscle strength, which makes bathing, dressing, and getting out of bed challenging. Balance and coordination disorders further increase the risk of falls, creating a vicious cycle of inactivity and worsening functional decline. Our findings corroborate these studies, as we observed the most pronounced decrease in handgrip strength in the SO group compared to the S group. This suggests that the combined effects of sarcopenia and obesity have a compounding negative impact on muscle strength and function.

4.2. Cognitive Decline in Sarcopenic Obesity

Growing evidence links SO to cognitive decline in older adults [19,20]. Chronic low-grade inflammation, insulin resistance, and hormonal imbalances are potential mechanisms contributing to this association [19–21]. SO may also impair cardiovascular health, reducing blood flow and oxygenation to the brain. Consistent with prior research, our study found that SO was associated with greater cognitive decline than sarcopenia or obesity alone. These findings highlight the need for future studies to establish causality and develop preventive strategies for cognitive health in older adults with SO.

4.3. Nutritional Implications of Sarcopenic Obesity

The relationship between SO and malnutrition is bidirectional, with shared risk factors exacerbating both conditions. Decreased appetite, altered taste perception, and reduced nutrient absorption contribute to inadequate calorie and protein intake, leading to muscle loss and fat accumulation [6]. Consistent with these findings, our study found the lowest MNA values in the SO group. This underscores the compounded nutritional challenges posed by the coexistence of sarcopenia and obesity, both of which are forms of malnutrition in their own right.

4.4. Handgrip Strength and Physical Performance

Handgrip strength is a key indicator of sarcopenia. Studies suggest that obesity can negatively affect muscle strength due to increased fat infiltration, chronic inflammation, and metabolic changes [22]. Despite increased body weight, muscle mass may be underdeveloped or decreased in obese individuals. Prolonged obesity can lead to a decrease in muscle mass and function. Excess body fat can impair muscle function and strength. In addition, inflammation and metabolic changes due to obesity can reduce muscle strength. Lower physical activity levels in obese individuals also contribute to reduced muscle strength and mass [23,24]. Some studies have reported varying results regarding the relationship between handgrip strength and body mass index [25]. Our study found that individuals with SO exhibited lower handgrip strength than those with either sarcopenia or obesity alone. This suggests that the combined impact of sarcopenia and obesity on muscle strength may be more severe.

4.5. Frailty and Mortality in Sarcopenic Obesity

Frailty is a syndrome characterized by decreased reserve and resistance to stressors, often linked to sarcopenia and obesity [26,27]. This change significantly affects their mobility, balance, energy production, and general endurance, making them extremely susceptible to health problems [28–30]. The combination of these conditions exacerbates frailty, as seen in our study, where SO patients had the highest clinical frailty scores. The metabolic and physical burdens associated with SO significantly affect mobility, balance, and endurance, increasing susceptibility to adverse health outcomes.

Our study also found that the mortality rate was highest in the SO group. While sarcopenia and obesity independently increase mortality risk, evidence regarding their combined effect is mixed [1,3,31,32]. Differences in SO definitions across studies may contribute to this variability. Despite these limitations, our findings align with studies suggesting that SO poses a greater mortality risk than either condition alone.

4.6. Depression and Sarcopenic Obesity

Interestingly, our study found higher depression levels in the S group compared to the SO group. While physical limitations and social isolation increase depression risk, the "obesity paradox" may explain [33–35]. Mild to moderate obesity has been suggested to exert protective effects against depression in some cases.[36–38]. Social stigma and body image issues linked to obesity may raise depression rates in older obese adults [39,40]. These findings warrant further investigation into the complex interactions between sarcopenia, obesity, and mental health.

4.7. Walking Speed and Sarcopenic Obesity

Walking speed is a critical marker of functional status. Our study found the lowest walking speed in the O group despite its inclusion in sarcopenia grading. Individuals with sarcopenia may experience weakness and decreased muscle function, leading to slower walking speeds [41–43]. Obesity may also slow walking owing to increased energy costs and stress on muscles and joints [44,45]. This unexpected result highlights the need for further prospective cohort studies to better understand the interplay between obesity, sarcopenia, and walking speed.

4.8. Interactions of Risk Factors and Their Effects on the Development of SO

The relationships between sarcopenic obesity (SO) and risk factors such as diabetes and hypertension have been prominent in our studies. However, how these risk factors contribute to SO development through biological pathways should be examined in more detail. The effect of diabetes on SO can be explained through insulin resistance and chronic inflammation [46]. Insulin resistance can increase muscle protein breakdown by causing muscle cells to use glucose inadequately. This process accelerates muscle loss and may also trigger obesity-related fat accumulation. The connection between hypertension and SO can be associated with vascular disorders and reduced peripheral blood flow. Hypertension can prevent adequate oxygen and nutrient supply to muscle tissue, impairing muscle function. In addition, both conditions are associated with low-grade chronic

inflammation, which can create an environment that promotes muscle loss and fat accumulation [47,48]. Considering the potential interactions between these risk factors, it can be suggested that diabetes, for example, may combine with hypertension to create a “double-whammy” effect that further worsens SO. Although limited studies in the literature support these mechanisms, future studies are needed to better understand this issue. Further elaboration of these biological pathways in the development of SO may contribute to developing more effective prevention and treatment strategies for this group of individuals.

4.9. Limitations and Strengths

This study has several limitations that should be acknowledged to provide a comprehensive understanding of the findings: The retrospective cross-sectional design of our study limits the ability to infer causal relationships between sarcopenic obesity (SO) and the clinical outcomes observed. While associations are evident, the lack of temporal data prevents establishing whether SO is a direct cause or a consequence of these outcomes. Future longitudinal studies are necessary to confirm causality and explore the progression of SO over time. Although multivariate analyses were conducted to adjust for some variables such as hypertension and diabetes, there are other potential confounding factors that were not accounted for in this study. Systemic syndromes, other comorbidities (e.g., chronic kidney disease, cardiovascular diseases), and lifestyle factors (e.g., physical activity levels, dietary habits) could have influenced the results. The inability to fully control for these factors is a limitation that may have introduced bias into our findings. Our sample was derived from hospitalized patients, which may not represent the general geriatric population. Hospitalized individuals often exhibit more severe health conditions and frailty compared to community-dwelling older adults. This selection bias limits the external validity of our findings and may overestimate the prevalence and impact of SO in the broader population. Future research involving community-based samples could provide more generalizable insights.

This study has several strengths that contribute significantly to the understanding of sarcopenic obesity (SO) in older adults. First, the comprehensive use of geriatric assessment tools, including ADL, IADL, MMSE, MNA, GDS, and handgrip strength, allows for a multidimensional evaluation of physical, cognitive, and psychosocial health. Additionally, the study provides a detailed analysis of SO, highlighting its distinct impacts compared to obesity or sarcopenia alone. By utilizing logistic regression, the study identifies independent risk factors for SO, such as diabetes, hypertension, and poor nutritional and cognitive status, offering valuable insights for targeted interventions. Furthermore, the association of SO with frailty and mortality underscores its clinical importance and emphasizes the need for early detection and management. The hospital-based real-world data strengthen the study's relevance, while the inclusion of a diverse range of functional, cognitive, and biochemical parameters enriches its findings. Overall, the study addresses an underexplored area in the literature, providing a robust foundation for future research and clinical applications.

5. Conclusions

Our results indicate that individuals with SO have greater physical dependence, mortality, and frailty than those with sarcopenia or obesity alone. Individuals with sarcopenia are more likely to show depressive symptoms, possibly due to the effects of muscle loss and functional impairment on mental health. Notably, individuals with obesity demonstrated slower walking speeds compared to those with sarcopenia and sarcopenic obesity, reflecting the specific biomechanical challenges of excess body fat. Additionally, factors such as poor nutritional and cognitive status, low muscle strength, hypertension, and diabetes may contribute to the risk of developing SO.

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Informed Consent Statement: Written informed consent was not required for this study due to its retrospective design. The study was conducted in accordance with ethical standards and approved by the Clinical Research Ethics Committee (Approval No: [E1-22-2391]), which waived the requirement for individual consent.

Data Availability Statement: For reasons of privacy and ethics, access to the data is restricted.

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