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Original article

Sarcopenic obesity versus sarcopenia alone with the use of probable sarcopenia definition for sarcopenia: Associations with frailty and physical performance

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SUMMARY

Background & aims: Ageing brings alterations in body composition, as skeletal muscle gradually declines and accumulation of adipose tissue accompanies it. Although sarcopenia (S) and obesity (O) were separately reported to be associated with frailty and poor physical performance, whether they bring more detrimental or favorable effect when they coexist (i.e. sarcopenic obesity; SO) is an issue needs clarification. We aimed to study the associations of SO and S alone with frailty and poor physical performance, by using probable S definition.

Methods: This was a retrospective, cross-sectional study including community dwelling older adults who were ≥60 years old and admitted to the outpatient clinic of a tertiary hospital between 2012 and 2020. We measured handgrip strength via hand dynamometer and defined decreased muscle strength as probable S. We performed bioimpedance analysis to evaluate body composition and used fat percentile method to define obesity. We assessed nutritional status via Mini-Nutritional Assessment-Short Form, frailty via FRAIL scale, and physical performance via Timed Up and Go (TUG) test. We examined the associations of four body phenotypes, i.e. *non-S non-O*, *SO*, *S alone* and *O alone* with frailty and impaired TUG in univariate and multivariate analyses (Model 1). We further performed a head to head analysis of *SO* vs *S* to see if one of them was associated more with frailty and impaired TUG (Model 2).

Results: There were 1366 older adults included in the study (mean age: 74.6 ± 6.9; 68.3% female). The prevalences of *non-S non-O*, *S alone*, *SO* and *O alone* were 53.5, 7.5, 2.8 and 36.2%, respectively. Multivariate analysis adjusted for age, gender and nutritional status revealed that both *SO* and *S alone* were independently associated with frailty and impaired TUG, with *SO* demonstrating lower odds than *S alone* (OR = 5.9 and 6.05 for frailty, and 3.9 and 4.4 for TUG, respectively). Head-to-head comparison between *SO* and *S alone* in Model 2 showed that two groups did not demonstrate significant difference in terms of the frailty and impaired TUG risk.

Conclusion: Although *SO* and *S groups* demonstrated similar risks, obesity accompanying sarcopenia might show a favorable trend in terms of frailty and poor physical performance, compared to sarcopenia alone. Longitudinal studies are needed to reveal whether an obesity paradox exists for frailty and physical performance in older adults.

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1. Introduction

Ageing brings several structural and biochemical changes that result in functional decline in multiple systems. Alterations in body composition, primarily in skeletal muscle and adipose tissue, form the most prominent phenotypical changes that occur with ageing. Skeletal muscle gradually declines with age and especially after age of 60, this decline is often accompanied by increase in adipose tissue [1]. Extremes of both conditions, i.e. sarcopenia and obesity may be accompanying each other and since both are separately associated with negative outcomes [2,3], it can be thought that their combination, known as sarcopenic obesity (SO), may create a synergistic effect. In fact, while there are studies reporting unfavorable results of

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this synergy [4], there are others claiming that when sarcopenia is accompanied by obesity, it may paradoxically show a protective effect on several outcomes, and named this phenomenon as 'obesity paradox' [5,6]. The concept of obesity paradox mostly depends on a simplistic obesity definition based on BMI, hence it may also be called as 'BMI paradox' in some cases. However, there are some other studies that came up with this thesis in which other diagnostic methods were used rather than BMI for obesity (i.e. fat percentage method by dual energy X-ray absorptiometry [DXA] or bioelectrical impedance analysis [BIA]) [5,6]. The reasons behind this diversity in 'sarcopenic obesity field' are primarily caused by absence of a standard definition, and use of different measurement methods, thresholds and study populations [7].

In order to speak the same language, and create a standard definition, various reports have been presented in recent years, especially for sarcopenia. The European Working Group on Sarcopenia in Older People 2 (EWGSOP 2) consensus report is one of the most referred one for assessment of sarcopenia and it put muscle strength forward rather than muscle mass [8], since decrease in muscle strength precedes decrease in muscle mass [9], and is better associated with adverse outcomes like decreased functionality and mortality [7,10]. Therefore, EWGSOP2 recommends measuring muscle strength first, and called decrease in muscle strength 'probable sarcopenia' [8]. In fact, probable sarcopenia requires same interventions as confirmed sarcopenia, and diagnostic equipments for 'confirmation' may not be readily available in many clinics. Therefore, muscle strength evaluation creates the center of sarcopenia diagnosis recently [8]. When it comes to obesity diagnosis, the definition of World Health Organisation (WHO) has been preferred mostly for years [11], however it does not take gender difference into account. In addition, thresholds defined for obesity or overweightness were created using data from adults aged 18 years. Stature decrease that occurs by ageing is expected to affect body mass index (BMI) and there is not an age specific, alternative but also standard threshold of BMI for older adults [12]. Recently, several reports have suggested using fat percentage method for appropriate evaluation of obesity in older sarcopenic cases, rather than BMI [13,14].

Sarcopenia and obesity, as two different concepts, are separately associated with increased risk of frailty, another geriatric syndrome, and poor physical performance as well [8,15]. As such, there is a gap in the literature on whether the coexistence of two concepts brings out protective or detrimental effects on frailty and physical performance than *S alone*. In a recent study, we showed that *SO* was associated with a lower risk of disability (i.e. instrumental activities of daily living) when compared to *S alone* [6]. Functionality, frailty and physical performance have interactions with each other, however, they are three different concepts. In this study, we aimed to investigate the associations of *SO* versus *S alone* with frailty and physical performance by using probable sarcopenia definition for sarcopenia assessment.

2. Materials and methods

2.1. Population and setting

This retrospective, cross-sectional study was conducted on community-dwelling older adults aged ≥60 years who admitted to our tertiary health clinic between November 2012–April 2020 and gave informal consent for participating comprehensive geriatric assessment. Exclusion criteria were being younger than 60 years old, refusal to participate, having certain conditions that might interfere with reliable measurements (i.e. moderate to severe dementia and moderate to severe depression, which might affect patients' focus and judgement and hence, reliability of the answers to the screening tools or chronic illnesses and medication use and cooperation to the tasks and measurements such as muscle strength; hearing impairment that caused trouble hearing speech; hand osteoarthristis, stroke or peripheral artery disease that might interfere with measurement of handgrip strength; having metal implants or pacemakers and edematous state that might interfere with BIA measurements and insufficient vision to walk three meter line or being unable to leave a seated position for TUG test). The results of the study that examined the associations of *SO vs S alone* with functionality in this patient population has been published very recently [6]. We followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [16]. Istanbul University, Istanbul Medical School Ethics Committee approved the study protocol (reference: 1296/2020).

2.2. Sample size estimation

Based on the reported prevalences of low muscle strength and obesity co-existence in older adults, we performed a sample size calculation [17,18]. Using a power of 80% and two-sided significance level of 0.05, the minimum required sample size would be 306. Considering the stratifications to be made, we decided to expand the sample size by twice and reached out a minimum final sample size of 612.

2.3. Measurements

We obtained participants' demographic and clinical variables. Before moving on to further assessments, we screened for moderate to severe cognitive impairment via Mini-Mental State Examination (MMSE) and excluded the individuals scoring less than 21 points [19]. We also performed Geriatric Depression Scale-Short Form (GDS-SF) and excluded individuals scoring 10–15 points [20]. We assessed the nutritional status via Mini-Nutritional Assessment-Short Form (MNA-SF): a score of less than 12 was defined as 'undernutrition (malnutrition risk + malnutrition)' and less than eight as malnutrition [21]. We used five-item FRAIL (Fatigue, Resistance, Ambulation, Illnesses, Loss of weight) scale in order to assess participants' frailty status. We assessed "fatigue" by asking how much time participants had felt tired/exhausted during last four weeks, with responses 'all the time' or 'most of the time' scored one point. We assessed "resistance" by asking participants whether they had any difficulty in climbing 10 steps alone without resting or any personal-equipmental help. We asked whether they had any difficulty walking approximately 200 m alone and without any aid to assess "ambulation"; and each of these two items deserved one point, if responded as 'yes'. We asked whether any doctor had told them they had: hypertension, diabetes mellitus, chronic lung disease, heart attack, congestive heart failure, angina pectoris, asthma, arthritis, stroke, kidney disease or cancers (other than minor skin cancers) and a total of five or more illnesses deserved one point. We assessed weight loss by asking how much did they weigh with their clothes on and without shoes recently and one year ago and calculated the weight change as percentage [weight 1 year ago - current weight]/weight 1 year ago]] * 100. 5% loss of weight scored 1 point. We interpreted total FRAIL score as follows: 0 point = robust, 1–2 points = prefrail and ≥3 out of 5 points = frail [22].

We evaluated physical performance via Timed-Up and Go (TUG) test. We asked participants to rise from a standard chair with an approximate seat height 46 cm, walk to a marker 3 m away, turn around, walk back and sit down again. Time needed to complete the steps exceeding 20 s interpreted as 'impaired TUG test' [23].

Recent consensus report on definition and diagnosis of *SO* have recommended that muscle functional parameters should be

assessed as the first step in *SO* diagnosis and also indicated that muscle strength should be the functional parameter of choice [24]. In this context, we defined sarcopenia (S) component of *SO* according to the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) consensus definition as 'probable S (low muscle strength)'. We preferred to use probable S definition, since strength was reported to be better than mass in predicting adverse outcomes [8]. We assessed muscle strength by measuring handgrip strength (HGS) via Jamar hydraulic hand-dynamometer and by using a standard protocol [25]. We asked participants to get their elbow in 90° flexion and wrist in a neutral position and apply their maximum HGS with both hands separately three times with 30 s rest between each measurement. We used the thresholds recommended by EWGSOP2, i.e. <27 kg and <16 kg in males and females, respectively [8]. For obesity definition, recent guide on *SO* diagnosis have recommended measuring fat mass percentage [24]. We used the fat percentage method defined by Zoico et al. (fat percentile above 60th) [26]. We derived fat percentage from electric data generated by Tanita BC-532 bioimpedance analyzer, which has been showed to estimate body composition comparable with magnetic resonance imaging [27]. We used population specific thresholds for obesity diagnosis, i.e. > 27%/41% in males and females, respectively [13]. Accordingly, we categorized the participants into four groups by the stated parameters: *Non-sarcopenic non-obese, S alone, SO and O alone*. A single qualified physiotherapist performed all of the above-mentioned measurements.

2.4. Statistical analysis

We performed normality analysis using visual (histogram and probability plots) and analytical methods. We presented categorical variables as number and frequencies, normally distributed numerical variables as mean ± standard deviation and skew distributed variables as median (minimum and maximum). We used Chi-square test with Yates correction and Fisher's exact test for 2 × 2 contingency tables when appropriate for non-numerical data. We used independent samples t-test or Mann Whitney U test for comparison of differences between two independent groups. We used Chi-square test for comparison of more than two categorical variable groups. We created two models and run logistic regression analysis: We included parameters that were significantly associated with frailty and impaired TUG in univariate analyses and run regression analysis to assess whether frailty or impaired TUG were independently related to four body phenotypes: *Non S non Obese*, *S alone*, *SO* and *Obesity alone* (Model 1). We further defined a different independent variable as *SO vs S* (Model 2), in order to make a head-to-head comparison between two body phenotypes. We presented findings of regression analyses as Odds Ratio (OR) and 95% Confidence Interval (CI). We accepted a p value of less than 0.05 as statistical significance. We used Statistical Package for Social Sciences (SPSS) for Windows 21.0 program for statistical analysis.

3. Results

3.1. General characteristics and prevalences of different body phenotypes

A total of 6278 patients applied to the outpatient clinic in the study period. Among them, 1822 were not offered participation due to staffing issues, 915 had problems that interfered with healthy communication, 1846 had conditions that prevent reliable measurements and 329 refused to undergo a comprehensive geriatric assessment. The remaining 1366 participants constituted the final study population (Fig. 1). The mean age was 74.6 ± 6.9 (minimum–maximum: 60–99), and 933 patients (68.3%) were female. According to FRAIL index, 207 (15.2%) participants were frail, 577 (42.2%) were prefrail and 582 (42.6%) were robust. According to TUG test results, 90 patients (6.6%) showed poor physical performance. There were 1365 participants with data on nutritional status; 339 (24.8%) had undernutrition and 38 (2.8%) had malnutrition, according to MNA-SF. We presented the characteristics of the study population in Table 1.

Probable S prevalence was 10.4% and obesity prevalence was 39.0%. The prevalences of *non-S non-Obese*, *S alone (probable)*, *SO (probable)* and *Obesity alone* body phenotypes were 53.5, 7.5, 2.8 and 36.2%, respectively. Overall probable S and *S alone (probable)* and *SO (probable)* prevalences were significantly higher in male participants ($p < 0.001$, for each) (Table 1).

3.2. Univariate association of different body phenotypes, age, sex and nutritional status with frailty and poor physical performance

3.2.1. Univariate analyses of body phenotypes with frailty and poor physical performance

Frailty and poor physical performance were more prevalent in participants with *S alone* (probable) compared to *non-S non-Obese* (44.1% vs 12.2% for frailty and 28.4% vs 4.8% for impaired TUG test, respectively; $p < 0.001$) and *Obesity alone* groups (44.1% vs 12.1% for frailty and 28.4% vs 3.8% for impaired TUG test, respectively; $p < 0.001$). Furthermore, both frailty and impaired TUG test were significantly more prevalent in *SO* group compared to *non-S non-Obese* (33.3% vs 12.2% for frailty and 17.9% vs 4.8% for impaired TUG test, respectively; $p < 0.001$) and *Obesity alone* group (33.3% vs 12.1% for frailty and 17.9% vs 3.8% for impaired TUG test, respectively; $p < 0.001$).

There was no significant difference in prevalences of frailty and poor physical performance between *SO* and *S alone (probable)* groups (33.3% vs 44.1% for frailty; $p = 0.24$ and 17.9% vs 28.4% for impaired TUG test, $p = 0.20$). The detailed results can be found in Table 2.

3.2.2. Univariate association of frailty and poor physical performance with age, sex and undernutrition

Patients with frailty and poor physical performance were significantly older (77 vs 74 for frailty and 80.5 vs 74 for impaired TUG test, $p < 0.001$ for both). Frailty was significantly more prevalent in female compared to male participants (18.5% vs 7.9%, respectively; $p < 0.001$); but the prevalence of poor physical performance was similar between both genders (7.4% vs 4.8%, for female and male groups, respectively; $p = 0.07$). Furthermore, frailty and impaired TUG test were significantly more prevalent in participants with undernutrition compared to those without undernutrition (31.9% vs 9.6% for frailty and 15.6% vs 3.6% for impaired TUG test, $p < 0.001$ for both). Detailed results can be found in Table 2.

3.3. Multivariate analyses of body phenotypes with frailty and poor physical performance

In order to find out the relationship between body phenotypes and frailty, we determined frailty as dependent variable and factors significantly associated with frailty in univariate analysis as independent variables (four body phenotypes, age, sex and nutritional status). For associations between different body phenotypes and poor physical performance, we determined impaired TUG test as dependent variable, and factors significantly associated with poor physical performance as independent variables (four body phenotypes, age and nutritional status).

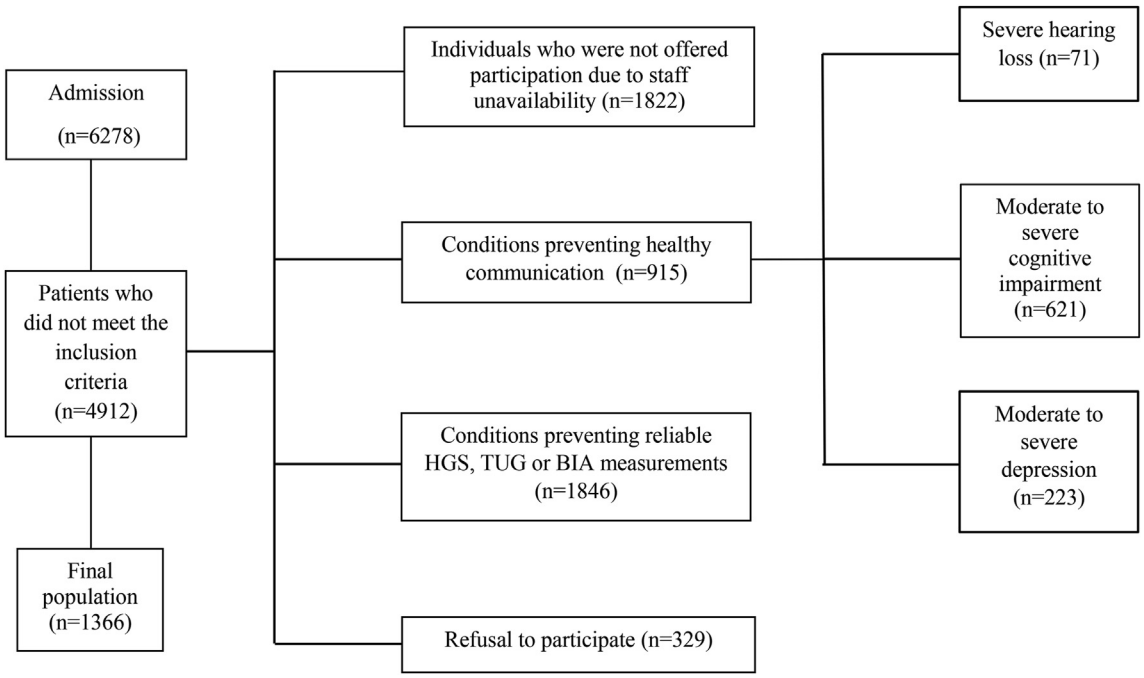


Fig. 1. Flowchart on the number of patients included in the study.

Table 1
Characteristics of the study population and prevalences of different body phenotypes (n = 1366).

	Total	Female	Male	p value
Study population (n, %)	1366	933 (68.3%)	433 (31.7%)	N/A
Age (years) (median, IQR)	75 (69–80)	74 (71–81)	76 (68–79)	<0.001
Number of regular drugs (median, IQR)	6 (4–8)	6 (4–8)	6 (3–9)	0.9
Number of chronic diseases (median, IQR)	4 (2–5)	4 (2–5)	4 (2–5)	0.6
Chronic diseases[a]				
Hypertension	429 (73.1%)	306 (75.4%)	123 (68.0%)	0.06
Diabetes mellitus	178 (30.1%)	125 (30.8%)	53 (29.3%)	0.7
Ischemic heart disease	113 (19.3%)	54 (13.3%)	59 (32.6%)	<0.001
Dyslipidemia	184 (31.3%)	127 (31.3%)	57 (31.5%)	0.9
COPD	38 (6.5%)	17 (4.2%)	21 (11.6%)	0.001
Assessment of frailty, physical performance and nutritional status				
Frailty[b]	207 (15.2%)	173 (18.5%)	34 (7.9%)	<0.001
Impaired TUG test	90 (6.6%)	69 (7.4%)	21 (4.8%)	0.08
Undernutrition (MNA-SF<12)	339 (24.8%)	244 (26.2%)	95 (22.0%)	0.1
Malnutrition (MNA-SF<8)	38 (2.8%)	25 (2.7%)	13 (3.0%)	0.7
Body phenotype (S vs Obesity)				
Sarcopenia[c]	141 (10.3%)	68 (7.3%)	73 (16.9%)	<0.001
Obesity[d]	533 (39%)	348 (37.3%)	185 (42.7%)	0.06
Body phenotypes (Non S Non Obese vs S alone vs SO vs Obesity alone)				
Body phenotype				
Non-S non-Obese	731 (53.5%)	533 (57.1%)	198 (45.7%)	<0.001
S alone	102 (7.5%)	52 (5.6%)	50 (11.5%)	<0.001
SO	39 (2.8%)	16 (1.7%)	23 (5.3%)	<0.001
Obesity alone	494 (36.2%)	332 (35.6%)	162 (37.4%)	0.5

Abbreviations and acronyms: COPD: Chronic Obstructive Pulmonary Disease, IQR: interquartile range, MNA-SF: Mini Nutritional Assessment-Short Form, S: sarcopenia, SO: sarcopenic obesity, TUG: Timed Up and Go.
[a] The data regarding chronic diseases was available for 587 participants.
[b] Frailty was assessed via FRAIL scale.
[c] S was defined as probable via measuring handgrip strength.
[d] Obesity was defined using fat-percentage via bioimpedance analysis.

3.3.1. Multivariate analyses of associations of four body phenotypes with frailty and poor physical performance (Model 1)

3.3.1.1. Frailty. We determined the *non-S non-Obese* participants as reference body phenotype group and performed the analyses accordingly. Both *S alone (probable)* and *SO* were independently associated with frailty (OR = 6.05 and 5.9, respectively; $p < 0.001$). On the other hand, there was no significant association between frailty and *obesity* (OR = 1.39, $p = 0.08$). Older age (OR = 1.4, $p = 0.007$), female gender (OR = 4.1, $p < 0.001$) and undernutrition (OR = 3.7, $p < 0.001$) were independently associated with frailty as well.

3.3.1.2. Poor physical performance. When we determined *non-S non-Obese* group as the reference group, both *S alone (probable)* and *SO* were independently associated with impaired TUG test (OR = 4.4 and 3.9 for *S alone* and *SO* groups; $p < 0.001$ and

Table 2
Associations of body phenotypes, age, sex and nutritional status with frailty and poor physical performance (univariate analyses).

	Frailty	p	Impaired TUG	p
Body phenotype		***<0.001***		***<0.001***
Non-S non-Obese (n = 731)	89 (12.2%)		35 (4.8%)	
S alone (n = 102)	45 (44.1%)	<0.001[a,b] 0.24[c]	29 (28.4%)	<0.001[a,b] 0.2[c]
SO (n = 39)	13 (33.3%)	<0.001[d,e]	7 (17.9%)	<0.001[d,e]
Obesity alone (n = 494)	60 (12.1%)		19 (3.8%)	
Age (years) *(Impaired/Non-impaired)*^	77/74	<0.001	80.5/74	<0.001
Minimum-maximum	(60–99)/(60–98)		(62–99)/(60–99)	
Sex		<0.001		0.08
Male (n = 433)	34 (7.9%)		21 (4.8%)	
Female (n = 933)	173 (18.5%)		69 (7.4%)	
Undernutrition		<0.001		<0.001
MNA-SF ≥12 (n = 1026)	99 (9.6%)		37 (3.6%)	
MNA-SF<12 (n = 339)	108 (31.9%)		53 (15.6%)	

Abbreviations: MNA-SF: Mini Nutritional Assessment-Short Form, S: sarcopenia, SO: sarcopenic obesity, TUG: Timed Up and Go.
Categoric variables were given as number (%).
*Frailty was assessed via FRAIL scale.
**S was defined as probable via measuring handgrip strength.
***Obesity was defined by fat percentile method (>60th percentile) using bioimpedance analysis.
^Median.
[a] *S alone* vs *Non S Non Obese*.
[b] *S alone* vs *Obesity alone*.
[c] *S alone* vs *SO*.
[d] *SO* vs *Non S Non Obese*.
[e] *SO* vs *Obesity alone*.

p = 0.004, respectively). However, there was no significant association between *Obesity alone* and impaired TUG test (OR = 1.2, p = 0.5). Both older age and undernutrition were the other variables independently associated with poor physical performance (OR = 1.1 and 3.8 for older age and undernutrition respectively; p < 0.001). The results were given in detail in Table 3.

3.3.2. Multivariate analyses of association of SO vs S alone with frailty and poor physical performance (Model 2)

3.3.2.1. Frailty. The risk of frailty was similar between *SO* and *S alone* groups (p = 0.9). On the other hand, female gender and undernutrition significantly increased the risk of frailty (OR = 3.4, p = 0.02 and OR = 5.2, p < 0.001 for female gender and undernutrition, respectively).

3.3.2.2. Poor physical performance. There was no difference in the risk of poor physical performance between SO and S alone groups (p = 0.4). Neither older age nor undernutrition were independently associated with increased risk of impaired TUG test (p = 0.09 and 0.9 for older age and undernutrition, respectively). The results were detailed in Table 3.

4. Discussion

In this study, we aimed to investigate the associations of *SO* and *S alone* with frailty and poor physical performance and found out that: (i) When compared to *non-S non-Obese* body phenotype, both *SO* and *S alone* were independently associated with increased risks of frailty and poor physical performance measures. However, the risks of frailty and impaired TUG test were lower in *SO* phenotype than the *S alone*; (ii) When we compared *SO* vs *S* head-to-head specifically for their relationship with frailty and poor physical performance, we found that neither was more detrimental than the other in terms of the aforementioned negative outcomes.

When S was defined as decline in muscle strength, *SO* prevalence ranges between 3.2 and 11% in the literature [6,17,18]. This

Table 3
Multivariate analyses for associations of body phenotypes with frailty and physical performance measures.

	Frailty[a]	p	Impaired TUG[b]	p
	OR (95% CI)		OR (95% CI)	
Model 1				
Body phenotype (all four phenotypes)				
Non S non Obese	*Reference*		*Reference*	
S alone	6.05 (3.6–10.2)	<0.001	4.4 (2.4–7.9)	<0.001
SO	5.9 (2.6–13.2)	<0.001	3.9 (1.5–9.9)	0.004
Obesity alone	1.4 (0.9–2.03)	0.08	1.2 (0.7–2.2)	0.49
Nutritional status (undernutrition)	3.7 (2.7–5.2)	<0.001	3.8 (2.4–6.1)	<0.001
Age	1.03 (1.00–1.05)	0.007	1.1 (1.06–1.14)	<0.001
Sex (Female)	4.1 (2.6–6.4)	<0.001	N/A	
Model 2				
Body phenotype (*SO vs S alone*)	1.02 (0.4–2.5)	0.96	0.6 (0.2–1.7)	0.36
Nutritional status (undernutrition)	5.2 (2.9–11.3)	<0.001	1.9 (0.9–4.3)	0.09
Age	1.02 (0.9–1.08)	0.35	1.00 (0.9–1.05)	0.93
Sex (Female)	3.4 (1.6–7.4)	0.02	N/A	

S: sarcopenia, SO: sarcopenic obesity, TUG: Timed Up-And-Go Test, CI: confidence interval.
S was defined as probable sarcopenia (decreased handgrip strength); obesity was defined by fat percentile method (>60th percentile) using bioimpedance analysis.
[a] Dependent variable was frailty; independent variables were different definitions of body phenotypes, nutritional status, age, sex.
[b] Dependent variable was impaired TUG; independent variables were different definitions of body phenotypes, nutritional status, age.

wide range is primarily caused by use of different methods and definitions for identifying poor muscle strength and obesity. In these studies, anthropometric indices were most used ones for obesity evaluation, especially BMI and waist circumference. Although anthropometric measurements are more practical and do not require any specialty or expensive equipments, they show poor sensitivity in identifying obesity in older adults. Batsis et al. reported that only approximately 41% of older men and 45% of older women were correctly identified as obese via BMI definition, while waist circumference promised relatively higher percentages (64.2 and 81%, for men and women, respectively); using fat percentage measurement via DXA as gold standart method [28]. Kemmler et al. also reported that BMI would underestimate obesity prevalence, and body fat percentage should be used for assessment of *SO*; as applying fat percentage method resulted in 3–4 fold higher prevalence rates compared with a BMI based approach [14]. Similarly, we have previously reported that *SO* prevalences were negligible with an obesity diagnosis made via using BMI compared to fat percentage method, with an S definition as low muscle mass and function [13]. In order to avoid overlook of obesity in older adults, measurement of fat percentage seems more appropriate, especially for *SO* assessment.

Both S and obesity are reported as significantly associated with frailty and poor physical performance [8,15,29–32]. On the other hand, obesity may have favorable effects in older adults [33,34]. However, whether the coexistence of both concepts exacerbate frailty and poor physical performance is still unclear [4–6]. Indeed, there are two opposite views about the effects of SO in the literature. Firstly, ageing brings a gradual increase in adipose tissue (up to very old age). Accumulation of adipose tissue causes a low-grade chronic inflammatory status and can result in disruption of beneficial metabolic activities of both adipose and muscle tissue, increase in oxidative stress, insulin resistance, and create a vicious cycle of decline in muscularity and increase in adiposity [35]. Furthermore, redistribution of adipose tissue with increasing age can cause myosteatosis and the lipotoxic environment in muscle tissue can decrease muscle quality [36]. It is also stated that myosteatosis can cause conversion of type II rapid twitch muscle fibers to type I slow-twitch fibers and decrease muscle strength as well [37]. Besides negative effects on frailty and physical performance through muscle strength and quality/quantity, high levels of adiposity can cause several metabolic and cardiovascular diseases and cancers and increase risk of frailty and low physical performance as well [38]. In addition, obesity may be the result or cause of a more sedantary life style, and this vicious cycle probably bring an increased risk of frailty and decline in physical performance status.

Besides the reported (or expected) negative results associated with obesity accompanying sarcopenia, some other authors support the opposite view, as obesity might be protective against the unfavorable results associated with S. Obesity paradox term was firstly used for addressing the inverse relationship found between overweight/obesity and mortality in several patient groups with chronic heart failure, diabetes or hypertension [39]. In terms of possible beneficial effects on muscle health, it was shown that obese people maintain a higher quantity of muscle mass compared to the lean group [40]. Reasons behind this finding is that obese individuals consuming meals that are rich not only in calories but also in protein would probably overcome the anabolic resistance in muscle protein synthesis occurring with older age [41]. Furthermore, higher muscle quantity would occur as a response to an increased overload stimulus applied to the muscles, in order to enable a heavy weighted body to create a certain movement. This overload stimulus would result in not only muscle mass, but also bone mineral density increase in obese individuals and would provide a protective effect against osteoporotic fractures and associated adverse outcomes that can further aggrevate muscle loss [42]. In addition, hypogonadism is one of the main causes of sarcopenia in older adults, and it is known that adipose tissue is a source of estrogen production. Estradiol increases muscle strength and mass, and stimulates muscle repair through stimulating proliferation of satellite cells in muscle tissue [43]. Hence, it can be considered that obesity accompanying sarcopenia would promise a more favorable profile in terms of physical frailty and performance, compared to sarcopenia alone counterparts, due to its beneficial effects on muscle tissue and bones.

Recently, there is a postulation of 'obesity threshold' after which obesity might become detrimental. Some authors claimed that an aging-obesity 'muscle quality threshold' might exist, beyond which a rapid decline in muscle mass and function occur [44]. It was also reported that obesity might not be deleterious for muscle mass and function unless present under situations like increased age, chronic inactivity or other conditions that result in anabolic resistance [44]. Hence, obese older adults with a relatively younger age and physically active lifestyle would benefit from advantages of increased adiposity over muscle tissue.

Back to our findings, we found out that the odds ratios for frailty and impaired TUG test were higher in *S alone* group compared to *SO* group (Model 1). This result would support the obesity paradox theory in regards of frailty and physical performance in older adults. However, when we defined *SO vs S* as a single independent variable for a head to head comparison and adjusted for age and nutritional status (and gender for frailty), we revealed that neither group had an advantage over the other in terms of frailty and physical performance (Model 2). Considering the points mentioned previously about the possible benefits and harms of obesity, it can be thought that the positive and negative effects of adipose tissue on muscles may be balanced, at least in the status of probable sarcopenia.

Although there are several publications on the relationship between *SO* and frailty and poor physical performance, the number of studies comparing *SO* to *S alone* is limited and the results are mixed. The longitudinal study of Concord Health and Aging in Men Project was conducted with 1700 community dwelling older men and revealed that *SO* had lower odds ratios for frailty compared to *S alone* in models adjusted for age, income, smoking status, physical activity, number of comorbidities, myocardial infarction, dementia, depressive symptoms, low haemoglobin, polypharmacy and white cell count. Different from our study, the authors did not perform a head-to-head analysis for *SO* vs *S alone*, to look for any significant diffference between each other. The authors used fat percentage method for obesity diagnosis and used FNIH definition for sarcopenia [45]. Although it was conducted on a specific population (hemodialysis patients), Saitoh et al. reported that odds ratios for frailty was higher in *SO* group compared to *S alone* group after adjusting for age, gender and diabetes mellitus. S definition they used was based on the first consensus report of the Asian Working Group for Sarcopenia for sarcopenia and they defined obesity via fat percentage method [46]. In a cross sectional study including 2039 older adults, the authors compared dynapenic obese (corresponding to *SO*) group with three other groups similar to our study [non-dynapenic non-obese (*non-S non-O*), non-dynapenic obese (*O alone*) and dynapenic non-obese (*S alone*)] in terms of associations with impaired 20-feet walking speed test. They measured fat mass via DXA and leg strength via dynamometer, and reported that dynapenic obesity (*SO*) was associated with a poorer physical function than dynapenia alone (*S alone*); in different models adjusted for age, gender, race/ethnicity, alcohol intake, smoking status, chronic conditions like diabetes and cardiovascular disease and physical activity [47]. A very recent study has defined obesity as

abdominal obesity (via measuring waist circumference) and dynapenia with grip strength, and reported that only dynapenic abdominal obesity was associated with decline in gait speed after eight-year follow-up, compared to non-dynapenic non-obese group. Neither dynapenia, nor abdominal obesity only was associated with gait speed decline [48]. In fact, there are two main anatomical compartments in white adipose tissue: subcutaneous (SAT) and visceral adipose tissue (VAT). By ageing and obesity, redistribution of fat tissue occurs as beneficial SAT decreases and VAT, which is stated to play an essential role in *SO* pathophysiology increases and becomes dysfunctional [35]. Hence, in the context of obesity accompanying sarcopenia, it is suggested that focusing more on visceral adiposity rather than total obesity would be more ideal. Finally, the effect of dynapenic obesity (corresponding to *SO*) on decline in mobility was examined based on six-year follow-up data from InCHIANTI study, using BMI for obesity and knee-extensor strength measurement for dynapenia assessment. It was reported that the most dramatic decrease in physical performance was detected in dynapenic obese (corresponding to *SO*) group, compared to other groups [dynapenic-non-obese (corresponding to *S alone*); non-dynapenic-non-obese (corresponding to *Non S Non O*) and obese-non-dynapenic (*O alone*)] [17]. It is well known that decline in muscle strength develops faster than decline in muscle mass [9]. As the process progresses, accompanying obesity may become more detrimental as some of the individuals might be crossing the so-called obesity-threshold after which muscle quality decreases [39]. In addition, U-shaped relationship between adiposity and frailty, disabilities and mortality was reported in older adults [39,49]. While overweight or mild degree of obesity were associated with lower risks, extreme levels of obesity were related to poorer outcomes. In this context, one can question whether the studies that documented higher risks of impaired physical performance and frailty with *SO* when compared to *S alone*, had included *SO* patients with more severe levels of obesity than the studies that found a lower risk of impairment with *SO*. We intended to compare the obesity levels of our study group to those studies to check whether this assumption was correct, however studies used different diagnostic methods and did not allow us to make a direct comparison. Very recently, a consensus report for *SO* was published, developed by a total of 38 researchers working on *SO* and endorsed by ESPEN (European Society for Clinical Nutrition and Metabolism) and EASO (The European Association for the Study of Obesity). In this report, diagnosis of obesity in the context of *SO* was recommended to be made via fat percentage method, rather than fat mass or BMI [24]. Common use of fat percentage method in future SO studies will probably allow us to obtain healthier and comparable results.

This study has some limitations. Firstly, it is a retrospective, cross-sectional study and a causal-effect relationship cannot be claimed. In addition, it was conducted on community-dwelling older adults admitted to an outpatient clinic in a tertiary hospital. Therefore, the findings cannot be generalized to the whole older adult population. Furthermore, evaluating frailty in obese individuals with a tool that questions weight loss might have created a handicap. In this context, an alternative tool that does not include weight loss item could be used (for example, SARC-F questionnaire with a cut-off of ≥ 2 was reported to be effective in identifying physical frailty [50]). One of the major strengths of our study is we have a high number of study participants which increases the reliability of the findings. Secondly, obesity was evaluated with fat-percentage method which was reported to be better than anthropometric indices in terms of examining sarcopenia and obesity relations. Thirdly, we evaluated physical performance with TUG test, which is a valuable tool since it enables evaluation of gait, dynamic balance and fall risk at the same time. Finally, this is the first study that evaluates both frailty and poor physical performance at the same time and examined their associations with different body phenotypes, to our knowledge.

In conclusion, our findings suggest that the associations of *SO* and *S alone* with frailty and poor physical performance are similar in the older adults, when we define *S* as 'probable sarcopenia' and *obesity* by fat-percentage method. The supposed beneficial and harmful effect that adipose tissue accumulation brings might be balanced when it accompanied to sarcopenia. In the context of *SO*, it seems that paying more attention to *S* component rather than obesity would be more appropriate in order to prevent adverse outcomes. Further studies with longitudinal design would reveal whether an obesity paradox really exists in older adults with sarcopenia.

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Author contribution

SO: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing- Original draft preparation; Writing-Reviewing and Editing; COA: Investigation, Data curation; DES: Investigation, Data curation; NMC: Investigation, Data curation; TE: Investigation, Data curation; MEB: Investigation, Data curation; CK: Data curation; MAK: Supervision; GB: Conceptualization, Methodology, Investigation, Writing- Reviewing and Editing.

Conflict of interest

There are no relationships/conditions/circumstances that present a potential conflict of interest.

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