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ORIGINAL CONTRIBUTIONS

The Impact of Age on the Prevalence of Sarcopenic Obesity in Bariatric Surgery Candidates

Judit Molero[1] · Violeta Moizé[1] · Lilliam Flores[1,2,3] · Ana De Hollanda[1,2] · Amanda Jiménez[1,2,4] · Josep Vidal[1,2,3]

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Abstract

Background Sarcopenia pre-dating bariatric surgery (BS) has been suggested as concern for the use of BS in older-adults with morbid obesity.

Objective To evaluate the impact of age on the prevalence of sarcopenic obesity (SO) in BS-candidates.

Methods Cross-sectional study including 1370 consecutive BS-candidates aged ≥18, and grouped according to age: 18–39 (reference group), 40–49, 50–59 and ≥ 60 years. From body composition analysis data obtained using bioelectrical impedance, skeletal muscle mass (SMM), SMM index (SMMI=SMM/height^2), and percentage of SMM (%SMM = SMM/BW*100) were calculated. Class I or class II SO was adjudicated, respectively, when a value between > − 1 and − 2, or > −2 standard deviations from the regression line from the gender-specific distribution of the relationship between BMI and SMMI or the %SMM in the reference group was encountered.

Results According to the SMMI distribution, prevalence of class I and class II SO in the whole cohort was respectively 16.4% and 4.6%. SO was more prevalent in females ($p < 0.005$). Proportion of subjects with SO positively correlated with older age category in females (Tau-c = 0.149, $p < 0.001$) but not in males. In females aged ≥60, class I SO was present in 29.1%, and class II in 12.8%. Similar results were obtained when %SMM was used (Cohen's k-coefficient = 0.886, $p < 0.001$). Age and female gender were identified as independent preditors of SO, whereas CRP or the presence of obesity-associated comorbidities were not.

Conclusion Age is a risk factor for SO in BS-candidates. SO is fairly common in female subjects aged >60 years that are candidates to BS.

Keywords Sarcopenia · Obesity · Bariatric surgery · Elderly · Aging

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✉ Josep Vidal
jovidal@clinic.cat

Judit Molero
jmolero@clinic.cat

Violeta Moizé
vmoize@clnic.cat

Lilliam Flores
lflores@clinic.cat

Ana De Hollanda
amdehol@clinic.cat

Amanda Jiménez
ajimene1@clinic.cat

[1] Obesity Unit, Endocrinology Department, Hospital Clinic Barcelona Universitar, Villarroel 170 08036 Barcelona Spain

[2] Institut d'Investigacions Biomèdiques August Pi Sunyer (IDIBAPS), Barcelona Spain

[3] Centro de Investigación Biomédica en Red de Diabetes y Enfermedades Metabólicas Asociadas (CIBERDEM), Barcelona Spain

[4] Centro de Investigación Biomédica en Obesidad y Nutrición (CIBEROBN), Barcelona Spain

Introduction

Prevalence of severe obesity in older-adults is in the rise [1]. Recognition of the safety and efficacy of bariatric surgery (BS) in this age group has prompted scientific societies endorse BS should not be denied to the elderly solely on age grounds [2, 3]. Nonetheless, some questions on the use of BS in older-adults still remain [4].

Sarcopenia (SO), a condition including reduced muscular mass (MM) and muscle strength [5], has been suggested as potential concern for the use of BS in the elderly [4]. Although loss of fat free mass (FMM) of approximately 25% of total WL is considered adequate following intentional WL interventions, it has been shown proportion FFM loss varies depending on age, gender, and speed of WL [6, 7] . Thus, concern has been raised on the potentially detrimental impact of BS in older-adults with SO prior to BS because of the poor health outcomes associated with low MM and impaired muscle function [4, 8]. Admittedly, the efficacy and safety of BS in the elderly is well grounded [2, 9]. However, data on the prevalence of SO in obese subjects presenting for BS are scant, and difficult to interpretate because of the use of different indices of MM, and lack of proper control groups [10–14]. Moreover, these studies included mainly subjects aged <60 years.

Against this background, the primary aim of our study was to evaluate the impact of age on the prevalence of SO in a large cohort of BS-candidates. Diagnosis of SO was based on the gender-specific distribution of the relationship between MM and body mass index (BMI) in a control group of young-adult (18–39 years of age) BS-candidates with comparable BMI distribution. Additionally, we evaluated the concordance between two body composition indices commonly used to define sarcopenia and assessed the association between SO and metabolic comorbidities in our cohort.

Subjects and Methods

Consecutive subjects aged ≥18 years evaluated for BS at our Institution (2006–2017) participated in this cross-sectional study. Indication of BS was based on the presence of a BMI ≥40.0 or 35.0–39.9 kg/m^2 if comorbid conditions present. Exclusion criteria included renal insufficiency (estimated glomerular filtration rate < 45 ml/min), severe hepatic disease (based on clinical criteria or liver enzymes ≥×3 upper limit of normal values), being a pacemarker carrier, presence of lower limb edema, uncontrolled hypothirodism, being diagnosed with cancer, active treatment with drugs associated with BW gain or WL, and prior BS.

Study subjects were grouped according to age: 18–39, 40–49, 50–59 and ≥ 60 years. Subjects aged 18–39 (young adults) were used as reference. Smoking habit, presence of type 2 diabetes (T2D), hypertension (HTN), dyslipidemia, and sleep apnea-hypopnea syndrome (SAHS) was recorded. Height and waist circumference were measured as previously reported [15].

Body composition (BC) was assessed using bioelectrical impedance analysis (BIA, Tanita BC418) according to the manufacturer specifications. From BIA, BW (to the nearest 0.1 kg), body mass index (BMI, weight in kg/height^2 in meters), electrical impedance (in ohms), FM (expressed in kg or as percentage of BW), FFM (expressed in kg) were obtained. The BIA-based formula proposed by Jansen was used to calculate skeletal muscle mass (SMM) [16], and SMM was used to calculate the SMM index (SMMI=SMM/height^2), and the percentage of SMM (%SMM = SMM/BW*100). Class I and class II SO were defined respectively as a value between > − 1 and − 2, or > −2 standard deviations (SD) from the gender-specific regression line of the BMI versus SMMI- or %SMM-relationship in the reference group [17].

A venous blood sample was obtained in the fasting state from the antecubital vein, and C-reactive protein, pre-albumin, HDL-cholesterol, triglycerides, glucose, insulin, and glycated hemoglobin were measured as previously reported [15].

The study was approved by the Institutional Ethics Committee. Informed consent was obtained from all study participants.

Statistical Analysis

Differences among groups for continuous variables were assessed using Analysis of Variance (ANOVA), with Bonferroni's test as post-hoc analysis. Chi^2 and Thau-C statistics were used, respectively, to evaluate differences and correlations among categorical variables. Variables that were not normally distributed (Kolmogorov-Smirnov test) were logarithmically transformed. Spearman's rho was used to evaluate bivariate correlations between continuous variables. Logistic regression analysis was performed to assess the independent effect of age and other variables on the presence of SO. Data are shown as mean ± SD unless stated otherwise. Statistical analysis was performed using SPSS 23.0 (SPSS Inc., Chicago, IL), with significance set at $p < 0.05$.

Results

Clinical, biochemical characteristics, and BC data of the 1370 study subjects are shown in Table 1. Respectively, 465 (33.9%), 412 (30.1%), 358 (26.1%), and 135 (9.9%) of the study subjects corresponded to the 18–39, 40–49, 50–59, and ≥ 60 age groups. BMI was slightly albeit statistically significantly lower in groups aged 50–59 and ≥ 60 years compared to the reference group (both $p < 0.05$). Correlation analysis showed an association between older-age group and larger prevalence of T2D, HTN, dyslipidemia, and SAHS (all $p < 0.001$). Age groups also differed in BC parameters. In

Table 1 Clinical, biochemical, and body composition characteristics of the study participants

		18–39 years ($n = 465$)	40–49 years ($n = 412$)	50–59 years ($n = 358$)	≥ 60 years ($n = 135$)	P value
Clinical data						
Sex (M/F, %)		26,5/73,5	28,4/71,6	26,0/74,0	13,3/86,7	0,006
Height (m)		1.66 (0.1)	1.64 (0.1) [a]	1.62 (0.1) [a]	1.58 (0.1) [a]	<0.001
Weight (kg)		127.4 (19.0)	123.1 (19.4) [a]	118.2 (18.6) [a]	112.1 (15.6) [a]	<0.001
BMI (kg/m^2)		46.1 (5.2)	45.5 (5.8)	45.1 (5.9)	44.3 (5.2) [c]	0.003
WC (cm)		130.5 (13.5)	130.5 (14.9)	129.8 (13.3)	127.2 (11.7)	0.080
SBP (mmHg)		128.6 (15.8)	135.2 (17.1) [a]	136.1 (16.2) [a]	137.0 (18.1) [a]	<0.001
DBP (mmHg)		79.6 (13.1)	82.6 (11.1) [a]	81.6 (9.8)	79.3 (9.1)	<0.001
Smoker (yes,%)		32.7	24.3	19.3	6.7	<0.001
T2D (yes,%)		11.2	25.0 [a]	33.8 [a]	40.7 [a]	<0.001
HTN (yes,%)		17.7	42.7 [a]	64.0 [a]	76.3 [a]	<0.001
Dyslipidemia (yes,%)		12.3	26.2 [a]	42.2 [a]	51.1 [a]	<0.001
SAHS (yes, %)		13.5	26.0 [a]	36.6 [a]	35.6 [a]	<0.001
Biochemical data						
hs-CRP (mg/dL)		1.3 (1.0)	1.1 (1.5)	1.0 (1.0)	0.9 (0.6) [a]	0.007
Glucose (mg/dL)		107.4 (34.3)	118.4 (43.7) [a]	125.9 (42.2) [a]	129.7 (46.4) [a]	<0.001
A1C (%)		5.2 (1.1)	5.6 (1.4)	6.0 (2.5)	6.1 (1.6)	<0.001
Insulin (mU/L)		29.2 (19.2)	28.6 (18.8)	27.1 (19.2)	24.4 (15.8)	0.173
HDL-C (mg/dL)	Male	36.8 (7.3)	39.7 (7.9) [a]	39.6 (7.8)	40.3 (8.3) [a]	0.017
	Female	45.1 (10.7)	46.0 (10.0)	48.5 (10.8) [a]	49.7 (10.6) [a]	<0.001
Triglycerides (mg/dL)		130.9 (74.0)	143.5 (95.9)	146.8 (80.7) [a]	139.5 (58.9)	0.029
Albumin (g/L)		44.2 (3.4)	43.9 (2.8)	43.9 (2.8)	43.5 (2.3)	0.091
Body composition data						
FM (%)	Male	38.6 (4.0)	38.3 (4.7)	40.1 (5.0)	40.1 (3.7)	0.021
	Female	50.6 (3,6)	49.6 (3.9) [a]	49.8 (3.5)	49.4 (3.3) [a]	0.001
FM (kg)	Male	55.2 (11.5)	53.2 (12.7)	54,6 (13.5)	52.3 (9.9)	0.565
	Female	62.2 (11.8)	58.5 (11.4) [a]	56.3 (10.4) [a]	54.1 (9.2) [a]	<0.001
FFM (kg)	Male	86.6 (9.3)	84.2 (8,9)	80.2 (8.9) [a]	77.1 (6.2) [a]	<0.001
	Female	60.0 (6.1)	58.7 (6.9)	56.3 (6.2) [a]	54.9 (5.5) [a]	<0.001
SMM (kg)	Male	35.1 (3.7)	34.9 (4.2)	33.6 (4.0) [a]	32.5 (3.0) [a]	0.005
	Female	23.7 (2.7)	22.9 (3.1) [a]	21.4 (2.7) [a]	20.5 (2.7) [a]	<0.001
SMMI (kg/m^2)	Male	11.4 (1.0)	11.5 (1.2)	11.4 (1.2)	11.1 (0.9)	0.559
	Female	8.9 (0.8)	8.8 (0.9)	8.5 (1.0) [a]	8.3 (1.0) [a]	<0.001
SMMI (%)	Male	24.9 (2.5)	25.6 (2.8)	25.2 (3,0)	25.3 (2.2)	0.350
	Female	19.5 (2.0)	19.6 (2,1)	19.2 (2.0)	18.8 (2.1) [a]	0.002

Data are presented as mean (SE) or as percent. M, male; F, female; BMI, body mass index; WC, waist circumference; SBP: systolic blood pressure; DBP: diastolic blood pressure; T2D: type 2 diabetes; HTN: hypertension; SAHS, sleep apnea-hypopnea syndrome; hs-CRP, high-sensitivity C-reactive protein; A1C, glycated hemoglobin; HDL-C, high-density lipoprotein Cholesterol; FM, fat mass; FFM, fat free fass; SMM, skeletal muscle mass; SMMI, Skeletal muscle mass index. *P* values for the comparison among groups (ANOVA) are presented in the p value column. a: p value<0.05 for the comparison between the reference group and other age groups in the post-hoc analysis (Bonferroni)

females, but no in males, subjects in the 50–59 and ≥ 60-years age group presented lower SMMI ($p < 0.001$) compared to the reference group. Moreover, %SMM was significantly lower in subjects aged ≥60 relative to young adults. Indeed, in females SMMI ($p < 0.001$), %SMM ($p < 0.01$) inversely correlated with age.

Gender-specific relationships between BMI and SMMI or %SMM are shown in Supplemental Fig. 1. Based on the above, prevalence of class I or II SO by age-group is shown in Table 2. According to the SMMI distribution, prevalence of class I and class II SO in the whole cohort was respectively 16.4% and 4.6%. Overall, SO based on this criterion was more prevalent in females (22.9% versus males 16.2%; $p < 0.005$). Proportion of subjects with SO positively correlated with older age category in females (Tau-c = 0.149, $p < 0.001$) but not in males. In females aged ≥60 class I SO was present in

Table 2 Prevalence of sarcopenia in the study participants

		Total (n = 1370)	18–39 years (n = 465)	40–49 years (n = 412)	50–59 years (n = 358)	≥ 60 years (n = 135)
According to SMMI						
No sarcopenia (%)	Male	83.8	84.6	83.8	82.8	83.3
	Female	77.1	85.4	82.0	69.4	58.1
Class I sarcopenia (%)	Male	14.8	15.4	15.4	12.9	16.7
	Female	17.3	12.0	13.9	22.6	29.1
Class II sarcopenia (%)	Male	1.4	0.0	0.9	4.3	0.0
	Female	5.6	2.6	4.1	7.9	12.8
According to %SMM						
No sarcopenia (%)	Male	84.9	85.5	83.9	83.3	84.9
	Female	78.7	87.1	83.7	70.9	59.0
Class I sarcopenia (%)	Male	13.4	14.6	12.0	12.9	16.7
	Female	15.6	10.5	12.2	20.8	27.4
Class II sarcopenia (%)	Male	1.7	0.0	2.6	3.2	0.0
	Female	5.7	2.3	4.1	8.3	13.7

Data are presented as percent. SMMI, skeletal muscle mass index; %SMM, percent skeletal muscle mass. a: p value<0.05 for the paired comparison between the reference group and other age groups

29.1%, and class II in 12.8%. Similar results were obtained when SO was ascertained from the the %SMM distribution (Table 2). Cohen's kappa coefficient of concordance for the diagnosis of SO between the two indices was 0.886 ($p < 0.001$). In both genders, %FM was larger in subject with class I and class II SO as compared to non-sarcopenic individuals (females: non-sarcopenic 49.2 ± 3.5%, class I-SO 52.3 ± 2.7%, class II-SO 53.5 ± 2.9, both $p < 0.001$; males: non-sarcopenic 38.2 ± 4.2%, class I-SO 42.7 ± 4.2%, class II-SO 46.4 ± 3.6, both $p < 0.001$).

Sarcopenic obesity (class I or class II) was associated with lower prevalence of active smoking, higher prevalence of hypertension, and lower levels of albumin compared to subjects without SO (Table 3). In logistic regression analysis with SO as dependent variable, age [HR 1.038 (95% CI 1.025–1.052), $p < 0.001$], and female gender [HR 1.440 (95% CI 1.041–1.992), $p < 0.001$], but not BMI, emerged as independent predictors. Addition to the logistic regression model of tobacco use, T2D, HTN, dyslipidemia, or SAHS did not modify the association between age and SO. Indeed, in the corresponding models active smoking [HR 0.687 (95%CI 0.485–0.972), $p = 0.034$], and T2D [HR 0.659 (95%CI 0.475–0.914), $p = 0.013$] emerged as independent predictors of the absence of SO. The later association was not modified when logistic regression analysis was limited to subjects aged≥50 years [HR 0.697 (95%CI 0.490–0.991), $p = 0.045$].

Discussion

Our data show SO is fairly common in Caucasian morbidly obese females aged >60 years that are candidates to BS. To our knowledge, this is the first study in BS candidates in which definition of SO was based on cut-off values derived from the SSM versus BMI distribution in young adults with comparable BMI distribution to the study population. Prevalence estimates of SO in our cohort did not differ when two commonly used indices of MM were used.

Reports on the prevalence of SO in BS-candidates are scant and preponderantly limited to subjects aged <60 [10–14]. The highly variable prevalence estimates of SO (0–100% in males and 0–84.5% in females) in 120 subjects with morbid obesity using up to 16 indices defining SO from DEXA BC-data reflects the difficulties in interpretating current literature in the field [10]. Of note, at variance to what we are reporting herein, none of the cut-offs used in these studies was derived from young-adult reference populations with class II or III obesity. As lean and fat mass increase with increasing BMI, consideration of control groups with similar BMI distribution of the study group is important when assessing SO [18]. Mastino et al. used the BIA-derived SMI to determine the prevalence of SO in a cohort of 69 BS candidates [11]. Unfortunately, no reference population was used. Rather, SO was ascertained as a SMM index in the lowest tertile within the cohort. In another study by Johnson Stoklossa et al. used cut-off values were based on the association between BC indices and activity disability scores within the same study cohort [12]. Of note, among the different indices used to evaluate SO in BS-candidates in this study, %SMM was indentified as the best BC estimate of the functional component of sarcopenia. Finally, in two independent studies computed tomography estimates of SMM were used in short series of BS-candidates subjects aged <60 years showed prevalence of SO of 8% and 32% respectively [13, 14]. Again, differences in

Table 3 Clinical, and biochemical characteristics of the study participants according to the presence of sarcopenia

	No sarcopenia ($n = 1080$)	Sarcopenia ($n = 290$)	P value
Clinical data			
Age (years)	43.6 (10.8)	48.4 (10.7)	<0.001
Sex (M/F, %)	27.2/72.8	19.7/80.3	0.009
BMI (kg/m^2)	45.6 (5.6)	45.1 (5.5)	0.372
WC (cm)	130.0 (13.8)	130.2 (13.6)	0.823
SBP (mmHg)	133,5 (17.2)	132.9 (15.7)	0.625
DBP (mmHg)	81.0 (11.8)	81.0 (10.1)	0.962
Smoker (yes, %)	26.1	16.6	0.003
T2D (yes,%)	24.8	21.7	0.088
HTN (yes,%)	41.2	50.2	0.006
Dyslipidemia (yes,%)	27.1	31.7	0.122
SAHS (yes,%)	26.0	23.4	0.372
Biochemical data			
hs-CRP (mg/dL)	1.1 (1.0)	1.2 (1.6)	0.300
Glucose (mg/dL)	118.1 (42.0)	116.2 (39.2)	0.490
A1C (%)	6.0 (1.2)	6.0 (1.1)	0.527
Insulin (mU/L)	28.7 (19.4)	25.8 (16.4)	0.064
HDL-C (mg/dL) Male	38.4 (7.9)	40.1	0.156
Female	45.9 (10.6)	49.7	<0.001
Triglycerides (mg/dL)	142.9 (83.1)	128.0 (76.0)	0.006
Total protein (g/L)	73.2 (5.7)	73.1 (5.1)	0.833
Albumin (g/L)	44.1 (3.1)	43.6 (2.7)	0.008

Data are presented as mean (SE) or as percent. M, male; F, female; BMI, body mass index; WC, wasit circumference; SBP: systolic blood pressure; DBP: diastolic blood pressure; T2D: type 2 diabetes; HTN: hypertension; SAHS, sleep apnea-hypopnea syndrome; hs-CRP, high-sensitivity C-reactive protein; A1C, glycated hemoglobin; HDL-C, high-density lipoprotein Cholesterol

prevalence could be attributed to differences in the definition of SO. In the former study, definition of SO was based on sex-specific cut-offs for SMM index associated with mortality in patients with solid tumors of the respiratory and gastrointestinal tracts [19]. In the second study SO was defined as the lowest tertile of SMM index in each gender of the study participants [14].

As MM declines after the forth decade of life [20], it could be argued the larger prevalence of SO in subjects aged >60 in our study was predictable. However, to our knowledge, the association of SO with age in subjects with severe obesity about to undergo BS has not previously been reported. Bouchard et al. reported larger prevalence of sarcopenia in non-obese vs females (23% vs 15%) or males (40% vs 37%) with moderate obesity [21]. On the other hand, Batsis et al. reported a prevalence of 22% and 6.7% respectively in moderately obese females and males aged 60–69 years [22]. In both studies percent body fat measured by DEXA was used as criterion to define obesity, whereas mean BMI of the study participants was in the overweight range. Differences in the reported prevalences of SO in this age group could be attributed to the obesity definition used and/or BMI category of the study subjects.

We also identified female gender as independent predictor of SO. On the contrary, neither C-reactive protein nor comorbidities associated with obesity emerged as independent predictors. Studies on the association between gender and SO have yielded conflicting results [8]. Sex steroids play a role as determinants of MM in both genders, and thus could be involved in the development of sarcopenia. Nonetheless, we acknowledge the number of males in our series was too small to draw definite conclusions on gender differences in the prevalence of SO. Although low-grade inflammation has been associated with SO [23], lack of association in our study could be attributed to the confunding effect of the large contribution of morbid obesity to CRP levels [24]. Finally, differences in the characterstics of study participants may account at least in part for the discordance among studies on the association between SO and obesity-associated comorbidities [7, 25, 26]. However, lack of association between SO and a comorbid state does not rule out the potential role of impaired MM in the pathogenesis of the evaluated comorbidities. Indeed, muscle function rather than muscle mass has recently been associated with the presence of the metabolic syndrome in adult women with obesity [26].

We acknowledge lack of BS-outcome data of our cohort is a major limitation of our study. Mastino et al. reported weight loss and improvement of obesity-related comorbidities up to 1 year after BS in a cohort of 69 subjects did not differ between SO and non-SO subjects [11]. On the other hand, Gaillard et al. showed pre-op SO was associated with the occurrence of gastric leak after sleeve gastrectomy [14]. Furthermore, lower skeletal muscle mass (SMM) and lower hand grip strength prior to BS have been identified as predictors of poorer MM retention after surgery [13, 27]. Importantly, poorer MM and poorer muscle function have been identified as predictors of mortality [22, 28]. Thus, although available data on the use of BS in older adults suggest outcomes do not differ from those in younger subjects additional data with longer follow up is needed to establish the safety and efficacy of BS in the subgroup of elderly patients with SO.

Additional limitations of our study include the use BIA use as method for BC analysis, and lack of consideration of physical function in our definition of sarcopenia [5]. However, BIA is currently accepted as a non-invasive, affordable, and simple method to assess BC in the field of sarcopenia [5, 29]. Studies including physical function in elderly morbidly obese subjects are warranted. However, within its limitations, we deem our data adds to the field since SMM indexes adjusted for body mass as in our study have been found to be the best correlates with performance of activities physical function [12].

In summary, our data show SO is fairly common in subjects aged >60 years that are candidates to BS. Additional studies are needed to establish the clinical relevance of our findings, and the potential need for nutritional and exercise programs to prevent the potential detrimental consequences of SO in subjects undergoing BS.

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Compliance with Ethical Standards

Conflict of Interest The authors of this manuscript have no personal or financial conflicts of interest to declare.

Statement of Authorship and Ethics The individual contribution of the authors to this manuscript was as follows: (1) the conception and design of the study, or acquisition of data, or analysis and interpretation of data: JM, LF, JV; (2) drafting the article or revising it critically for important intellectual content: JM, LF, VM, AA, AdH, AJ, JV; (3) final approval of the version to be submitted: JM, LF, VM, AA, AdH, AJ, JV. We alse declare the present work complies with all requirements from the Committee on Publication Ethics.

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