ORIGINAL CONTRIBUTIONS





Effects of Bariatric Surgery on Sarcopenic Obesity Outcomes: A One-Year Prospective Study in Middle-Aged Women

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Abstract

Introduction Sarcopenic obesity (SO) is characterised by the confluence of muscle deterioration and high adiposity. When non-surgical interventions prove insufficient, bariatric surgery (BS) becomes the primary approach. This study aimed to address BS effects on SO outcomes 1 year post-surgery among middle-aged women, also considering physical exercise's impact.

Methods Prospective single-centre study of 140 patients who underwent Roux-en-Y gastric bypass or sleeve gastrectomy between November 2019 and December 2022. Participants were categorised into tertiles according to SO's diagnosis and severity (group 1—patients with the most severe SO; group 2—intermediate; group 3—the least severe or without SO), calculated considering the consensus issued by ESPEN and EASO in 2022. Evaluations of clinical and biochemical parameters were conducted before and 12 months after BS, and the variation was used for comparative purposes. Body composition was assessed using bone density scans. Linear regression analysis accounted for both surgery type and baseline body mass index (BMI).

Results Before BS, SO prevalence in the overall sample was 89.3%, decreasing to 2.9% after BS. Group 1 had more body fat mass (56.9 vs 54.8 vs 50.7 kg, p < 0.001), total, trunk and leg fat at baseline and a significantly lower total skeletal muscle mass (47.2 vs 49.4 vs 51.8 kg, p < 0.001). One year post-BS, group 1 presented more weight loss (-39.8 ± 11.4 kg, p = 0.031), BMI reduction (-15.9 ± 4.6 kg/m², p = 0.005) and lost more fat mass (-32.6 vs -30.5 vs -27.9 kg, p = 0.005), but not total skeletal muscle mass (-5.8 vs -5.9 vs -6.8 kg, p = 0.130). Remission rates for comorbidities were substantial among all groups, but more marked among patients within group 1 (type 2 diabetes mellitus 75%, hypertension 47.1% and dyslipidemia 52.8%). Engagement in physical exercise of any kind has increased post-BS (33.1% vs 79.1%).

Conclusion Despite concerns about malabsorptive mechanisms potentially worsening muscle loss, patients with the most severe SO undergoing BS lost more fat mass while experiencing the smallest reduction in total skeletal muscle mass. Remission rates for comorbidities following BS were notable among all groups.

Keywords Bariatric surgery · Sarcopenic obesity · Metabolic outcomes · Muscle mass · Weight loss · Physical exercise

Pietra Rodrigues and Fernando Mendonça are both co-first authors and they contributed equally to this work.

Key Points

- Bariatric surgery emerges as both a risk factor and a management approach in sarcopenic obesity.
- Patients with severe sarcopenic obesity lost more fat mass and less muscle mass after bariatric surgery.
- Bariatric surgery reduces sarcopenic obesity's prevalence and improves comorbidities.

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Introduction

Sarcopenic obesity (SO), a condition initially described by Baumgartner in 2006 [1], has garnered considerable attention from the medical community in recent years. The confluence of muscle deterioration, encompassing low muscle mass and/or strength loss, and high adiposity, creates a synergistic health impact that transcends the consequences of either of these conditions, and that is the main reason for its increasing recognition as an entity itself. Regrettably, the absence of a universal consensus on diagnostic criteria has compounded the challenges faced in approaching this condition. To answer this problem, the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) jointly issued a consensus statement addressing SO's definition, diagnostic procedures, and established cut-off values for diverse ethnic groups, sexes and age categories. This effort was undertaken in response to the limitations of the previously widely used consensus, the Revised European Working Group on Sarcopenia in Older People (EWGSOP2), which solely focused on an older population and was designed exclusively to address sarcopenia [2–4].

SO is strongly associated with a plethora of adverse health outcomes and comorbidities, including atherosclerosis, insulin resistance (IR), type 2 diabetes mellitus (DM2), hypertension (HT), dyslipidemia, metabolic syndrome, nonalcoholic fatty liver disease (NAFLD), heart failure (HF), osteoporosis and a higher mortality rate especially in women [5–7], bearing a substantial economic and biopsychological burden.

Studies suggest a reciprocal relationship between skeletal muscle and adipose tissue, exacerbated by factors like aging, malnutrition and sedentary lifestyle, triggering chronic inflammation, oxidative stress and IR in adipose tissue, which ultimately leads to muscle apoptosis, fat accumulation [8] and compromised bone health [9].

The management of SO should be approached in a multidisciplinary perspective, with dietary guidance and a combination of aerobic and resistance exercises constituting the primary intervention. However, the efficacy of these measures in the long term has, thus far, proven less than promising [10]. When both lifestyle modifications and medical therapies fall short, bariatric surgery (BS) emerges as the most viable option for morbidly obese individuals who meet the criteria, as it addresses the health-related comorbidities of SO [11]. Conversely, it has been proposed that bariatric surgery may have detrimental muscle effects in obese patients with diminished muscle mass or function, primarily due to the malabsorptive mechanisms involved, which are risk factors to SO, although its impact in women under the age of 65 remains relatively underexplored [3].

Thus, this study aims to assess the effects of BS on SO outcomes within a 1-year follow-up period in women, considering their metabolic parameters, the remission of multiple comorbidities (such as HT, DM2 and dyslipidemia) and the possible impact of physical exercise in SO's management.

Materials and Methods

Subjects and Study Design

This single-centre study employed a prospective design and involved the initial recruitment of 140 female participants who had undergone Roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG), consecutively selected from November 2019 to December 2022 during pre-operative evaluation by the Integrated Responsibility Center for Obesity (CRIO group). The eligibility criteria for this study were the following: (a) women with obesity, aged 40–65 years; (b) meeting the criteria for BS, which included having a body mass index (BMI) of $\geq 35 \text{ kg/m}^2$ with at least one comorbidity such as metabolic syndrome, DM2, dyslipidemia, HT or cardiorespiratory disease, among others, or BMI of $\geq 40 \text{ kg/m}^2$ despite their comorbidities; (c) at least 1 year of non-surgical approaches to obesity, which had not resulted in satisfactory weight loss; and (d) obesity not attributable to classical endocrine diseases.

The exclusion criteria encompassed the following: (a) second or revisional BS; (b) weight exceeding 140 kg, the maximum weight supported by our dual-energy X-ray absorptiometry (DXA) equipment; (c) non-stabilized psychiatric illnesses or narcotic or alcohol dependence; (d) pharmacological therapy that could impact muscle mass, such as corticosteroids; and (e) having a severe clinical condition, including cardiovascular, pulmonary, hepatic, renal, osteo-articular and malignant neoplasms.

The choice of surgery technique was determined by the surgical team according to the best clinical practice. The study received approval from the ethics committee for health of our hospital centre (reference no. 276/2018).

Sarcopenic Obesity Classification and Cut-off Points

The definition and diagnosis of SO were based on the 2022 consensus proposed by ESPEN and EASO, which provides specific cut-off values suitable for our study population [3]. Body composition was assessed using DXA, with an increased percentage of fat mass (FM), considered as > 43% for Caucasian women [3, 12]. As for the reduced muscle mass, it was determined by the sum of the lean mass of arms and legs (appendicular lean mass (ALM)) adjusted to body weight (W) in accordance with the consensus recommendation for Caucasian women aged 18–65 years, which stipulates an ALM/W ratio < 23.47% [3, 13]. As an additional analysis, we also evaluated the study population according to tertiles of ALM/W (group 1—patients with the most severe SO; group 2—intermediate; group 3—the least severe or without SO).

Pre- and One Year Post-operative Evaluation

All evaluations were conducted both before and 12 months after bariatric surgery. The variation between those values was used for comparison between groups. Body composition was assessed by DXA using a properly calibrated densitometer (model Lunar iDXA). The studied parameters were body fat, proportion of total fat mass, trunk fat mass and leg fat mass, arm lean mass, leg lean mass and bone mineral content (BMC). Other assessed paraments were bone mineral density, T score, Z score at the lumbar spine (L1–4), total femur, femoral neck and full body.

Anthropometric measurements were taken, including body weight, height, waist circumference and BMI. Blood pressure was measured during their Endocrinology appointments, with the patient seated for at least 5 min. Blood samples were collected after a fasting period of at least 12 h. Biochemical parameters analysed in our study included albumin, serum total protein (STP), total cholesterol (CT), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglycerides (TG), gammaglutamyltransferase (GGT), alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), iron, transferrin, ferritin, folic acid, vitamin B12, 25-OH-vitamin-D, total calcium, ionized calcium, phosphorus, magnesium (Mg), parathyroid hormone (PTH), thyroid-stimulating hormone (TSH), free thyroxine (T4L), fasting glucose (FG), insulin, hemoglobin A1c (HbA1c) and microalbuminuria. IR was assessed using the homeostasis model assessment for insulin resistance (HOMA-IR). Estimated glomerular filtration rate (eGFR) was calculated for all subjects using the 4-variable modification of diet in renal disease (MDRD) formula.

Information regarding medical history and comorbidities, medications in use, menopause history and supplementation prior and post-surgery was collected based on medical records. Physical exercise was retrospectively collected after surgery and included the type (aerobic, resistance training or both) and duration of activity (in minutes per week) before and after surgery.

The study also evaluated the presence of DM2, HT and dyslipidemia, both at baseline and after a 1-year follow-up period. DM2 was defined by a fasting glucose level ≥ 126 mg/dL or HbA1c level $\geq 6.5\%$ or antidiabetic medication. HT was characterized by a systolic blood pressure (SBP) ≥ 140 mmHg and/or a diastolic blood pressure (DBP) ≥ 90 mmHg. The current use of antihypertensive medication was also diagnostic. Dyslipidemia was diagnosed by LDL ≥ 130 mg/dL, HDL ≤ 50 mg/dL, TG ≥ 150 mg/dL or CT ≥ 240 mg/dL. Participants under medication with statins or fibrates were also considered as dyslipidemic.

Remission of any of these comorbidities was considered when a patient diagnosed with a comorbidity during the preoperative period no longer met the diagnostic criteria 1 year post-bariatric surgery.

Statistical Analysis

Continuous variables with normal distribution were presented as means and standard deviations, being compared using one-way ANOVA. Continuous variables with nonnormal distribution were described as medians and interquartile range, being compared using Kruskal-Wallis test. Categorical variables were expressed as percentages, being compared using a χ^2 test. Reported p values are two-tailed, and $p \leq 0.05$ was considered statistically significant. Linear regression models unadjusted and adjusted for surgery type (SG vs RYBG) and baseline BMI were performed to evaluate the predictors of outcomes at 12 months. Analyses were conducted with the use of STATA 18.0® software.

Results

A total of 140 female participants, with a mean age of 50.9 years old, mean weight of 107.9 kg and mean BMI of 42.1 kg/m², were enrolled in the study. The prevalence of SO, determined by specific cut-offs outlined by the ESPEN and EASO's consensus, was 89.3% in the overall sample before surgery and decreased to 2.9% 1 year post-BS. Most participants underwent RYGB (n = 100, 71.4%). Patient stratification into three groups was conducted, with group 1 being defined as the lowest tertile of subjects (the most severe degree of SO). The baseline characteristics of both the overall sample and the three individual groups are presented in Table 1.

Only 33.1% of participants engaged in any form of physical exercise before surgery, while 79.1% were doing some type of exercising after the follow-up period. The duration of their exercise regimen, measured in minutes per week, exhibited an increase across all groups following surgery, although without statistical significance. Notably, both before and after BS, group 1 individuals allocated more time per week to augment their physical exercise levels, when compared to the other tertiles (Supplementary Table 1). However, concerning the nature of physical exercise 1 year post-BS, only 20.1% of the total sample engaged in both aerobic and resistance training. This still marked an improvement from the baseline rate (6.5%). Remarkably, group 1 showed a tendency towards increased engagement in combined aerobic and resistance training compared to the other groups (23.4% vs 19.6% vs 17.4%, p = 0.090), yet most participants who exercised primarily focused on solo aerobic exercises (54.0%).

Anthropometric parameters, blood pressure and body composition at baseline and 1 year after BS are presented in Table 2. At baseline, group 1 exhibited an increased proportion of total fat, trunk fat and leg fat compared to other groups, the same occurring with body fat mass (56.9 ± 8.8 vs 54.8 ± 7.2 vs 50.7 ± 8.4 kg, p < 0.001). Group 1 also demonstrated significantly lower total skeletal muscle mass (47.2 ± 5.5 vs 49.4 ± 5.0 vs 51.8 ± 5.8 kg, p < 0.001). Regarding bone parameters on DXA, none were statistically

Variable	Overall sample ($n = 140$)	Group 1 ($n = 47$)	Group 2 ($n = 47$)	Group 3 ($n = 46$)	Pvalue
Age*	50.9 ± 7.0	51.9 ± 7.3	50.7 ± 6.7	50.2 ± 7.1	0.250
Weight (kg)*	107.9 ± 13.2	109.1 ±14.7	108.6 ± 11.7	106.0 ± 13.2	0.270
BMI (kg/m ²)*	42.1 ± 4.6	43.4 ± 5.6	42.1 ± 3.6	40.6 ± 3.9	0.004
Waist circumference (cm)*	118.5 ± 10.5	121.4 ± 8.2	121.2 ± 10.2	113.9 ± 11.0	0.023
Surgical procedure					0.600
RYGB, <i>n</i> (%)	100 (71.4)	34 (72.3)	35 (74.5)	31 (67.4)	
SG, n (%)	40 (28.6)	13 (27.7)	12 (25.5)	15 (32.6)	
Sarcopenic obesity, n (%)	125 (89.3)	47 (100.0)	47 (100.0)	31 (67.4)	<0.001
SBP (mmHg)	140.3 ± 16.7	140.7 ± 20.4	140.6 ± 16.9	139.7 ± 13.3	0.830
DBP (mmHg)	86.5 ± 10.8	86.0 ± 11.3	84.8 ± 11.2	88.8 ± 9.7	0.330
Menopause, n (%)	72 (51.4)	26 (55.3)	25 (53.2)	21 (45.7)	0.360
DM2, <i>n</i> (%)	41 (29.3)	12 (25.5)	11 (23.4)	18 (39.1)	0.150
HT, <i>n</i> (%)	98 (70.0)	34 (72.3)	31 (66.0)	33 (71.7)	0.950
Dyslipidemia, n (%)	110 (78.6)	36 (76.6)	38 (80.9)	36 (78.3)	0.840

BMI body mass index, RYGB Roux-en-Y gastric bypass, SG vertical sleeve gastrectomy, SBP systolic blood pressure, DBP diastolic blood pressure, DM2 type 2 diabetes mellitus, HT hypertension

*Data are expressed as mean ± SD

significant 1 year after BS, although group 1 displayed slightly diminished BMC at baseline $(2.5 \pm 0.3 \text{ vs } 2.6 \pm 0.3 \text{ vs } 2.7 \pm 0.3 \text{ g}, p = 0.004)$. One year after BS, there has been an anthropometric difference between groups regarding both their weight (p = 0.031), showing a mean reduction of 39.8 kg in group 1, and BMI (p = 0.005), with a mean reduction of 15.9 kg/m² in this same group. Although all groups exhibited decreased fat-related parameters, only body fat mass, leg fat (%)and both arm and leg lean masses reached statistical significance. Notably, group 1 lost more fat mass compared to other groups ($-32.6 \pm 8.6 \text{ vs} - 30.5 \pm 7.3 \text{ vs} - 27.9 \pm$ 7.6 kg, p = 0.005), but did not experience a greater reduction in their total skeletal muscle mass. In fact, group 1 had a mean variation of -5.8 kg of total skeletal muscle mass (vs -5.9 vs -6.8 kg, p = 0.130).

To mitigate potential disparities in our primary body composition findings that may stem from the type of surgery conducted, the authors conducted a comparative analysis of the 1-year variation in weight, BMI, fat and lean mass induced by restrictive and malabsorptive surgeries across the entire sample. Patients undergoing RYGB displayed more pronounced reductions in all parameters. However, statistical significance was observed solely in the differences in fat mass when comparing SG and RYGB (-26.7 ± 9.1 vs -31.9 ± 7.0 , $p \le 0.001$) (Supplementary Table 2).

A comparison of laboratory parameters has been performed at Table 3. At baseline, group 1 had a higher eGFR compared to the overall sample (mean difference of 5.6 mL/ min/1.73 m², p = 0.019), and so did TSH, although within the physiological range (2.6 ± 3.0 vs 1.7 ± 0.8 vs 1.6 ± 0.7, p = 0.017). After 1 year, both Mg and TSH showed statistically significant changes, with group 1 exhibiting a larger decrease in TSH values ($-1.0 \pm 3.0 \mu \text{UI/mL}, p = 0.006$), but no differences were found regarding T4L (p = 0.610). There was improvement in CT, HDL, LDL and TG levels in all groups, with group 1 showing the most significant improvement in CT levels (although being non-statistically significant). Regarding FG, HbA1c, HOMA-IR and insulin, all groups presented some metabolic improvement, but also not enough to be considered significant. As for iron's metabolism, there has been an increase after surgery in iron and ferritin levels, both being slightly more pronounced in the most severe group. AST showed increased levels in all groups after surgery when compared to baseline, especially in group 1 (7.1 \pm 15.8 vs 3.9 \pm 19.1 vs 0.4 \pm 12.7, p = 0.05). During the follow-up period, there has been an increase in vitamin B12 and 25-OH-vitamin-D levels within group 1.

A detailed breakdown of multivitamin and mineral supplement usage is presented in Supplementary Table 3, providing a better insight into these findings. Of participants in group 1, 42.6% were receiving 25-OH-vitamin-D supplementation following surgery, while 19.1% were under vitamin B12 supplementation. None of the variables related to supplement usage exhibited statistical significance.

Table 4 presents the assessment of comorbidities 1 year post-surgery, indicating a notable remission rate for DM2, HT and dyslipidemia in all groups, regardless of the absence of statistically significant differences between them. Despite this fact, there was a tendency for increased dyslipidemia remission in group 1, with a remission rate of 52.8%

Overa	Baseline				Pvalue	Variation one year after BS			Pvalue
	Overall sample $(n = 140)$	Group 1 (<i>n</i> = 47)	Group 2 (<i>n</i> = 47)	Group 3 (<i>n</i> = 46)		Group 1 (<i>n</i> = 47)	Group 2 (<i>n</i> = 47)	Group 3 (<i>n</i> = 46)	
Weight (kg)	107.9 ± 13.2	109.1 ±14.7	108.6 ± 11.7	106.0 ± 13.2	0.270	-39.8 ± 11.4	-38.3 ± 9.6	-35.3 ± 9.1	0.031
BMI (kg/m ²)	42.1 ± 4.6	43.4 ± 5.6	42.1 ± 3.6	40.6 ± 3.9	0.004	-15.9 ± 4.6	-14.8 ± 3.5	-13.6 ± 3.4	0.005
SBP (mmHg)	140.3 ± 16.7	140.7 ± 20.4	140.6 ± 16.9	139.7 ± 13.3	0.830	-7.8 ± 21.4	-9.8 ± 28.5	-17.3 ± 15.8	0.250
DBP (mmHg)	86.5 ± 10.8	86.0 ± 11.3	84.8 ± 11.2	88.8 ± 9.7	0.330	-13.0 ± 17.7	-12.1 ± 16.1	-19.3 ± 14.1	0.250
BMC (g)	2.6 ± 0.3	2.5 ± 0.3	2.6 ± 0.3	2.7 ± 0.3	0.004	-0.2 ± 0.1	-0.2 ± 0.1	-0.2 ± 0.1	0.120
Body fat mass (kg)	54.2 ± 8.5	56.9 ± 8.8	54.8 ± 7.2	50.7 ± 8.4	<0.001	-32.6 ± 8.6	-30.5 ± 7.3	-27.9 ± 7.6	0.005
Total body fat (%)	52.2 ± 3.5	54.6 ± 2.6	52.6 ± 2.8	49.3 ± 3.0	<0.001	-18.3 ± 6.6	-17.5 ± 6.2	-16.3 ± 5.9	0.130
Trunk fat (%)	55.9 ± 3.8	58.1 ± 2.9	56.3 ± 3.2	53.3 ± 3.6	< 0.001	-22.5 ± 8.4	-22.1 ± 8.2	-20.8 ± 7.7	0.340
Legs fat (%)	49.2 ± 5.5	52.0 ± 5.0	49.8 ± 4.9	45.7 ± 4.8	<0.001	-14.1 ± 5.4	-12.6 ± 5.1	-11.3 ± 4.9	0.012
Total skeletal muscle mass (kg)	49.4 ± 5.7	47.2 ± 5.5	49.4 ± 5.0	51.8 ± 5.8		-5.8 ± 2.5	-5.9 ± 3.2	-6.8 ± 2.9	0.130
Arms lean mass (kg)	5.5 ± 1.2	4.7 ± 0.9	5.7 ± 1.2	6.0 ± 1.0	<0.001	-0.3 ± 0.8	-0.9 ± 1.2	-0.9 ± 0.8	0.010
Legs lean mass (kg)	17.4 ± 2.6	16.3 ± 2.3	17.4 ± 2.1	18.7 ± 2.8	<0.001	-3.0 ± 1.2	-3.2 ± 1.4	-3.8 ± 1.6	0.006
L1-L4 BMD (g/cm ²)	1.2 ± 0.2	1.2 ± 0.1	1.2 ± 0.2	1.2 ± 0.1	0.620	-0.0 ± 0.1	-0.1 ± 0.1	-0.1 ± 0.1	0.330
L1-L4 T-score	0.3 ± 1.2	0.3 ± 1.1	0.3 ± 1.3	0.3 ± 1.1	0.700	-0.4 ± 0.6	-0.5 ± 0.5	-0.5 ± 0.6	0.540
L1-L4 Z-score	-0.0 ± 1.2	-0.0 ± 1.1	-0.1 ± 1.4	-0.1 ± 1.2	0.840	0.7 ± 0.6	0.6 ± 0.6	0.5 ± 0.6	0.090
Femoral neck BMD (g/ cm ²)	1.0 ± 0.1	1.0 ± 0.1	1.0 ± 0.1	1.1 ± 0.1	0.130	-0.1 ± 0.1	-0.1 ± 0.1	-0.1 ± 0.1	0.910
Femoral neck <i>T</i> -score	0.5 ± 1.1	0.4 ± 1.1	0.2 ± 1.0	0.8 ± 1.2	0.110	-0.3 ± 0.9	-0.5 ± 1.1	0.0 ± 0.8	0.060
Femoral neck Z-score	0.2 ± 1.0	-0.0 ± 1.1	-0.1 ± 1.4	-0.1 ± 1.2	0.840	0.3 ± 0.9	0.0 ± 0.9	0.4 ± 0.8	0.550
Total femur BMD (g/ cm ²)	1.2 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	0.220	-0.1 ± 0.1	-0.1 ± 0.1	-0.1 ± 0.1	0.570
Total femur <i>T</i> -score	1.5 ± 1.2	1.5 ± 1.2	1.3 ± 1.2	1.8 ± 1.0	0.200	-0.9 ± 0.5	-1.0 ± 0.5	-1.0 ± 0.5	0.290
Total femur Z-score	1.0 ± 1.1	1.0 ± 1.1	0.8 ± 1.2	1.2 ± 0.9	0.400	-0.1 ± 0.4	-0.2 ± 0.5	-0.3 ± 0.6	0.060
Total body BMD (g/ cm ²)	1.2 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	0.970	-0.1 ± 0.1	-0.1 ± 0.0	-0.0 ± 0.1	0.640
Total body <i>T</i> -score	1.8 ± 1.1	1.8 ± 1.1	1.8 ± 1.1	1.8 ± 1.0	0.970	-0.5 ± 0.6	-0.6 ± 0.4	-0.5 ± 0.5	0.490
Total body Z-score	0.5 ± 1.0	0.6 ± 1.0	0.5 ± 1.0	0.5 ± 0.9	0.750	0.8 ± 0.6	0.7 ± 0.6	0.7 ± 0.8	0.310

 Table 2
 Comparative analysis of anthropometric parameters, blood pressure and body composition assessed by DXA at baseline and 1 year after BS

All data in this table are expressed as mean \pm SD

BS bariatric surgery, BMI body mass index, SBP systolic blood pressure, DBP diastolic blood pressure, BMC bone mineral content, L1–L4 lumbar spine from 1 to 4, BMD bone mineral density

Table 3 Comparative analysis of laboratory parameters at baseline and 1 year after BS

Variable	Baseline			value	Variation 1 year after BS		value		
Overall sample $(n = 140)$		Group 1 (<i>n</i> = 47)	Group 2 (<i>n</i> = 47)	Group 3 (<i>n</i> = 46)		Group 1 (<i>n</i> = 47)	Group 2 (<i>n</i> = 47)	Group 3 (<i>n</i> = 46)	
STP (g/L)*	70.6 ± 4.5	70.6 ± 3.9	69.8 ± 4.8	71.4 ± 4.7	0.410	-4.8 ± 3.6	-4.1 ± 4.5	-3.1 ± 4.2	0.060
Albumin (g/L)*	40.6 ± 2.5	40.4 ± 2.0	40.7 ± 3.1	40.8 ± 2.3	0.460	-0.6 ± 2.1	-0.8 ± 2.7	-0.5 ± 2.0	0.910
FG (mg/dL)**	103.0 [94.0, 119.0]	106.0 [98.0, 118.0]	101.0 [93.0, 110.0]	112.0 [94.0, 123.0]	0.660	-19.3 ± 32.3	-7.8 ± 32.0	-26.2 ± 47.7	0.410
HbA1c (%)**	5.8 [5.5, 6.0]	5.6 [5.4, 5.9]	5.9 [5.6, 6.1]	5.7 [5.5, 6.0]	0.520	-0.6 ± 0.6	-0.5 ± 0.4	-0.7 ± 1.1	0.340
HOMA-IR**	5.1 [3.1, 7.1]	5.1 [2.3, 6.5]	4.8 [3.3, 7.1]	5.2 [3.2, 9.4]	0.160	-3.4 ± 3.7	-3.6 ± 2.6	-5.7 ± 12.3	0.150
Insulin (µU/ mL)**	19.6 [13.0, 25.7]	17.9 [9.2, 23.8]	19.6 [13.1, 26.4]	20.5 [13.8, 27.6]	0.130	-11.0 ± 11.6	-13.4 ± 8.7	-16.6 ± 29.2	0.150
CT (mg/dL)*	190.8 ± 46.2	189.0 ± 38.7	188.3 ± 35.5	195.4 ± 61.8	0.520	-25.1 ± 33.2	-23.4 ± 34.8	-20.7 ± 53.9	0.620
HDL (mg/dL)*	51.6 ± 11.2	51.8 ± 9.3	51.5 ± 12.1	51.3 ± 12.3	0.810	4.0 ± 6.7	3.6 ± 7.2	6.1 ± 9.8	0.220
LDL (mg/dL)*	114.1 ± 36.6	111.0 ± 32.1	114.3 ± 26.6	117.0 ± 48.9	0.440	-19.9 ± 30.1	-21.3 ± 29.7	-17.2 ± 42.2	0.720
TG (mg/dL)**	109.0 [83.0, 154.0]	120.0 [87.0, 151.0]	102.0 [77.0, 126.0]	109.0 [87.0, 170.0]	0.920	-47.1 ± 51.9	-28.7 ± 46.2	-59.2 ± 89.0	0.400
GGT (U/L)*	25.0 [18.0, 38.0]	26.0 [18.0, 44.0]	23.5 [16.0, 34.0]	25.0 [18.0, 38.0]	0.780	-9.2 ± 43.9	-10.0 ± 10.8	-27.5 ± 98.0	0.170
ALP (U/L)*	78.0 [65.0, 95.0]	78.5 [65.0, 95.0]	79.0 [61.0, 100.0]	75.0 [65.0, 93.0]	0.960	0.5 ± 22.4	0.2 ± 19.4	-5.4 ± 19.0	0.180
AST (U/L)*	20.0 [17.0, 26.0]	19.0 [16.0, 28.0]	21.0 [18.0, 26.0]	20.5 [16.0, 27.0]	0.430	7.1 ± 15.8	3.9 ± 19.1	0.4 ± 12.7	0.050
ALT (U/L)*	22.0 [16.0, 29.5]	19.5 [16.0, 28.0]	23.0 [19.0, 30.0]	22.0 [16.0, 30.0]	0.540	7.8 ± 23.9	5.7 ± 35.5	-0.7 ± 16.9	0.140
eGFR (mL/ min/1.73 m ²)*	108.7 ± 24.4	114.3 ± 31.6	109.0 ± 18.4	102.2 ± 19.8	0.019	-5.9 ± 23.8	0.2 ± 16.9	1.5 ± 13.5	0.060
Microalbuminu- ria (mg/L)**	8.7 [4.6 ,20.8]	13.6 [4.8, 23.0]	9.0 [4.7, 23.8]	6.8 [4.3, 15.0]	0.120	-12.0 ± 51.2	1.1 ± 66.2	-7.1 ± 28.9	0.660
Iron (µg/dL)*	77.5 ± 27.0	76.0 ± 23.8	82.0 ± 27.3	74.0 ± 30.0	0.780	8.6 ± 30.0	0.2 ± 33.8	1.5 ± 13.5	0.060
Transferrin (mg/ dL)*	275.0 ± 43.6	271.2 ± 42.5	266.6 ± 40.2	288.9 ± 46.3	0.070	-37.3 ± 34.0	-29.2 ± 34.5	-43.1 ± 63.9	0.590
Ferritin (ng/ mL)**	77.5 [39.7, 169.9]	80.8 [50.0, 160.1]	82.6 [40.7, 180.8]	65.9 [29.6, 173.5]	0.450	28.3 ± 53.7	13.7 ± 64.2	12.3 ± 66.2	0.220
Folic acid (ng/ mL)*	6.6 ± 3.5	6.8 ± 4.4	6.9 ± 3.4	6.1 ± 2.4	0.390	5.0 ± 5.5	5.0 ± 5.2	5.8 ± 4.9	0.540
Vitamin B12 (pg/mL)*	388.0 ± 215.4	378.3 ± 270.2	387.8 ± 129.3	399.1 ± 228.0	0.650	20.9 ± 221.4	-76.3 ± 156.6	-48.8 ± 229.6	0.100
Vitamin D (ng/ mL)*	19.7 ± 8.6	19.2 ± 7.5	19.0 ± 8.2	21.0 ± 10.1	0.330	10.1 ± 13.7	9.0 ± 12.3	7.5 ± 12.4	0.330
$P+(mg/dL)^*$	3.3 ± 0.5	3.4 ± 0.5	3.3 ± 0.5	3.3 ± 0.4	0.590	0.4 ± 0.5	0.7 ± 0.5	0.6 ± 0.5	0.190
Total Ca (mEq/L)*	4.7 ± 0.2	4.7 ± 0.1	4.7 ± 0.2	4.7 ± 0.2	0.990	0.0 ± 0.2	0.0 ± 0.2	0.1 ± 0.2	0.200
Ionized Ca (mEq/L)*	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.1	0.810	0.0 ± 0.1	0.0 ± 0.1	0.0 ± 0.2	0.680
Mg (mEq/L)*	1.6 ± 0.1	1.6 ± 0.1	1.6 ± 0.2	1.6 ± 0.1	0.740	-0.0 ± 0.1	0.0 ± 0.1	0.1 ± 0.1	0.003
PTH (pg/mL)*	56.2 ± 21.5	54.9 ± 21.1	58.0 ± 17.6	55.6 ± 25.6	0.860	-4.0 ± 20.4	-8.3 ± 16.9	-8.4 ± 21.0	0.280
T4L (ng/dL)*	0.9 ± 0.1	0.9 ± 0.1	1.0 ± 0.1	0.9 ± 0.1	0.980	0.1 ± 0.1	0.0 ± 0.1	0.0 ± 0.1	0.610
TSH (µUI/mL)*	2.0 ± 1.9	2.6 ± 3.0	1.7 ± 0.8	1.6 ± 0.7	0.017	-1.0 ± 3.0	-0.2 ± 0.7	0.1 ± 1.2	0.006

BS bariatric surgery, STP serum total protein, FG fasting glucose, HbA1c hemoglobin A1c, HOMA-IR homeostatic model assessment for insulin resistance, CT total cholesterol, HDL high-density lipoprotein cholesterol, LDL low-density lipoprotein cholesterol, TG triglycerides, GGT gamma-glutamyltransferase, ALP alkaline phosphatase, AST aspartate aminotransferase, ALT alanine aminotransferase, eGFR estimated glomerular filtration rate, P+ phosphorus, Ca plasmatic calcium, Mg magnesium, PTH parathyroid hormone, T4L free thyroxine, TSH thyroid-stimulating hormone

*Data in this table are expressed as mean ± SD

**Data in this table are expressed as **median [min, max]

Table 4Assessment ofcomorbidity remission 1 yearafter BS

Variable	Remission one-year after BS			
	Group 1	Group 2	Group 3	
DM2 remission, <i>n</i> after/ <i>n</i> baseline (%)*	9/12 (75.0)	5/11 (45.5)	13/18 (72.2)	0.990
HT remission, <i>n</i> after/ <i>n</i> baseline $(\%)^*$	16/34 (47.1)	14/31 (45.2)	23/33 (69.7)	0.070
Dyslipidemia remission, <i>n</i> after/ <i>n</i> baseline (%)*	19/36 (52.8)	15/38 (39.5)	12/36 (33.3)	0.100

BS bariatric surgery, DM2 type 2 diabetes mellitus, HT hypertension

*The symbol '/' represents a fraction used to denote the ratio between the number of individuals with the disease at the 1-year follow-up evaluation and the baseline number

 Table 5
 Linear regression analysis adjusted to BMI and type of BS surgery

Variable	95.0% Confidence interval					
	Coefficient	P value	Lower bound	Upper bound		
Weight (kg)	.206	0.802	- 1.418	1.830		
BMI (kg/m ²)	.307	0.312	292	.906		
Mg (mEq/L)	.038	0.003	.0130	.064		
TSH (µUI/mL)	.580	0.007	.160	1.000		
Body fat mass (kg)	1.212	0.090	190	2.615		
Leg fat (%)	1.283	0.020	.200	2.364		
Arm lean mass (kg)	383	<0.001	579	188		
Leg lean mass (kg)	564	<0.001	837	292		

BS bariatric surgery, BMI body mass index, Mg magnesium, TSH thyroid-stimulating hormone

compared to 39.5% and 33.3% in the other two groups (p = 0.100).

After linear regression analysis adjustment for baseline BMI and type of surgery, only Mg, TSH, leg fat (%) and both lean masses remained statistically significant 1 year after BS (Table 5).

Discussion

In the present study, we aimed to evaluate the influence of bariatric surgery on sarcopenic obesity outcomes after a 1-year follow-up period in middle-aged women.

The consensus by ESPEN and EASO highlights that severe obesity correlates strongly with chronic non-communicable diseases. This correlation significantly impacts muscle anabolism and catabolism, with sedentarism being both a cause and a consequence of SO [3]. Managing these cases requires a multidisciplinary approach, where BS stands out for weight loss after failed non-surgical attempts. However, weight reduction post-BS often leads to muscle mass loss, especially in patients with additional health issues, which could worsen SO outcomes. Interestingly, the prevalence of SO seems consistent across different age groups, emphasizing the imperative need for universal management strategies [14].

To address SO after BS, careful monitoring of nutrition, suitable supplementation and tailored exercise programs are crucial. Yet, the main challenge lies in the varying definitions, thresholds and the absence of consensus on ideal body composition assessment methods [15-18]. This lack of clarity not only complicates diagnosis for physicians but also hampers the comparison of research findings, crucial for advancing global understanding and care for SO patients. The results of our study demonstrate that SO's prevalence is enormous among participants prior to BS, decreasing substantially after surgery. Our results contrast with some previous studies. For example, Sousa-Santos et al. [19] reported rates of 4.4% in Portugal using EWGSOP2 criteria, primarily observed in an older population. However, when comparing our study results with more recent studies using the 2022 ESPEN and EASO consensus, with cut-offs well determined for women within our age range and ethnicity, our results align more closely. An abstract published by Esposito et al. in April 2023, attempting to develop a valid SO screening tool for BS candidates, identified 99.4% of female participants as having SO [20].

Bariatric surgery did not have a deleterious impact in SO outcomes after a 1-year follow-up, as it could have had regarding worsening muscle mass loss in those patients. In fact, it helped induce SO remission in most participants, improving their body composition and metabolic parameters and mitigating comorbidities. Our follow-up period was decided based on previous research that demonstrated that the biggest changes in body composition following BS occurred in the first year [21], and the most important changes in total skeletal muscle mass and lean mass occurred especially within the initial 6 months [22, 23]. Our results showed that group 1 had a higher body fat mass and a lower total skeletal muscle mass prior to surgery, which is in line with previous research in this field and with the diagnostic criteria of SO [5, 24, 25]. Our study also showed that there was a high weight loss and a significant reduction in BMI and body fat mass, while there has been no statistically significant change in total skeletal muscle mass loss among our tertiles. This appears as an important outcome, since muscle mass loss was a main concern of ours, especially regarding group 1, which is the most severe group. BS appears as a risk factor for SO, contributing to muscle loss [3, 26], but our findings acknowledge bariatric surgery as a safe and emerging strategy in SO patients, being in line with previous research works [5, 23, 27]. Thus, health benefits of BS in weight reduction and SO outcomes seem to occur predominantly at the expense of fat mass alone and less by compromising lean mass [5, 23, 27].

However, there has been a statistically significant change in arm and leg lean mass in our study. Surprisingly, the most severe tertile (group 1) was the one that lost less lean mass post-surgery. The authors believe that this might be due to the increasing rate of the combination between aerobic and resistance training in that group during the follow-up period, and this fact may reduce nuclear FOXO1 protein level, therefore diminishing muscle atrophy [28]. Physical exercise is one of the first-line approaches to SO, since it increases myogenesis and interleukins 6, 10 and 15, stimulates brown fat tissue, while reducing white fat and inflammatory factors (myostatin, FOXO, leptin, resistin, NF-KB). The combined effect results in a decrease in adipose tissue and inflammation, and an increase in protein synthesis [29].

The surgical intervention led to improved metabolic markers and reduced comorbidities in women across all groups, aligning with earlier research [5, 25, 30, 31] that links significant weight loss post-surgery to reduced oxidative stress, improved insulin resistance and better lipid profiles, potentially contributing to a substantial remission rate of DM2 in those with more severe SO [32–34]. BS has also shown superior results over medical treatments in reversing dyslipidemia, significantly reducing cardiovascular risk [21]. However, HT remission rates were lower in group 1 compared to the less severe groups, albeit not statistically significant. This aligns with previous studies linking morbid obesity to hypertension [35], but it shows variability in associating SO with this condition due to inconsistent definitions [36, 37], leading to conflicting outcomes.

Within our study, TSH showed significance at baseline, displaying higher values within group 1, and a more pronounced decrease post-BS, remaining within the physiological range.

Previous longitudinal studies in obese or sarcopenic populations provide some insights, although showing conflicting associations between thyroid function and body composition. For instance, Itterman et al. identified a positive correlation between TSH levels and increases in both BMI and waist circumference across adults aged 20 to 90 [38]. In agreement to that, other population-based studies were able to demonstrate that a decline in TSH levels was associated with a decrease in body weight [39–41]. Our findings are consistent with these studies.

Strengths of This Study

Our study, utilizing the ESPEN/EASO criteria, pioneers the longitudinal evaluation of middle-aged women regarding SO post-BS, an area that remains largely unexplored. Moreover, our study boasts a robust sample size, enhancing the potential applicability of our findings, particularly among women.

In addition, this investigation used DXA as the primary assessment tool, as favoured by ESPEN and EASO consensus given its superior accuracy when compared to bioelectrical impedance analysis [3].

Limitations of This Study

Our study faced several limitations that warrant consideration. Notably, we did not assess skeletal muscle function parameters. Furthermore, our study encountered notable missing values for certain variables, such as SBP and waist circumference, both displaying high missing rates at baseline (42.86% and 60% at baseline, respectively) and during follow-up (69.29% missing rates for SBP variation at 1 year follow-up). The COVID-19 pandemic significantly contributed to these missing values, disrupting follow-up appointments that were crucial for this assessment.

Another limitation is our exclusive focus on a female cohort due to the substantial predominance of women (85%) over males (15%) undergoing BS at our institution. However, while not fully elucidated, there is growing evidence suggesting that SO may portend a more adverse prognosis in women [42, 43].

Conclusion

Bariatric surgery emerges as a promising approach for middle-aged female patients with SO. Despite concerns about potential suboptimal outcomes due to malabsorptive mechanisms that may exacerbate muscle loss and therefore worsen the condition, the most severe group of patients with SO lost more fat mass and simultaneously experienced the smallest reduction in their total skeletal muscle mass, when compared to the less severe groups. Nonetheless, remission rates for comorbidities following BS were notable. A comprehensive assessment is necessary to accurately determine the substantial impact of physical exercise on enhancing the management and outcomes of SO following BS.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11695-024-07164-x.

Declarations

Ethics Approval All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent Written informed consent was obtained from all individual participants included in the study.

Conflict of Interest The authors declare no competing interests.

References

- Baumgartner R. Body composition in healthy aging. Ann N Y Acad Sci. 2006;994(1):437–48.
- 2. Hsu K, Liao C, Tsai M, et al. Effects of exercise and nutritional intervention on body composition, metabolic health, and physical performance in adults with sarcopenic obesity: a meta-analysis. Nutrients. 2019;11(9):2163.
- Donini LM, Busetto L, Bischoff SC, et al. Definition and Diagnostic Criteria for Sarcopenic Obesity: ESPEN and EASO Consensus Statement. Obes Facts. 2022;15(3):321–35.
- Cruz-Jentoft AJ, Bahat G, Bauer J, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019;48(1):16–31.
- 5. Mastino D, Robert M, Betry C, et al. Bariatric surgery outcomes in sarcopenic obesity. Obes Surg. 2016;26(10):2355–62.
- Wang M, Tan Y, Shi Y, et al. Diabetes and sarcopenic obesity: pathogenesis, diagnosis, and treatments. Front Endocrinol (Lausanne). 2020;25:11.
- Lu C, Yang K, Chang H, et al. Sarcopenic obesity is closely associated with metabolic syndrome. Obes Res Clin Pract. 2013;7(4):301–7.
- Kalinkovich A, Livsgits G. Sarcopenic obesity or obese sarcopenia: a cross talk between age-associated adipose tissue and skeletal muscle inflammation as a main mechanism of the pathogenesis. Ageing Res Rev. 2017;1(35):200–21.
- Ciudin A, Simó-Servat A, Palmas F, et al. Sarcopenic obesity: a new challenge in the clinical practice. Endocrinol Diabetes Nutr. 2020;67(10):672–81.
- Wei S, Nguyen TT, Zhang Y, et al. Sarcopenic obesity: epidemiology, pathophysiology, cardiovascular disease, mortality, and management. Front Endocrinol (Lausanne). 2023;14:1185221.
- Fried M, Yumuk V, Oppert JM, et al. Interdisciplinary European guidelines on metabolic and bariatric surgery. Obes Facts. 2013;6(5):449–68.
- 12. Gallagher D, Heymsfield SB, Heo M, et al. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr. 2000;72(3):694–701.
- Poggiogalle E, Lubrano C, Sergi G, et al. Sarcopenic obesity and metabolic syndrome in adult Caucasian subjects. J Nutr Health Aging. 2016;20(9):958–63.
- Petermann-Rocha F, Balntzi V, Gray SR, et al. Global prevalence of sarcopenia and severe sarcopenia: a systematic review and meta-analysis. J Cachexia Sarcopenia Muscle. 2022;13(1):86–99.
- Kim T, Yang S, Yoo H, et al. Prevalence of sarcopenia and sarcopenic obesity in Korean adults: the Korean sarcopenic obesity study. Int J Obes. 2009;33:885–92.
- Vieira FT, Godziuk K, Lamarca F, et al. Sarcopenic obesity diagnosis by different criteria mid-to long-term post-bariatric surgery. Clin Nutr. 2022;41(9):1932–41.

- 17. Mijnarends D, Meijers J, Halfens R, et al. Validity and reliability of tools to measure muscle mass, strength, and physical performance in community-dwelling older people: a systematic review. J Am Med Dir Assoc. 2013;14(3):170–8.
- Prado C, Wells J, Smith S, et al. Sarcopenic obesity: a critical appraisal of the current evidence. Clin Nutr. 2012;31(5):583-601.
- 19. Sousa-Santos AR, Afonso C, Borges N, et al. Factors associated with sarcopenia and undernutrition in older adults. Nutr Diet. 2019;76(5):604–12.
- Esposito L, Valeriani L, Anzolin F, et al. A valid screening tool of sarcopenic obesity in patients candidates to bariatric surgery. Clin Nutr ESPEN. 2023;1(54):506.
- Sjöström L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351(26):2683–93.
- 22. Ciangura C, Bouillot J, Lloret-Linares C, et al. Dynamics of change in total and regional body composition after gastric bypass in obese patients. Obesity. 2010;18(4):760–5.
- 23. Sivakumar J, Chen Q, Sutherland TR, et al. Body composition differences between excess weight loss \geq 50% and < 50% at 12 months following bariatric surgery. Obes Surg. 2022;32(8):2556–66.
- 24. Crispim Carvalho NN, Baccin Martins VJ, da Nóbrega VA, et al. Effects of preoperative sarcopenia-related parameters on cardiac autonomic function in women with obesity following bariatric surgery: a one-year prospective study. Nutrients. 2023;15(12):2656.
- 25. Crispim Carvalho NN, Martins VJB, Filho JM, et al. Effects of preoperative sarcopenia-related parameters on the musculoskeletal and metabolic outcomes after bariatric surgery: a one-year longitudinal study in females. Sci Rep. 2023;13(1):13373.
- 26. Lynch DH, Spangler HB, Franz JR, et al. Multimodal diagnostic approaches to advance precision medicine in sarcopenia and frailty. Nutrients. 2022;14(7):1384.
- Coral RV, Bigolin AV, Machry MC, et al. Improvement in muscle strength and metabolic parameters despite muscle mass loss in the initial six months after bariatric surgery. Obes Surg. 2021;31(10):4485–91.
- Brocca L, Toniolo L, Reggiani C, et al. FoxO-dependent atrogenes vary among catabolic conditions and play a key role in muscle atrophy induced by hindlimb suspension. J Physiol. 2017;595(4):1143–58.
- 29. Alizadeh PH. Exercise therapy for people with sarcopenic obesity: myokines and adipokines as effective actors. Front Endocrinol (Lausanne). 2022;17:13.
- 30. Piché ME, Tardif I, Auclair A, Poirier P. Effects of bariatric surgery on lipid-lipoprotein profile. Metabolism. 2021;1:115.
- 31. Brethauer S, Aminian A, Resenthal R, et al. Bariatric surgery improves the metabolic profile of morbidly obese patients with type 1 diabetes. Diab Care. 2014;37(3):51–2.
- 32. Tumova E, Sun W, Jones PH, et al. The impact of rapid weight loss on oxidative stress markers and the expression of the metabolic syndrome in obese individuals. J Obes. 2013; Available from: / pmc/articles/PMC3880717/
- Wahlroos S, Phillips ML, Lewis MC, et al. Rapid significant weight loss and regional lipid deposition: implications for insulin sensitivity. Obes Res Clin Pract. 2007;1(1):7–16.
- Harder H, Dinesen B, Astrup A. The effect of a rapid weight loss on lipid profile and glycemic control in obese type 2 diabetic patients. Int J Obes Relat Metab Disord. 2004;28(1):180–2. Available from: https://pubmed.ncbi.nlm.nih.gov/14610532/.
- 35. Pasdar Y, Darbandi M, Rezaeian S, et al. Association of obesity, sarcopenia, and sarcopenic obesity with hypertension in adults: a cross-sectional study from Ravansar, Iran During 2014–2017. Front Public Health. 2022;2:9.

- Park SH, Park JH, Song PS, et al. Sarcopenic obesity as an independent risk factor of hypertension. J Am Soc Hypertens. 2013;7(6):420–5.
- Coelho Júnior HJ, Aguiar SDS, Gonçalves IDO, et al. Sarcopenia is associated with high pulse pressure in older women. J Aging Res. 2015.
- 38. Ittermann T, Markus MRP, Bahls M, et al. Low serum TSH levels are associated with low values of fat-free mass and body cell mass in the elderly. Sci Rep. 2021;11(1):10547.
- 39. Nyrnes A, Jorde R, Sundsfjord J. Serum TSH is positively associated with BMI. Int J Obes. 2006;30(1):100–5.
- 40. Tiller D, Ittermann T, Greiser KH, et al. Association of serum thyrotropin with anthropometric markers of obesity in the general population. Thyroid. 2016;26(9):1205–14.
- 41. Svare A, Nilsen TIL, Bjøro T, et al. Serum TSH related to measures of body mass: longitudinal data from the HUNT Study, Norway. Clin Endocrinol (Oxf). 2011;74(6):769–75.

- Petroni ML, Caletti MT, Dalle Grave R, et al. Prevention and treatment of sarcopenic obesity in women. Nutrients. 2019;11(6):1302.
- Stuck AK, Tsai LT, Freystaetter G, et al. comparing prevalence of sarcopenia using twelve sarcopenia definitions in a large multinational European population of community-dwelling older adults. J Nutr Health Aging. 2023;27(3):205–12.

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