Bariatric Surgery Outcomes in Sarcopenic Obesity

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Abstract

Background Sarcopenic obesity is the combination of low muscle mass and strength with increased fat mass. This condition is associated with negative health outcomes. We hypothesized that sarcopenia could be a pejorative factor on surgical weight loss.

Objective The objectives of the study are to determine the influence of sarcopenic obesity on gastric bypass and sleeve gastrectomy results regarding weight loss and comorbidities resolution at 3, 6, and 12 months.

Setting The study was conducted at the University Hospital.

Methods Sixty-nine obese patients who benefited from bariatric surgery were included. Skeletal muscle mass was determined by the Janssen's equation. Physical performance and muscle strength were determined using the 6-min walk test and the wall sit test. Obese subjects from the lowest tertile of the Skeletal Muscle mass Index (SMI) of Baumgartner were set as sarcopenic.

Results Weight loss outcomes and rate of weight loss failure were not influenced by sarcopenia. At 1 year, mean EBMIL% was 75.4 % ± 5 in sarcopenic subjects vs 67.8 % ± 4 in the non-sarcopenic subjects ($p = 0.242$). Improvement rates of co-morbidities were similar between groups. Skeletal muscle mass was no more different between groups at 1 year after surgery. There was no patient lost to follow-up.

Conclusions Bariatric surgery remains effective in achieving weight loss target in sarcopenic patients, with similar remission rates of main comorbidities and similar safety profile than in the non-sarcopenic group. Whether bariatric surgery could result in improvement or deterioration of daily living activities disabilities and functional autonomy in sarcopenic obese patients still have to be evaluated.

Keywords Sarcopenia · Muscle mass · Morbid obesity · Bariatric surgery · Weight loss outcomes

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Introduction

The combination of low muscle mass and strength with increased fat mass is a condition called sarcopenic obesity that can promote, with a synergistic effect, a further deterioration of metabolic disorders and physical disability. Whereas the definition of sarcopenic obesity is not yet consensual, sarcopenia is a well-defined condition characterized by progressive and generalized loss of skeletal muscle mass and function (strength and performance), in agreement with the latest consensus developed by the European Working Group on Sarcopenia in Older People

(EWGSOP) [1]. Usually, this process is age-related with a loss of 1–2 % of muscle mass per year after 50 years of age [2]. The muscle mass/fat mass ratio imbalance is not an exclusive prerogative of the elderly, but all obese, regardless of age, may have alterations of this ratio capable to affect significantly health outcomes, such as hypertension [3], arterial stiffness [4], nonalcoholic fatty liver disease [5], metabolic syndrome and insulin resistance [6], osteoporosis [7], or difficulties with physical function [8]. Furthermore, two recent studies have demonstrated respectively that women with sarcopenic obesity had a higher mortality risk than those without sarcopenia or obesity [9], and that men with sarcopenic obesity had a higher risk of all-cause mortality compared to only sarcopenic or only obese subjects [10].

Identifying obese subjects with sarcopenia can be challenging. The EWGSOP suggests two different methods to assess muscle mass in clinical practice: the dual energy X-ray absorptiometry (DEXA) and the bio-electrical impedance analysis (BIA) [1]. On one hand, DEXA is difficult to use and implement in routine, due to elevated cost and specialized professional requirements. On the other hand, BIA is relatively simple, quick, and non-invasive, but has been shown to slightly over-estimate fat free mass in obese subjects [11]. In 1996, sarcopenic obesity was defined for the first time by Heber et al. using BIA [12], and a reliable BIA equation was developed later on by Janssen et al. to assess muscle mass in a wide range of subjects including morbidly obese subjects [13].

Management strategies for sarcopenic obesity have to focus on loss of fat mass as well as maintenance or accretion of lean body mass in order to maintain muscular strength and function. Different approaches have been used for this purpose, such as diet changes, aerobic exercise, and resistance training, with results often not encouraging or limited in time. Given the difficulties related to the management of this syndrome, and the lack of evidence supporting effective strategies, sarcopenic obesity is generally considered as a possible predictive factor of bariatric surgery failure. Indeed, we previously reported that the whole body fat free mass including muscle mass, was a relevant predictive factor of weight loss following bariatric surgery [14], and it is also accepted that muscles are the main site of fat oxidation. Thus, sarcopenic obesity that combines low muscle mass and high fat mass could lead to poorer weight loss results after bariatric surgery. To date, no study has assessed the impact of sarcopenic obesity on bariatric surgery outcomes. In the present study, we aimed to determine the influence of sarcopenic obesity on gastric bypass and sleeve gastrectomy results regarding weight loss and comorbidities resolution at 3, 6, and 12 months.

Material and Method

Subjects

This is a retrospective monocentric study of prospectively collected data. Among the 239 patients who underwent either a gastric bypass or a sleeve gastrectomy between 2010 and 2012, we selected the 100 caucasian patients who benefited from a preoperative bioimpedance analysis (BIA) allowing quantification of pre-operative skeletal muscle mass. Twenty-four patients were excluded due to extreme BMI (>48 kg m^{-2}) that could lead to uncertainty concerning BIA validity. Five patients were excluded due to insufficient data during the follow-up. Two patients were excluded because of pregnancy respectively 5 and 8 months after the sleeve gastrectomy. A total of 69 patients were finally taken on in the analysis. Twelve patients had a previous history of bariatric surgery and benefited from revisional surgery.

Pre-Operative Evaluation

Body Composition

Body weight and height were measured to the nearest 0.5 k and 0.1 cm using standardized equipment and procedures. Body mass index (BMI) was calculated as weight/height^{-2} (kg m^{-2}). Body composition was assessed by bio-impedance analysis (BIA). BIA resistance (ohms) was obtained with the QuadScan 4000 multi-frequency bioelectrical impedance analyzer (BodyStat®, UK). An electrical current of 50 kHz and 0.8 mA was produced by the generator and applied to the skin between the right wrist and ankle using adhesive electrodes with subject in supine position on a non-conducting surface. By measuring the impedance at 50 kHz and applying the BodyStat® equation, fat mass (FM) and free fat mass (FFM) were calculated. The standard error of estimate (SEE) or prediction error for BIA is known to be about 3.5 % [15] Fat mass was reported in kilograms (FM), percentage of total body weight (FM%), and as the fat mass index (FMI FM (kg)/height^{-2} (m^{-2})). Plasma leptin was quantified as a biological marker of fat mass.

Fat free mass, that includes skeletal muscle mass + bones + lean soft tissue + body fluids, was reported in kilograms, percentage of total body weight (FFM%), and as the fat free mass index (FFMI FFM (kg)/height^{-2} (m^{-2})). Dry FFM was also reported.

Skeletal Muscle Mass (SSM)

Skeletal muscle mass was predicted by BIA resistance using the Janssen's equation [13] developed and cross-validated against magnetic resonance imaging measures of whole body

muscle mass in a sample of 269 men and women varying widely in age (18–86) and adiposity (BMI = 16–48 kg m^2).

This equation is widely used in literature dealing with sarcopenic obesity.

$$\text{Skeletal muscle mass (kg)} = \left[\left(\text{height}^{-2}/\text{BIA resistance} \times 0.401\right) + \left(\text{gender} \times 3.825\right) + \left(\text{age} \times -0.071\right)\right] + 5.102$$

Height is in cm; BIA resistance is in ohms; for gender, men = 1 and women = 0; and age is in years.

The skeletal muscle mass adjusted for squared height (SM/height^{-2}), also known as the skeletal muscle mass index (SMI) developed by Baumgartner was used in that study to identify sarcopenia [16]. Skeletal muscle mass (kg) was also converted to skeletal muscle mass percentage, defined as skeletal muscle mass index (SMI% = total muscle mass/body mass × 100). SMI% adjusts SMM for stature and the mass of non-skeletal muscle tissues (fat, organ, bone). Finally, we calculated the ratio of skeletal muscle mass (SM or SM/H^2) to fat mass (kg or %) to quantify the relative imbalance between musculature and adiposity, independently of the absolute skeletal muscle mass.

Plasma creatinine was quantified and could be used as an indirect biological marker of skeletal muscle mass because chronic kidney disease was an exclusion criterion for that study.

Physical Performance and Muscle Strength

All the subjects were evaluated for preoperative physical performance and muscle strength using the 6-min walk test (6MWT) and the wall sit test (WST), respectively. The 6MWT measures the distance an individual is able to walk over a total of 6 min on a hard, flat surface. It is a submaximal exercise test to measure functional exercise capacity. The wall sit test evaluates the strength endurance of the lower body, particularly the quadriceps muscle group. The outcome is the time the subject can maintain a position with the back against a vertical wall with both knees and hips at a 90° angle [17, 18].

Sarcopenia

A few definitions of sarcopenic obesity have been proposed in the recent years. We used the SMI of Baumgartner (SM/Height^{-2}) to identify sarcopenia in our obese population [16]. We set that obese subjects from the lowest tertile of SMI were sarcopenic whereas those from the two highest tertiles were not.

Surgical Procedures

All the patients were operated on by laparoscopy, by the same experienced surgeon.

Roux-in-Y Gastric Bypass A 5-port technique was used as described by Lonroth et al. It consisted in a small gastric pouch (30 cc) by stapling the stomach using a linear stapler. The first jejunal loop was used and moved up into an antecolic position after an epiploic transection so as to perform the gastro-jujenal anastomosis. An end-to-side gastro-jejunal anastomosis was performed using a linear stapler. Closure of the anterior part of the anastomosis was done using a running suture. The alimentary limb was 150-cm long. A latero-lateral jejuno-jejunal anastomosis was performed with a linear stapler. Closure of the mesenteric defect was systematic, using a non-absorbable silk suture (2/0).

Sleeve Gastrectomy A 5-port technique was used. A 37 French tube was used to calibrate the sleeve gastrectomy. The gastric section began 6 cm away from the pylorous. The staple line was reinforced using a running suture.

Post-Operative Data Collection

Duration of the surgical procedure, conversions to laparotomy, operative complications, and early complications (within 30 days after surgery) were recorded. Postoperative features at 3, 6, and 12 months were prospectively collected using a computerized database devoted to bariatric surgery.

Absolute body weight loss in Kg (aWL), percentage of body weight loss (WL%), delta body mass index (BMI) from baseline, percentage of excess BMI loss (EBMIL%), and percentage of excess weight loss (EWL%) were recorded or calculated to quantify weight loss at 3, 6, and 12 months after surgery. Weight loss failure was defined as BMI >35 kg.m^{-2} or EBL% <50 % 1 year after surgery. Body composition using BIA was redone for each subject 12 months after surgery.

Co-morbidities including type 2 diabetes (T2D), high blood pressure (HBP), dyslipidemia, non-alcoholic steatosis hepatitis (NASH), severe obstructive sleep apnea syndrome (OSAS), and degenerative joint disease (knee joint pain) were evaluated and recorded at baseline and 12 months after surgery. In that study, the remission of T2D was defined as cessation of anti-diabetic treatments and HbA1c < 6 % and/or fasting blood glucose levels < 100 mg/dl, according to the American Diabetes Association (ADA) guidelines. Severe OSAS was considered improved when continuous positive airway pressure was stopped. Dyslipidemia was considered improved when medications were stopped. HBP was

considered improved when at least one medication was discontinued during the follow-up. Hepatitis on fatty liver was considered improved when liver enzymes were normalized. Degenerative joint disease was considered as functionally improved when painkillers were stopped and knee pain no more reported.

Statistical Analysis

All the analyses were performed using IBM SPSS Statistics 19 (SPSS inc, Chicago, Il, USA). Data were presented as arithmetic means ±1 SEM. Quantitative differences between groups were assessed by a two-tailed Student's t test. A Pearson chi-square test with a Yates' correction for continuity was applied for comparison of proportions. A multiple regression analysis was performed to take into account age, gender, surgical procedure, height, and initial weight in determining the impact of sarcopenia on weight loss outcomes. ANOVA repeated measures were performed to analyze the influence of sarcopenia on weight loss during the first 12 months following surgery. $p < 0.05$ was considered significant.

Results

Descriptive Statistics of Both Groups

The sarcopenic group was defined as the tertile of the population with the lowest SM/H^2 index (mean SM/H^2, 9.41 kg/m^2), whereas the two others were defined as the non-sarcopenic group (mean SM/H^2, 11.77 kg/m^2). Sarcopenic subjects were not older (44y ±2 vs 47y ±1, $p = 0.18$). Detailed body composition and anthropometric characteristics are presented in Tables 1 and 2. There was

no significant anthropometric difference between groups regarding weight, height, and BMI. By study design, sarcopenic subjects exhibited significantly lesser skeletal muscle mass whatever the way to express it, in kilograms (SMM) or after adjustment for height (SM/H^{-2}), body weight (SMI), and fat mass ($SM/FM \times 100$ and (SM/H^{-2})/ (FM%)). Sarcopenic subjects had an average of 6 kg of muscle mass less than subjects without sarcopenia, representing a gap between groups of 18.3 % of skeletal muscle mass. Furthermore, plasma creatinine, which was used as a surrogate biological marker of skeletal muscle mass, tended to be lesser in sarcopenic subjects (58 μmol/L ±2 vs 80 μmol/L ±8, p = 0.073). Fat-free mass (FFM), that includes skeletal muscle mass + bones + lean soft tissues + body fluids, tended to be lesser in sarcopenic subjects when expressed in kilograms, percentage, or kilograms adjusted for height (FFMI). Concerning fat mass, there was a trend for sarcopenic subjects to be fatter with a percentage of fat mass of 47,1 % ±1,6 vs 43,2 % ±1,2 ($p = 0.054$). This trend was no more present when fat mass was adjusted for height (FMI). Furthermore, plasma leptin, used as a biological marker of fat mass, was not different between groups ($p = 0,626$). Thus, both study groups were clearly different according to the preoperative skeletal muscle mass, while BMI and fat mass were similar. Despite different skeletal mass between groups, physical performance assessed by the 6-min walk test distance was identical between groups (407 m ±16 vs 399 m ±12, $p = 0.70$) as well as the muscle strength assessed by the wall sit test (36.1 s ±9 vs 31.2 s ±4, $p > 0,05$). Biological features and co-morbidities, presented in Table 2, were not different between sarcopenic and non-sarcopenic groups.

Surgical characteristics are presented in Table 3. The proportion of revisional surgery was balanced between groups

Table 1 Pre-operative body composition of subjects with or without sarcopenia

	n	Sarcopenia 23	No sarcopenia 46	p
Fat mass (FM) (Kg)	69	54.5 ± 2.4	51.3 ± 1.71	0.272
FM %	69	47.1 % ± 1.6	43.2 % ± 1.2	0.054
FMI (Kg/m^2)	69	19.3 ± 0.9	18.8 ± 0.7	0.629
Leptin (ng/ml)	69	56 ± 9	51 ± 6	0.626
Fat-free mass (FFM) (Kg)	69	61.5 ± 2.9	67.2 ± 1.9	0.096
FFM % (%)	69	52.9 % ± 1.6	56.8 % ± 1.2	0.054
FFMI (Kg/m^2)	69	21.6 ± 0.5	24.2 ± 0.4	**<0.001**
Dry FFM (Kg)	69	16.5 ± 1	15.6 ± 0.6	0.415
Skeletal muscle mass (SMM) (Kg)	69	26.8 ± 1.4	32.8 ± 1	**<0.001**
SM/H^2	69	9.41 ± 0.28	11.77 ± 0.25	
SMI (%)	69	23.1 ± 0.9	27.6 ± 0.7	**<0.001**
$SM/FM \times 100$	69	52.2 ± 4.4	68.7 ± 4	**0.012**
$(SM/H^2)/(FM\%)$	69	21.5 % ± 1.5	28.8 % ± 1.4	**0.002**
Plasma creatinine (μmol/L)	69	58 ± 2	80 ± 8	0.073

Table 2 Baseline characteristics of obese subjects with or without sarcopenia

n	Sarcopenia 23	No sarcopenia 46	p
Sex ratio (% of men)	39.1 %	41.3 %	0.864
Age (yr)	44 ± 2	47 ± 1	0.180
Height (cm)	167 ± 2	166 ± 1	0.541
Weight (Kg)	116.0 ± 3.8	118.6 ± 2.4	0.544
BMI (Kg/m^{-2})	41.1 ± 0.7	42.9 ± 0.6	0.071
Excess BMI (Kg/m^{-2})	16.1 ± 0.7	17.9 ± 0.6	0.071
HBP (%)	34.8 %	54.3 %	0.125
OSAS(%)	52.2 %	47.8 %	0.733
T2D (%)	56.5 %	50.0 %	0.609
Insulin treatment (%)	17.4 %	19.6 %	0.828
Dyslipidemia (%)	39.1 %	58.7 %	0.125
Hepatitis on fatty liver (%)	8.7 %	21.7 %	0.178
Knee joint pain (%)	56.5 %	73.9 %	0.144
HbA1c (%)	6.8 ± 0.4	7.1 ± 0.3	0.680
FPG (mmol/L)	6.4 ± 0.5	6.9 ± 0.5	0.472
HOMA-IR	3.11 ± 0.48	7.27 ± 2.17	0.188
TG (mmol/L)	2.10 ± 0.40	1.96 ± 0.15	0.706
HDLc (mmol/L)	1.24 ± 0.07	1.16 ± 0.05	0.336
LDLc (mmol/L)	3.14 ± 0.19	2.94 ± 0.14	0.431
CRP (mg/L)	12 ± 2.6	7.7 ± 1.3	0.072
Uric Acid (μmol/L)	355 ± 23	357 ± 17	0.971
ALAT (UI/L)	30 ± 3	33 ± 3	0.488
ASAT (UI/L)	25 ± 2	29 ± 2	0.230
GGT (UI/L)	50 ± 9	60 ± 16	0.709
Prealbumin (g/L)	0.25 ± 0.01	0.26 ± 0.01	0.462
Albumin (g/L)	39 ± 1	40 ± 1	0.415
Microalbuminuria (μg/mL)	7.5 ± 3	15 ± 4	0.210
TSH (mUI/L)	2.11 ± 0.26	2.74 ± 0.32	0.207

representing 17.4 % of each group. Distribution of GBP and sleeve gastrectomy was balanced between the groups with three-quarters of GBP and a quarter of sleeve gastrectomy. Baseline sarcopenia was not associated with a longer surgical mean duration of the surgical procedure (128 vs 138 min, $p = 0.43$). Neither conversion to laparotomy nor intraoperative complications occurred. Early complications rate was not significantly different between the two groups ($p = 0.807$).

Table 3 Surgical characteristics of subjects with or without sarcopenia

	Sarcopenia 23	No sarcopenia 46	p
Gastric ByPass (%)	73.9 %	76.1 %	0.843
Sleeve Gastrectomy (%)	26.1 %	23.9 %	0.843
Revision Surgery (%)	17.4 %	17.4 %	1
Early Complications (%)	9.5 %	7.7 %	0.807
Surgery Time (min)	128 ± 6	138 ± 10	0.433

Weight Loss Outcomes

We did not observe any difference at 3, 6, and 12 months after surgery between sarcopenic and non-sarcopenic groups regarding weight loss outcomes. At 1 year, mean EBMIL% was 75.4 % ± 5 versus 67,8 % ± 4, respectively ($p = 0.242$), mean EWL% was 65 % ± 4 versus 60 ± 3 ($p = 0.338$), mean weight loss % was 28.6 % ± 2 versus 27.4 % ± 1 ($p = 0.56$) and mean absolute weight loss was 33.1 kg ± 2 versus 32.3 kg ± 2 ($p = 0.78$) (Fig. 1).

Weight loss outcomes were analyzed using a multiple regression model to take into account several potential confounding factors and we did not find any additional independent influence of gender, age, surgical technique, height, and initial weight that could explain our weight loss results. Rate of weight loss failure at 1 year was not different between groups (13.0 % vs 26.1 %, $p = 0.216$). Metabolic and non-metabolic co-morbidities were similarly improved or cured in sarcopenic and non-sarcopenic subjects (Table 4).

It is worth noting that skeletal muscle mass at 1 year after surgery was no more different between sarcopenic and non-sarcopenic group with a SMM, respectively, of 29.2 kg ± 2 and 30.5 kg ± 1 ($p = 0.54$).

Discussion

In the current study, we tried to evaluate the influence of sarcopenia on bariatric surgery outcomes. Indeed, we hypothesized that sarcopenia could be a pejorative factor on weight loss and could result in weight loss failure. Moreover, sarcopenia remains a difficult condition to treat, and medical strategies are often ineffective.

Sarcopenic obesity is currently considered one of the variables to take into account when considering bariatric surgery as an option to treat obesity. However, the real extent of this pathological condition and the resulting burden on public health and economic resources of the health-care systems in developed countries are not yet well defined [19].

The use of different definitions, thresholds, indexes, and methods to determine fat mass and skeletal muscle mass have generated an extreme variability in the assessment of prevalence of sarcopenic obesity [20, 21]. Therefore, it clearly appears that there is an urgency to establish a universal, standardized definition of this condition to identify its pathophysiology, its consequences, and its medical care. In our study, skeletal muscle mass was assessed by bioelectrical impedance analysis (BIA) Janssen's formula [13] as described earlier, that allows to obtain reliable bioelectrical impedance analysis (BIA) measures. Although in 2009, the European Working Group on Sarcopenia in Older People (EWGSOP) developed a practical clinical definition and consensus diagnostic criteria for age-related sarcopenia [1], the majority of published

Fig. 1 Weight loss during the first year following bariatric surgery in obese subjects with sarcopenia (*dark gray histograms*) and subjects without sarcopenia (*light grey histograms*). Excess BMI loss (EBL) in panel (**a**). Excess weight loss (EWL) in panel (**b**). Percentage of total weight loss from baseline in panel (**c**). Absolute weight loss in panel (**d**) and delta BMI from baseline in panel (**e**)
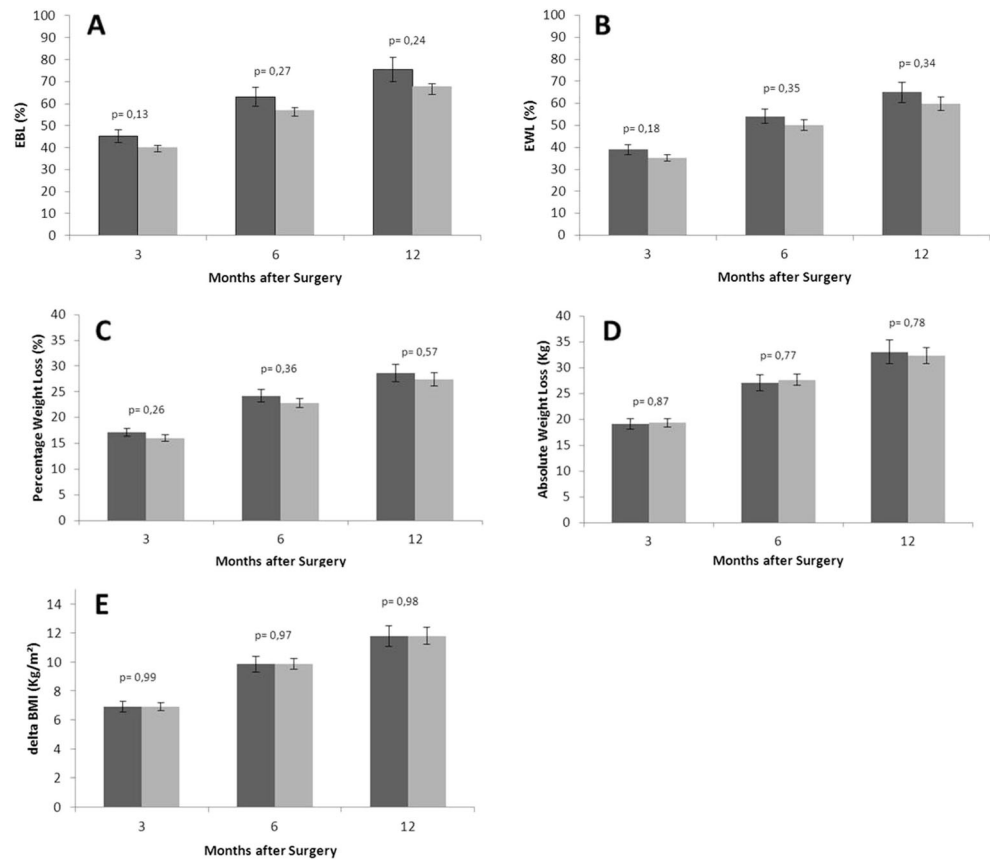
studies on sarcopenic obesity do not use this definition. Indeed, we know that weight gain in obese people is mainly caused by an increase of fat mass; however, a contemporary rise of skeletal muscle mass is often observed. Therefore, using EWGSOP cut-offs for skeletal muscle mass index (SMI) to define sarcopenia in obese people may be misleading. For that reason, we arbitrarily considered in the present study that the tertile of our population with the lowest SMI would be the sarcopenic group. Due to the fact that our sarcopenic and non-sarcopenic groups are similar in terms of baseline BMI, fat mass, co-morbidities, and biological features, we assume that our results concerning surgical weight loss and co-morbidities resolution depend on the presence of sarcopenia.

The main result that we observed in the present study was that sarcopenia did not impact bariatric surgery outcomes, 1 year after RYGB or sleeve gastrectomy. These results are consistent with previously published data from our group on elderly patients [22]. Indeed, we observed that RYGB remains effective in patients over 65 years old, a population in which sarcopenia is highly prevalent. Several other authors have also reached these conclusions [23–25].

Several studies have examined whether obesity and sarcopenia may potentiate to induce metabolic, mechanical, psychosocial, and degenerative comorbidities. The interconnection between obesity and sarcopenia is very deep. Several studies indicated that obesity and/or insulin resistance may underlie the development of sarcopenia. Reciprocally, loss of skeletal muscle mass might decrease energy expenditure and basal metabolic rate and therefore could facilitate weight gain and obesity. Besides, because skeletal muscle mass is the main target tissue of insulin, sarcopenia can lead to insulin resistance [26]. Thus, sarcopenia in obese subjects has been previously associated with higher risk of HBP [3], arterial stiffness [4], dyslipidemia [27], nonalcoholic fatty liver diseases (NAFLD) [5], insulin resistance [6], knee arthritis and osteoporosis causing an increasing risk of falls and fractures [7, 28], and lower cognitive functioning [29]. Moreover, two recent

Table 4 Proportion of improved co-morbidities 1 year after bariatric surgery in subjects with or without sarcopenia

Improved co-morbidity % (*n*)	Sarcopenia	No sarcopenia	*p*
HBP	50 % (8)	24 % (25)	0.164
OSAS	42 % (12)	37 % (22)	0.761
T2D	77 % (13)	70 % (22)	0.636
Insulin treatment	75 % (4)	67 % (9)	0.764
Dyslipidemia	67 % (9)	52 % (27)	0.439
Hepatitis on fatty liver	100 % (2)	90 % (10)	0.640
Knee joint pain	62 % (13)	62 % (34)	0.989

studies have associated sarcopenic obesity in the elderly with higher mortality risk in comparison to obesity or sarcopenia alone [9, 10]. However, the other interesting result of the current study is that sarcopenic patients have a similar improvement rate of comorbidities than the non-sarcopenic group. They are also not at higher operative risk, as the early complication rate and the surgical procedure duration were similar in both groups. Therefore, it appears that weight loss is still beneficial, whatever the amount of skeletal muscle mass, to reduce knee joint loads, and to improve NASH, hypertension, type 2 diabetes, dyslipidemia, and OSAS. The discrepancy between our results and those previously reported in the literature could be due to both the younger age of our cohort and the similar amount of fat mass between sarcopenic and non-sarcopenic groups.

Management strategies for sarcopenic obesity have to focus on loss of fat mass as well as maintenance or accretion of lean body mass in order to maintain muscular strength and function. Poggiagalle et al., in a recent review, showed that weight loss based on calorie-restricted diet, combined with aerobic and/or resistance exercise seems to be the best medical strategy in sarcopenic obese patients, to achieve a fat mass decrease, preserving skeletal muscle mass [30]. Weinheimer et al. [31] reported that addition of exercise to energy restriction reduces the amount of weight lost as fat-free mass from approximately 11 % to approximately 24 %. Nevertheless, grade 2 obese individuals (BMI > 35 Kg/m^2), particularly sarcopenic obese subjects, are refractory to physical activity and lifestyle modification for many reasons (psychiatric/psychological issues, family/social barriers, arthritis-derived chronic pain, cardiovascular problems); therefore, in these patients, diet is often not coupled with exercise. Besides Nilsson et al. proved that in obese rats, the protein synthetic response to exercise was blunted compare to slim phenotype, suggesting a muscle resistance to the anabolic boost of exercise [32]. Obviously, these findings need to be confirmed in humans.

We know that in case of mild energy intake restriction (50–70 %), lean body mass loss is about 15–20 % of total body weight loss [33]. The decline of lean body mass promotes a worsening of obesity-related disability, making physical activity even more difficult. On the other hand, weight regain is associated with an almost exclusive accumulation of fat mass. Therefore, in case of discontinuation of dietary changes, the final result is a net reduction in skeletal muscle mass and the risk of experiencing a vicious cycle (the "yo-yo syndrome") that inexorably leads to the onset or worsening of sarcopenic obesity.

Considering these data, bariatric surgery represents an interesting therapeutic option to achieve a substantial weight loss, despite a reduction in skeletal muscle mass (sarcopenia).

Currently, the presence of sarcopenia in bariatric surgery candidates directs us to a more aggressive procedure, in order to be more effective in terms of weight loss, even in this unfavorable condition. This attitude is also supported by our results showing that the amount of skeletal muscle mass 1 year after surgery was no more different between sarcopenic and non-sarcopenic patients. To date, our study is the only one that assessed the influence of sarcopenia on bariatric surgery outcomes.

Nevertheless, this study has some limitations. It is a retrospective study with a small sample size. The mean follow-up is quite short which did not make it possible to assess the influence of sarcopenia on weight regain. The use of BIA, although validated and widely employed in the literature, may also be less accurate than DEXA, CT, or MRI to estimate skeletal muscle mass. Another weakness of the study is that we did not evaluate whether bariatric surgery resulted in improvement or deterioration of daily living activities disabilities and functional autonomy in sarcopenic patients. This will be the purpose of a future study.

Conclusion

In conclusion, this study showed that bariatric surgery remains effective in achieving weight loss target in sarcopenic patients, with similar improvement rates of main comorbidities and similar safety profile than in the non-sarcopenic group. Moreover, it appears that skeletal muscle mass 1 year after surgery was no more different between sarcopenic and non-sarcopenic group which suggests that sarcopenic patients don't loose more muscle mass despite weight loss. Nevertheless, as previously mentioned, some uncertainties regarding the correct definition of sarcopenic obesity [34], and regarding the best method to assess body composition remain, and apply for a standardized definition of sarcopenic obesity so as to assess the results properly.

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