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ORIGINAL RESEARCH

The Impact of Sarcopenic Obesity on Weight Loss Outcomes and Recurrent Weight Gain Following Laparoscopic Sleeve Gastrectomy

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Background: Sarcopenic obesity (SO) presents a significant public health challenge. Although the prevalence of SO is on the rise, there is a notable paucity of research examining the impact of SO on weight loss efficacy following laparoscopic sleeve gastrectomy (LSG).

Purpose: To investigate the impact of SO on weight loss outcomes and recurrent weight gain following LSG.

Methods: We retrospectively gathered clinical data of 104 patients with obesity who underwent LSG in our hospital between January 2020 and September 2023. The ratio of fat mass to lean mass ≥0.8 was calculated for SO. Among them, the SO group (n=34) and the non-sarcopenic obesity (NSO) group (n=70), and the effect of SO on weight loss outcomes and recurrent weight gain were comparatively analyzed.

Results: Within one year after LSG, SO patients had an apparently higher average body mass index, excess body mass index, and lower average percentage of excess weight loss than NSO patients (P<0.05). Furthermore, NSO patients had a statistically striking optimal weight loss rate more than SO patients in the first year after surgery (58.6% vs 35.3%, P=0.026). Within two years after LSG, SO patients had a lower occurrence rate of recurrent weight gain than NSO patients. Nonetheless, this difference was not statistically significant (P=0.212). Additionally, diabetes was an independent risk factor for recurrent weight gain postoperatively in patients with obesity when both univariate and multivariate analyses were conducted (P<0.05).

Conclusion: SO adversely affects short-term weight loss outcomes after LSG. However, the effect of SO on postoperative recurrent weight gain was not statistically significant, indicating that further research is needed. Furthermore, diabetes is an independent risk factor for postoperative recurrent weight gain in patients with obesity.

Keywords: sarcopenic obesity, laparoscopic sleeve gastrectomy, weight loss outcomes, postoperative recurrent weight gain, risk factors

Introduction

China and the rest of the world are facing the challenge of increasing obesity year by year.[1,2] According to the World Obesity Map 2023, the global population with overweight and obesity reached 2.6 billion in 2020, and it is expected to reach half of the global population by 2035.[3] Obesity can lead to other systemic diseases, such as fatty liver, diabetes, and sleep apnea syndrome, which bring serious harm to individuals with obesity.[4,5] However, traditional weight loss methods, such as diet control and exercise, are ineffective for weight loss and are prone to recurrent weight gain.[6,7] Bariatric surgery plays a key role in addressing this issue. Among all bariatric surgeries, laparoscopic sleeve gastrectomy (LSG) has gained popularity at home and abroad due to its ease of operation and significant and long-lasting weight loss effect.[8,9]

Sarcopenic obesity (SO) is a challenging public health problem. It is a clinical condition in which sarcopenia and obesity coexist.[8] The synergistic effect of sarcopenia and obesity leads to a severe chronic inflammatory response, insulin

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resistance, and other abnormalities. This results in a decline in muscle mass and function, while also promoting the synthesis of adipose tissue, leading to obesity.[9] It not only increases the incidence of cardiovascular diseases and malignant tumors but also significantly raises the risk of dysfunction and death.[10]

As the global aging and population with obesity continues to grow, SO is becoming increasingly common among elderly individuals and individuals with obesity.[11,12] Consequently, bariatric surgeons are encountering a higher number of patients with SO. Some studies have found the existence of some factors affecting the weight loss effect of LSG,[13] but the analysis of the effects of SO on weight loss outcomes and recurrent weight gain after LSG is lacking. Hence, the purpose of this study was to investigate the possibility of SO affecting weight loss outcomes and recurrent weight gain following LSG using retrospective analysis of clinical data from 104 patients with obesity.

Materials and Methods

General Information

This study was a retrospective cohort analysis that gathered clinical data of 104 patients with obesity who underwent LSG in Nanchang University's First Affiliated Hospital between January 2020 and September 2023. The ratio of fat mass to lean mass ≥0.8 was calculated as SO based on preoperative third lumbar spine CT images. Among them, the SO group (n=34) and non-sarcopenic obesity (NSO) group (n=70). And SO's impact on weight loss outcomes and postoperative recurrent weight gain was compared.

Inclusion criteria: 1. Surgical indications as per the guidelines for weight loss surgery;[14] 2. Acceptance of LSG surgery; 3. Completion of postoperative follow-up on time with complete data. Exclusion criteria: 1. Missing clinical data and loss of postoperative visits; 2. Patients who underwent other types of bariatric surgery or those with poor baseline conditions were unable to stand the operation. This study received approval from the Ethics Committee of the First Affiliated Hospital of Nanchang University (Ethics number: IIT [2024] Clinical Ethics Review No. 209) and adhered to the Declaration of Helsinki. The committee waived the requirement for informed consent because this study was retrospective and the patient data were used solely for research purposes. Additionally, all patient data were anonymized and handled with strict confidentiality to ensure patient privacy.

Perioperative Management

Before surgery, all patients with obesity should have a comprehensive assessment involving various medical professionals such as endocrinologists, dietitians, and psychiatrists if there is a history of psychiatric illness. They should also undergo routine pre-surgical examinations, including gastroscopy, abdominal CT scans, sleep apnea monitoring, and so on. The LSG procedure can be performed after excluding contraindications to surgery.

After successful anesthesia, the surgeon inserted a 32 Fr gastric tube as an intragastric support tube in the patient with obesity. The procedure was generally performed using a three-hole approach to perforation. Further, the surgeon used an ultrasonic knife to completely free the gastric curvature and fundus along the direction of the gastric curvature starting from 2–6 cm from the pylorus. The starting point of cutting was 2–6 cm from the pylorus. The surgeon completely resected the fundus and most of the gastric curvature along the gastric support tube. Then, the omentum and the gastric wall are reinforced with surgical sutures. Afterward, the surgeon cleaned up the abdominal cavity, took out the specimen, and removed the intragastric support tube.

After surgery, the vital signs of patients were regularly supervised, and their diet was gradually adjusted based on their recovery progress. Typically, the patient was discharged three days after the surgery. At the time of discharge, the patient received dietary counseling, which included the following instructions: (1) strictly adhering to dietary guidelines and consuming approximately 2000 mL of water per day; (2) taking oral gastric medication for two weeks post-discharge and continuing with long-term supplementation of adequate multivitamins, and proteins,[15] followed by regular hospital visits to monitor nutritional levels.[16]

Research Indicators

The study indicators were collected by reviewing cases, image data, and telephone records, including (1) baseline indicators: gender, weight, body mass index (BMI), grip strength etc.; (2) body composition and SO: lean mass, fat mass, skeletal muscle area (SMA), and SO; (3) perioperative indicators: operation time, intraoperative bleeding, and postoperative complications; (4) Indicators for assessing weight loss effect and postoperative recurrent weight gain: excess body mass index (EMBI), excess body mass reduction percentage (EWL%), percentage of total weight loss (TWL%) etc.

Diagnosis of SO

In 2022, the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) established an expert consensus on the definition and diagnostic criteria for SO.[17] According to this consensus, diagnosing SO involves two stages: screening and diagnosis. A positive screening test indicates the presence of both clinical signs or suspected factors of sarcopenia and specific indicators of obesity. The clinical signs of sarcopenia include muscle weakness and fatigue. The suspected factors that contribute to sarcopenia are being over 70 years old, having chronic diseases (such as inflammatory diseases or depression), and experiencing recent acute illness or nutrition-related events (like major surgeries, reduced food intake, or weight loss). Additionally, the indicators of obesity consist of having a high BMI or elevated waist circumference measurements.

When a patient with suspected obesity screens positive and advances to diagnosis, the first step is to evaluate skeletal muscle function through a grip strength test. If the results indicate abnormal skeletal muscle function (with grip strength less than 28.0 kg for males or less than 18.0 kg for females[18]), the next step in the assessment of body composition is required. Current expert consensus recommends using both SMA and fat mass for this evaluation.[17] Research has demonstrated that the ratio of fat mass to lean mass offers a more accurate assessment of the risk for adverse outcomes associated with altered body composition.[19] Furthermore, this ratio is a superior indicator of body composition compared to BMI and abdominal circumference.[19,20] The ratio of fat mass to lean mass greater than 0.8 indicates more severe metabolic and cognitive abnormalities. Therefore, many studies utilize a ratio above 0.8 as a diagnostic criterion for SO.[19–22]

Expert consensus indicates that using abdominal CT or MRI provides an accurate assessment of skeletal muscle and fat content in the body.[23,24] Research has shown that the distribution of skeletal muscle and fat at the level of the third lumbar vertebra can effectively reflect overall body composition.[25] Therefore, in this study, we collected preoperative CT imaging data at the level of the third lumbar vertebra. We utilized Sliceomatic 5.0 software,[26] which applies different ranges of Hounsfield Units values for various tissue types,[27] to calculate the cross-sectional SMA at the third lumbar vertebra, as well as the subcutaneous adipose tissue area (SATA) and visceral adipose tissue area (VATA). Finally, we calculated the ratio of fat mass to lean mass using these formulas:[28] lean mass = 0.3 × SMA (cm²) + 6.06, and fat mass = 0.042 × total fat area at L3 (cm²) + 11.2.

Assessment of Surgical Weight Loss Effect and Postoperative Recurrent Weight Gain

One way of assessing surgical weight loss effect was by measuring changes in weight, BMI, EMBI, TWL% and EWL% of patients with obesity perioperatively, and another way was by using evaluation criteria. The evaluation criteria include the following:[29] (1) BMI ≥37.5 kg/m^2 is classified as obesity of degree III. (2) EBMI: EBMI = follow-up BMI - ideal BMI; ideal BMI =25 kg/m^2. (3) EWL%: EWL% = (preoperative BMI-follow-up BMI) / (preoperative BMI-ideal BMI) ×100%. (4) TWL%: TWL% = (preoperative BMI-follow-up BMI) / (preoperative BMI) ×100%. (5) Evaluation criteria of weight loss effect: EWL%≥100% defined as optimal weight loss; EWL%≥80% as good weight loss; 50%≤EWL% <80% as effective weight loss, and EWL%<50% as poor weight loss. (5) The definition of co-morbid remission: 1. Diabetes remission is characterized by a glycated hemoglobin level of less than 6.5% that is maintained for at least three months after discontinuing glucose-lowering medications. Additionally, improvement in diabetes is indicated by a reduced need for antidiabetic medications to maintain normal blood sugar levels. This includes either discontinuation of insulin or an oral medication or a reduction of glucose-lowering medications by half their original dose and maintaining this blood glucose level for at least three months. 2. Hypertension remission is defined by a sustained systolic blood pressure of less than 140 mmHg and a sustained diastolic blood pressure of less than 90 mmHg after stopping antihypertensive medications for at least three months. Improvement in hypertension is characterized by either a decrease in the dosage or number of antihypertensive medications required to maintain normal blood pressure or a significant reduction in systolic or diastolic blood pressure while on the same medications, with these changes lasting for three months. 3. Obstructive sleep apnea (OSA) remission is assessed through polysomnography, which must show an apnea-hypopnea index (AHI) of fewer than 5 breaths per hour, maintained for at least three months after stopping

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treatment with continuous positive airway pressure (CPAP) ventilation. Furthermore, improvement in OSA is demonstrated by a reduction in the pressure settings of CPAP therapy needed to maintain a normal lifestyle or a postoperative assessment, indicating that the AHI has significantly improved but does not meet the criteria for remission. This improvement should also last for about three months. 4. If there is no change in diabetes, hypertension, or OSA, it is defined by the disease severity and the intensity of treatment remaining similar to the preoperative situation.

Postoperative recurrent weight gain was evaluated as weight gain >10 kg after weight loss to the nadir in patients with obesity.[30]

Follow-up and Statistical Methods

Within one year of surgery, patients with obesity were advised to come to Bariatric Surgery Clinic for review at the 1st, 3rd, 6th, and 12th months after surgery, where they would be weighed, followed by appropriate nutritional assessment and clinical examination. Annual follow-up is recommended after the first year of surgery. Patients who did not come to the outpatient clinic for review were regularly followed up via telephone and WeChat.

This study used SPSS 25.0, GraphPad Prism 9.5.0 statistical software for statistical analysis of data, normal distribution of measurement data expressed as $x \pm s$, t-test was used for comparison between groups; comparisons between groups with skewed distributions were conducted using nonparametric tests; count data expressed as absolute numbers, χ^2-test was used for comparison between groups; according to the principle of statistical significance, single-factor and multi-factorial analyses were used to find out the risk factors for postoperative recurrent weight gain; $P<0.05$ indicated that the difference was statistically significant.

Results

Baseline Data Analysis

Among the 104 patients with obesity studied, there were more females than males, with 27 female patients (79.4%) in the SO group and 45 female patients (64.3%) in the NSO group. In addition, a majority of the patients also had OSA, with 64.7% in the SO group and 72.9% in the NSO group (Table 1).

As shown in Table 1, compared to NSO patients, the average preoperative BMI and fat mass of SO patients were higher; However, the average lean mass was lower for SO patients, and these differences were statistically significant ($P<0.001$). Moreover, the SO group had a significantly higher percentage of patients with grade III obesity compared to the NSO group (76.5% vs 35.7%, $P<0.05$). Additionally, SO patients exhibited a significantly higher mean SATA but a lower mean SMA compared to NSO patients, with both differences being statistically significant ($P<0.001$). There was no statistically significant difference between SO and NSO patients on VATA ($P=0.185$), despite SO patients having a greater mean value than NSO patients. Furthermore, diabetes, hypertension, and hepatorenal function were not statistically significantly different between the two groups ($P>0.05$).

Perioperative Analysis

Patients in both groups completed LSG with similar intraoperative bleeding and no intraoperative deaths or conversions to the open abdomen. The average postoperative hospital stays for both groups were 3.5 days, and none of the patients were readmitted within 30 days after discharge (Table 2). Additionally, both the SO and NSO groups had one case of abdominal bleeding, which was successfully treated with medication and without other complications. While the mean operating time was longer for NSO patients compared to SO patients, this difference lacked statistical significance ($P=0.082$).

Analysis of Surgical Weight Loss Results

All 104 patients with obesity completed postoperative follow-ups at the 1st, 3rd, 6th, and 12th months, with no patients lost to follow-up. Figure 1 shows that the patients' weight, BMI, and EBMI in both groups consistently decreased within one year after LSG, remaining lower than preoperative levels, accompanied by an increasing EWL% and TWL%. Additionally, SO patients had higher mean BMI and EBMI values and smaller mean EWL% values one year

Table 1 Comparative Analysis of the Baseline Data in SO and NSO Patients

Characteristic	SO (n=34)	NSO (n=70)	P-value
Sex, n (%)			
Male	7 (20.6)	25 (35.7)	0.117
Female	27 (79.4)	45 (64.3)	
Degree of obesity, n (%)			
I degree obesity	2 (5.9)	15 (21.4)	<0.001
II degree obesity	6 (17.6)	30 (42.9)	
III degree obesity	26 (76.5)	25 (35.7)	
Age (yr)	27.82 ± 7.94	30.89 ± 8.41	0.079
Weight (kg)	108.90 ± 17.87	102.32 ± 19.11	0.096
BMI (kg/m^2)	40.14 ± 4.02	36.27 ± 5.19	<0.001
Fat mass (kg)	42.75 ± 5.79	34.64 ± 6.39	<0.001
Lean mass (kg)	48.34 ± 7.63	56.11 ± 11.26	<0.001
SMA (cm^2)	140.93 ± 25.43	166.85 ± 37.55	<0.001
SATA (cm^2)	545.78 ± 114.60	372.14 ± 119.78	<0.001
VATA (cm^2)	205.37 ± 72.78	185.94 ± 68.21	0.185
AST (U/L)	36.59 ± 29.44	32.80 ± 20.38	0.447
ALT (U/L)	49.48 ± 40.37	46.69 ± 34.39	0.715
ALB (g/L)	40.33 ± 2.55	42.26 ± 5.33	0.049
Cr (u mol/L)	55.35 ± 11.13	75.83 ± 121.65	0.331
UA (mmol/L)	469.53 ± 95.79	436.84 ± 109.96	0.142
BUN (u mol/L)	4.46 ± 0.93	4.95 ± 3.35	0.401
TG (mmol/L)	1.62 ± 0.66	2.71 ± 4.43	0.048
TC (mmol/L)	4.39 ± 0.88	4.63 ± 1.26	0.307
HDL-L (mmol/L)	1.09 ± 0.34	1.04 ± 0.33	0.402
LDL-L (mmol/L)	2.89 ± 0.63	2.93 ± 1.01	0.824
Comorbidities, n (%)			
DM			
Absent	27 (79.4)	56 (80.0)	0.944
Present	7 (20.6)	14 (20.0)	
HTN			
Absent	31 (91.2)	58 (82.9)	0.257
Present	3 (8.8)	12 (17.1)	
OSA			
Absent	11 (32.4)	19 (27.1)	0.394
Present	23 (67.6)	51 (72.9)	

Abbreviations: SO, sarcopenic obesity; NSO, non-sarcopenic obesity; BMI, body mass index; SMA, skeletal muscle area; SATA, subcutaneous adipose tissue area; VATA, Visceral adipose tissue area; ALT, alanine aminotransferase; AST, aspartate transaminase; ALB, albumin; Cr, creatinine; UA, uric acid; BUN, blood urea nitrogen; TC, total cholesterol; TG, triglyceride; HDL-L, high-density lipoprotein-cholesterol; LDL-L, low-density lipoprotein-cholesterol; DM, diabetes mellitus; HTN, hypertension; OSA, obstructive sleep apnea.

postoperatively than NSO patients, with statistically significant differences (P<0.01). AS shown in Table 3, our study revealed that 103 out of 104 patients with obesity (99%) had a favorable surgical weight loss effect one year after the operation. At the same time, patients in the SO group and the NSO group had a 33% and 32% reduction in total body weight at 1 year after surgery, respectively. Specifically, in the SO group, 70.6% of patients achieved good weight loss, while 82.9% did in the NSO group. Although the percentage of patients with good weight loss was higher in the NSO group, it did not reach statistical significance (P>0.05) Furthermore, our findings indicated that in the SO and NSO groups, 35.3% and 58.6% of patients achieved optimal weight loss separately, demonstrating that there is a statistically significant difference between the two groups (P=0.026).

In addition to this, this study also observed a great degree of remission of combined diabetes, hypertension, and OSA after LSG in both SO and NSO patients (Table 3). Among patients with diabetes, the remission rates in the NSO and SO groups

Table 2 Analysis of Perioperative Indicators After LSG in SO and NSO Patients

Characteristic	SO (n=34)	NSO (n=70)	P-value
Operation time (min)	154.12 ± 33.15	167.03 ± 36.14	0.082
Estimated blood loss (mL)	68.24 ± 45.02	64.00 ± 49.52	0.675
Postoperative complication, n (%)	1 (2.9)	1 (1.4)	0.598
Postoperative hospitalization days	3.53 ± 1.16	3.50 ± 0.83	0.882

Abbreviations: SO, sarcopenic obesity; NSO, non-sarcopenic obesity; LSG, laparoscopic sleeve gastrectomy.

were 100% and 71.4% respectively, this difference was statistically significant (P=0.04). Additionally, we found that the remission rates of hypertension and OSA in the SO group were 100.0% and 87.0%, respectively, whereas the remission rates in the NSO group were 75.0% and 74.5%, but none of the differences were statistically significant (P>0.05).

Postoperative Recurrent Weight Gain and Risk Factor Analysis

In this study, 74 eligible patients with obesity were followed up in the 2nd year after surgery, and no patient was lost to follow-up. According to Table 3, it was found that 27 patients (36.5%) out of 74 patients with obesity had recurrent weight gain within two years after LSG. Among these patients, 6 (26.1%) were in the SO group and 21 (41.2%) were in the NSO group.

As a result, NSO patients had a higher rate of postoperative recurrent weight gain than SO patients, but this difference was not statistically significant (P>0.05).

In addition, the study examined the factors contributing to recurrent weight gain after LSG in patients with obesity. Table 4 demonstrates that diabetes and lean mass was linked to recurrent weight gain after surgery (P<0.05) based on the

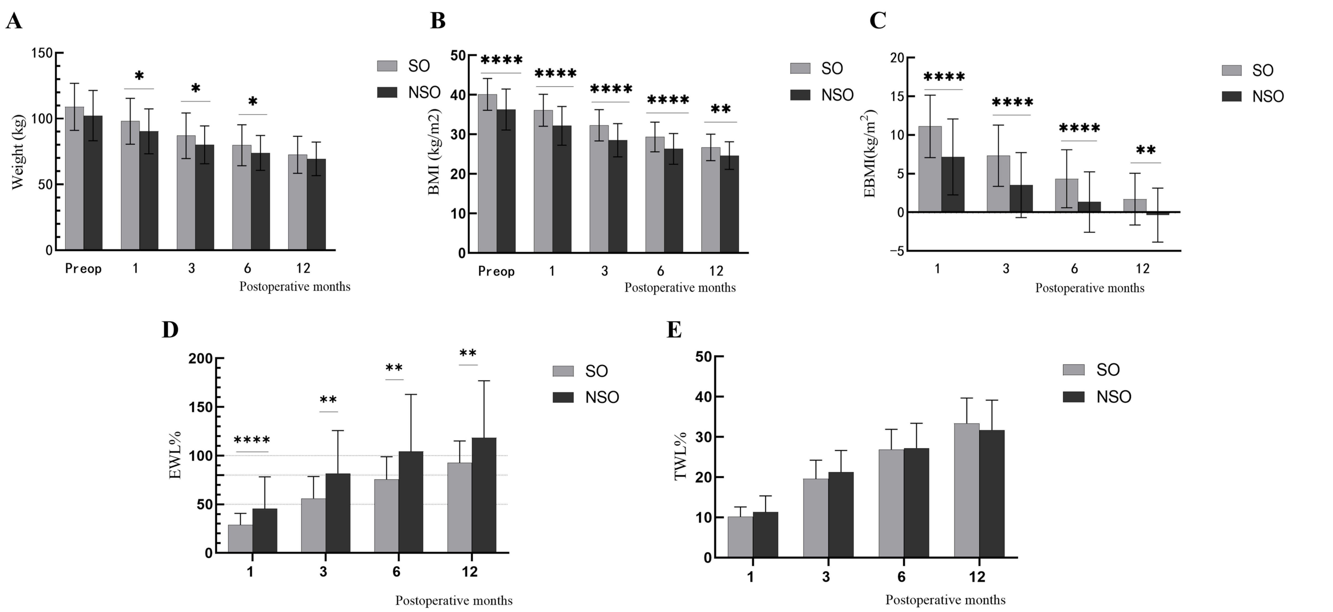

Figure 1 Weight loss results within one year after LSG in SO and NSO patients. (**A**) Changes in weight within one year after LSG in SO and NSO patients. (**B**) Changes in BMI within one year after LSG in SO and NSO patients. (**C**) Changes in EBMI within one year after LSG in SO and NSO patients. (**D**) Changes in EWL% within one year after LSG in SO and NSO patients. (**E**) Changes in TWL% within one year after LSG in SO and NSO patients. In Figure (**D**), distinct horizontal lines are added at the EWL% values of 50%, 80%, and 100%. * P<0.05; ** P<0.01; **** P<0.0001; Error bars indicate Standard deviation.

Abbreviations: SO, sarcopenic obesity; NSO, non-sarcopenic obesity; LSG, laparoscopic sleeve gastrectomy; BMI, body mass index; EWL%, percentage of excess weight loss; EMBI, excess body mass index; TWL%, percentage of total weight loss; Preop, preoperative.

Table 3 Analysis of the Effectiveness of Weight Loss Following LSG in SO and NSO Patients

Characteristic	SO (n=34)	NSO (n=70)	P-value
Optimal weight loss, n (%)	12 (35.3)	41 (58.6)	0.026
Good weight loss, n (%)	24 (70.6)	58 (82.9)	0.151
Effective weight loss, n (%)	10 (29.4)	11 (15.7)	0.103
Comorbidity remission			
DM, n (%)			
Remission	5 (71.4)	14 (100.0)	0.040
Improvement	1 (14.3)	0	
Unchanged	1 (14.3)	0	
HTN, n (%)			
Remission	4 (100.0)	9 (75.0)	0.285
Improvement	0	2 (16.7)	
Unchanged	0	1 (8.3)	
OSA, n (%)			
Remission	20 (87.0)	38 (74.5)	0.425
Improvement	3 (13.0)	13 (25.5)	
Recurrent weight gain, n (%)	6 (26.1)	21 (41.2)	0.212

Abbreviations: SO, sarcopenic obesity; NSO, non-sarcopenic obesity; LSG, laparoscopic sleeve gastrectomy; DM, diabetes mellitus; HTN, hypertension; OSA, obstructive sleep apnea.

univariate analysis. Further multifactorial analysis indicated that diabetes was an independent risk factor for post-operative recurrent weight gain in patients with obesity (P=0.042).

Discussion

When analyzing the baseline data, this study observed that the average preoperative BMI, SATA, and the percentage of patients with grade III obesity in the SO patients were higher than in NSO patients, and these differences were statistically significant (P

Table 4 Univariate and Multivariate Analysis of Factors Associated with Recurrent Weight Gain Within 2 years After LSG in a Whole Group of Patients

Characteristic	Univariable[a]		Multivariable[b]	
	OR (95% CI)	P	OR (95% CI)	P
DM				
Absent	1 (Reference)		1 (Reference)	
Present	3.36 (1.10–10.30)	0.034	3.34 (1.04–10.68)	0.042
SO				
Absent	1 (Reference)			
Present	0.50 (0.17–1.49)	0.216		
Degree of obesity				
I degree obesity	1 (Reference)			
II degree obesity	0.57 (0.13–2.42)	0.441		
III degree obesity	0.70 (0.18–2.72)	0.607		
Age (yr)	1.06 (0.99–1.13)	0.061		
Weight (kg)	1.01 (0.99–1.04)	0.376		
BMI (kg/m^2)	0.98 (0.88–1.08)	0.631		
Fat mass (kg)	1.05 (1.00–1.10)	0.044	1.05 (0.99–1.10)	0.054

(*Continued*)

Table 4 (Continued).

Characteristic	Univariable[a]		Multivariable[b]	
	OR (95% CI)	P	OR (95% CI)	P
Lean mass (kg)	1.00 (0.93–1.08)	0.940		
AST (U/L)	1.00 (0.98–1.02)	0.856		
ALT (U/L)	1.00 (0.99–1.02)	0.682		
ALB (g/L)	1.06 (0.95–1.18)	0.285		
Cr (u mol/L)	1.00 (0.99–1.02)	0.494		
BUN (u mol/L)	1.25 (0.81–1.92)	0.312		
TG (mmol/L)	1.05 (0.94–1.18)	0.410		
TC (mmol/L)	1.01 (0.69–1.48)	0.950		
HDL-L (mmol/L)	0.39 (0.08–1.95)	0.251		
LDL-L (mmol/L)	1.02 (0.62–1.68)	0.947		

Notes: a, Single -factor logistics regression analysis. b, Multi -factor logistics regression analysis.
Abbreviations: DM, diabetes mellitus; SO, sarcopenic obesity; BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate transaminase; ALB, albumin; Cr, creatinine; BUN, blood urea nitrogen; TC, total cholesterol; TG, triglyceride; HDL-L, high-density lipoprotein-cholesterol; LDL-L, low-density lipoprotein-cholesterol.

<0.05). These statistically significant indicators suggest that patients in the SO group were more obese. A clinical study involving 972 patients with obesity reached a similar conclusion.[31] These significant differences observed may stem from the interplay between sarcopenia and obesity. In these conditions, metabolic disturbances—such as chronic inflammation and hormonal imbalances—are more pronounced, resulting in the degradation of lean body tissue and an increase in inflammatory factors. This ultimately promotes the accumulation of adipose tissue.[32] Consequently, requiring more attention from clinicians.

By analyzing the perioperative data, our study found similar results for operative time, intraoperative bleeding, postoperative hospital stays, and postoperative complications between patients in the SO and NSO groups. Another cohort study with 245 patients with obesity also showed partially similar results but noted significant differences in the duration of surgery and postoperative hospital stays between the SO and NSO groups[20] ($P<0.001$). Its study attributed the significant difference to the greater area of visceral fat in the SO group. However, there may be individual differences in the body composition characteristics of SO patients.[33] Our study found that the difference in the VATA between the SO and NSO groups was not statistically significant ($P=0.185$, Table 1). Therefore, our study yielded the opposite result.

Many studies have found that weight reduction and improvement of comorbid diseases tend to stabilize within one year after LSG,[34] so we analyzed the weight loss results by examining the follow-up data of patients with obesity during the first year after surgery. Our findings revealed that the weight, BMI, and severity of comorbid diseases of patients with obesity in both groups were lower than the preoperative levels. And the EWL% and TWL% values gradually increased compared to those before the surgery. Similar findings have been reported in other studies; for example, A meta-analysis that included 2300 young patients with obesity found a mean BMI reduction of 17.8 kg/m^2 in patients with obesity after LSG and some relief from comorbidities such as diabetes and hypertension.[35] This reduction may be attributed to the decreased stomach volume and a decrease in the secretion of hunger hormones after surgery, leading to restricted food intake and subsequent weight loss.[36] Further, insulin resistance and inflammatory response improve, thereby assisting in the alleviation of diabetes, hypertension, and other co-morbidities.[37] Simultaneously, this study identified that SO patients had higher mean BMI, EBMI values, and smaller mean EWL% values one year postoperatively than NSO patients, with statistically significant differences ($P<0.01$). These results imply that SO probably negatively impacts weight loss outcomes following LSG. Furthermore, another study reported that SO led to a significant elevation in leptin levels among patients with obesity.[38] While LSG has the potential to ameliorate leptin resistance in patients with obesity,[39] the presence of SO results in persistently elevated leptin levels post-surgery. This exacerbates leptin resistance, subsequently influencing postoperative weight loss.

Some researchers have identified a strong link between SO and various comorbidities, including diabetes, hypertension, and OSA. SO can exacerbate these conditions through mechanisms such as increased insulin resistance, vascular dysfunction, and chronic inflammation.[31,40,41] Our study found that patients in the SO group experienced worse postoperative diabetes remission compared to those in the NSO group, and this difference was statistically significant. This finding aligns with the above studies.

Surprisingly, however, we discovered that patients in the NSO group had lower rates of remission for both hypertension and OSA than those in the SO group, although this difference was not statistically significant (P>0.05). Additionally, a cohort study involving 245 patients with obesity reported similar results.[20] Given the limited research on the impact of SO on the treatment of comorbidities in patients with obesity undergoing LSG, as well as the small number of patients included in existing studies with comorbidities, our findings may be subject to bias. Therefore, larger-scale, multicenter clinical studies are necessary to further explore the effect of SO on the efficacy of postoperative relief from comorbidities in patients with obesity. Evidence suggests that nutritional and exercise interventions are effective in mitigating SO;[42] therefore, patients with obesity with SO require more rigorous nutritional and exercise management.

Current evaluation metrics for surgical weight loss outcomes advocate the use of EWL% or TWL%.[43] In contrast to previous research, our study offers a comprehensive analysis of surgical weight loss outcomes in patients with obesity by examining the EWL% in the first postoperative year. Our findings indicate that SO patients exhibited lower rates of optimal weight loss and good weight loss compared to NSO patients. This provides additional evidence that SO negatively impacts short-term weight loss outcomes after LSG.

Numerous studies have indicated that the first year after LSG is characterized by a high prevalence of postoperative recurrent weight gain.[44] Consequently, in our study, 74 eligible patients with obesity were followed up in the 2nd year after surgery. Our findings revealed a postoperative recurrent weight gain rate of 36.5%. A review examined six different diagnostic criteria for postoperative recurrent weight gain after bariatric surgery. It concluded that the typical range of recurrent weight gain post-surgery is between 9% and 91%.[45] This suggests that the rate of postoperative recurrent weight gain observed in our study falls within a reasonable range. Furthermore, our study observed that NSO patients had a higher rate of postoperative recurrent weight gain than SO patients, this difference was not statistically significant (P=0.212). This discrepancy may be attributed to the more gradual nature of postoperative weight changes in the SO group, and the time required to achieve minimum body weight was extended in SO patients than NSO patients,[20] resulting in a reduced number of cases meeting the criteria for postoperative recurrent weight gain. However, it is possible that the small sample size led to a Type II error, rendering the differences between the two groups statistically insignificant. To date, no scholarly investigations have examined the impact of SO on postoperative recurrent weight gain following LSG in patients with obesity, this study provides a new direction for research in this area. At the same time, Further studies with larger samples are necessary to clarify the impact of SO on recurrent weight gain following LSG.

Additionally, this study investigated the risk factors associated with postoperative recurrent weight gain in the patients with obesity, revealing that diabetes serves as an independent risk factor. The relationship between diabetes and obesity is significant, as they synergistically exacerbate insulin resistance in obese individuals. This interaction is characterized by a marked elevation in insulin levels, subsequently promoting recurrent weight gain.[37] Additionally, a more stringent follow-up protocol should be implemented for patients with obesity with diabetes to address and mitigate postoperative recurrent weight gain promptly.

This study also has some limitations. First, this study is a retrospective clinical study, and there may be recall bias or potential confounding bias in the study, which may affect the results; second, the number of cases in this study is not rich enough, especially the cases of co-morbidities and postoperative recurrent weight gain, which may bias the assessment of comorbidities and postoperative recurrent weight gain, and therefore further studies with multi-centers and large sample sizes are needed; Third, the generalizability of the findings may be limited; this study is currently focused on 2-year follow-up after LSG, so it can only investigate the effect of SO in short-term weight loss efficacy after LSG; however, the role on mid- and long-term postoperative weight loss efficacy is still unclear, and longer follow-up observation is needed.

Conclusion

SO adversely affects short-term weight loss outcomes following LSG. However, the effect of SO on postoperative recurrent weight gain was not statistically significant, indicating that further research is needed. Furthermore, diabetes serves as an independent risk factor for postoperative recurrent weight gain in the patients with obesity. Consequently,

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bariatric surgeons should be taken more stringent dietary and exercise regimens, alongside comprehensive follow-up strategies for patients with obesity with concurrent SO and diabetes.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All authors declare no conflict of interest.

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