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Metabolic Syndrome Modifies Changes in Body Composition After Sleeve Gastrectomy: A Quantitative CT Study

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Objective: To investigate changes in body mass index (BMI), body composition, and laboratory indicators in obese patients with different metabolic status after laparoscopic sleeve gastrectomy (LSG).

Methods: This prospective study included 52 obese patients who underwent LSG. Clinical and quantitative CT-based body composition data were collected preoperatively and at the 12-month follow-up. Patients were categorized into metabolic syndrome (MS) and non-MS groups. Changes in BMI, body composition, and laboratory indicators after LSG were compared between groups, and the correlations between body composition and clinical indicators—at both baseline and postoperatively—were analyzed, stratified by MS status.

Results: Among 52 obese patients undergoing LSG, 24 (46%) had MS. All patients exhibited significant postoperative improvements in body weight, laboratory parameters, and body composition. The postoperative visceral fat area showed the greatest improvement ratio [non-MS: 0.62 (0.48, 0.70); MS: 0.60 (0.49, 0.67)] among abdominal fat compartments compared to preoperative levels, while liver fat (LF) demonstrated the most marked improvement [non-MS: 0.53 (0.24, 0.60); MS: 0.55 (0.28, 0.66)] among ectopic fat deposits. However, reductions in BMI ($P = 0.036$) and postvertebral muscle fat content ($P = 0.041$) were greater in the non-MS group. At the 12-month follow-up, changes in LF positively correlated with changes in blood glucose ($r = 0.414$, $P = 0.044$) in the MS group, whereas changes in BMI correlated with changes in triglyceride ($r = 0.427$, $P = 0.023$) and low-density lipoprotein ($r = 0.480$, $P = 0.01$) in the non-MS group.

Conclusion: LSG significantly improves body composition and metabolic parameters in obese patients. The correlations between body composition and laboratory indicators, both at baseline and in their postoperative changes, differed according to metabolic status. These findings suggest that a comprehensive assessment of body composition and metabolic markers in patients undergoing bariatric surgery enables targeted interventions against the key risk factors associated with distinct metabolic phenotypes.

Keywords: metabolic syndrome, obesity, CT, bariatric surgery

Introduction

Obesity results from a complex interplay of genetic, environmental, and behavioral factors and is a growing public health concern across all age groups.[1,2] It is frequently accompanied by metabolic abnormalities, including elevated triglycerides (TG), reduced high-density lipoprotein cholesterol(HDL-C), hypertension, and an increased fasting blood glucose.[3–5] The presence of three or more of these components concurrently is defined as metabolic syndrome (MS). It is important to recognize that obesity is not a homogeneous condition, often accompanied by different pathophysiological features, such as insulin resistance and chronic inflammation. Although elevated body mass index (BMI) is a criterion that remains constant, different obesity types—distinguished by patterns of fat deposition—can constitute distinct phenotypes of specific diseases.[6,7] Therefore, knowledge of the pattern of fat distribution would be helpful in the management of patients with obesity and MS. Quantitative computed tomography (QCT)allows precise quantification of fat (including abdominal and ectopic fat content),

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distribution of muscle tissue, and bone mineral density[8] and is a valuable tool for detection of changes in body composition. The current therapeutic approach for patients with obesity is laparoscopic sleeve gastrectomy (LSG), which is widely used because of its favorable effects in terms of weight loss and few complications.

Based on our previous study,[9] although obese patients showed significant improvements in body composition and metabolic indicators after LSG, no differences were observed between the MS and non-MS groups due to limited sample size. Recent studies also have highlighted the dynamic changes in ectopic fat depots following bariatric surgery, with patterns varying according to metabolic phenotypes.[10] To address this, the present study expanded the sample size to investigate whether there are differences in the degree of improvement in body composition and metabolic indicators between the two groups after LSG, and to evaluate the correlation of body composition and metabolic indicators between the two groups before and after LSG.

Materials and Methods

Study Population

Obese patients who participated in prospective clinical trials of LSG and underwent baseline QCT at our university-affiliated teaching hospital between January 2021 and May 2024 were retrospectively enrolled.

This study was conducted in accordance with the Declaration of Helsinki.

The study inclusion criteria were as follows: (1) age 18–65 years; (2) BMI ≥ 32.5 kg/m^2 with or without type 2 diabetes; (3) BMI 27.5–32.5 kg/m^2 with type 2 diabetes, suboptimal blood glucose control despite lifestyle interventions and optimal medical therapy; and the presence of obesity-related complications, such as nonalcoholic fatty liver disease, hyperuricemia, or polycystic ovary syndrome. The following exclusion criteria were applied: (1) incomplete clinical or imaging data available; (2) concomitant systemic disease, such as hypothyroidism; (3) another cause of metabolic abnormalities, such as use of certain medications (eg, glucocorticoids) or alcohol misuse; and untreated psychiatric illness.

Clinical and Laboratory Data

We collected general demographic and clinical data for all patients both preoperatively (one day before surgery) and at the 12-month postoperative follow-up. BMI was calculated as body weight (kg) divided by the square of height (m). Laboratory data included total cholesterol (TC), TG, HDL-C, low-density lipoprotein cholesterol (LDL-C), glycated hemoglobin, and plasma glucose. The rate of change in BMI and laboratory data was calculated as (preoperative data minus postoperative data)/preoperative data.

Diabetes was defined as a fasting blood glucose ≥ 7.0 mmol/L, a glycated hemoglobin ≥ 6.5%, or current use of antidiabetic medication. Hyperlipidemia was defined as a TG ≥ 1.7 mmol/L or HDL-C < 1.04 mmol/L. Hypertension was defined as systolic and/or diastolic blood pressure ≥ 140/90 mmHg or current use of antihypertensive medication.[11]

Acquisition of QCT Images

QCT examinations were conducted with a dual source CT scanner (SOMATOM Definition Flash; Siemens Healthcare). Scans covered the area from the lung apex to the third lumbar vertebra (L3). The scanning protocol included automatic current modulation, a tube voltage of 120 kV, a pitch of 1.75:1, a scan field of view of 500 mm, and a table height of 135 mm. Images were reconstructed using a 512×512 matrix and a bone algorithm, with both slice thickness and interslice spacing set at 2 mm. For quality control, QCT phantoms (model 4, QCTPro; Mindways) were scanned weekly, using the same parameters as patient scans.

Assessment of Body Composition

Body composition was quantified using Mindways QCT BMD software (QCT PRO), as illustrated in Figure 1. At the L2/3 disc level, the contour of the abdominal musculature was manually delineated using the Activate Closed Spline tool. The software subsequently automatically calculated the total fat areas (TFA), subcutaneousfat areas (SFA), and visceral fat areas (VFA) at this level. The skeletal muscle area (SMA) at L2/3 was manually marked, and the system generated the

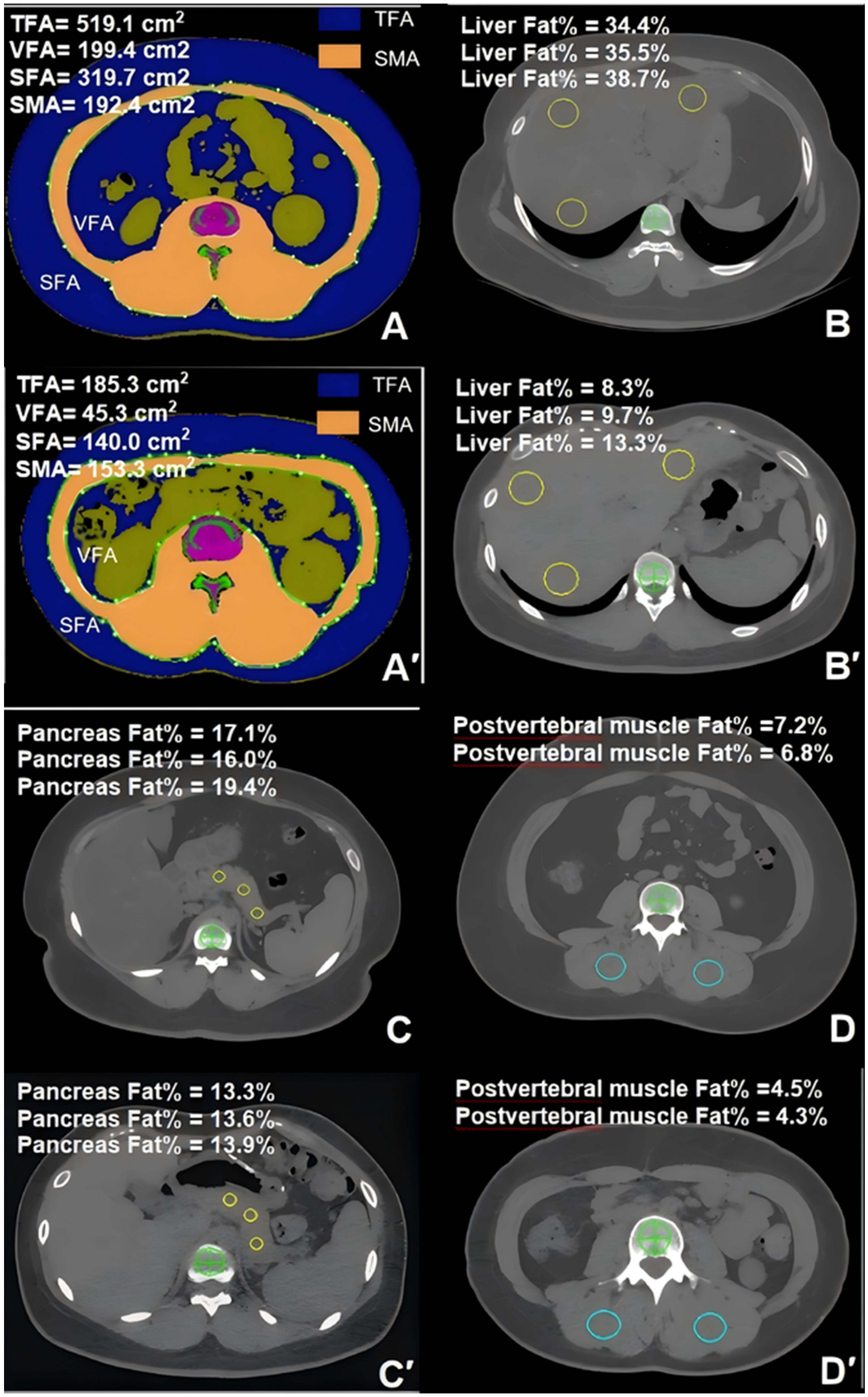

Figure 1 Comparison of body composition measured by quantitative CT in a 20-year woman before and after LSG. (**A**–**D**) show the measurements for TFA, VFA, SFA, SMA, LF, PF, and PMFC on the day before LSG. (**A′**–**D′**), show the corresponding measurements at 12 months postoperatively. VFA and LF showed the most significant improvement.

Abbreviations: LSG, laparoscopic sleeve gastrectomy; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMA, skeletal muscle area; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

corresponding area measurement. The skeletal muscle index (SMI) was determined by dividing the muscle area by the square of the patient's height. For the paraspinal muscles at the midpoint of L3, two regions of interest (ROIs) of 100 mm^2 each were selected symmetrically on both sides. The muscle fat percentage was derived from these ROIs, and the mean postvertebral muscle fat content (PMFC) was computed. The average percentage of liver fat (LF) was calculated. At the level of the right portal vein branch, ROIs measuring 250 mm^2 were placed in both the anterior and posterior segments of the right hepatic lobe, as well as in the left hepatic lobe, with care taken to avoid intrahepatic vessels and bile ducts. The average percentage values for pancreatic fat (PF) were also obtained. ROIs of 100 mm^2 were chosen in the head, body, and tail of the pancreas, excluding vessels, ducts, and surrounding fat tissue. Quantitative body composition measurements were independently conducted by two radiologists (with 7 and 3 years of experience in abdominal imaging) in 20 randomly selected cases to assess interobserver agreement using the intraclass correlation coefficient (ICC). An ICC value greater than 0.85 was considered to indicate good interobserver reliability. If the measurements showed good consistency, the results from the senior radiologist were used as the final outcome. The rate of change for each body composition parameter was calculated using the formula: (preoperative value - 12-month post-LSG value) / preoperative value.

Definition of MS

MS was diagnosed when a patient presented with three or more of the following components: (1) central obesity (waist circumference ≥ 85 cm in women and ≥ 90 cm in men); (2) HDL-C < 1.04 mmol/L; (3) TG ≥ 1.7 mmol/L; (4) systolic or diastolic blood pressure ≥ 130/85 mmHg or treatment for hypertension; and (5) fasting blood glucose ≥ 6.1 mmol/L or use of antidiabetic medication.[12] Based on these criteria, patients were categorized into MS and non-MS group.

Statistical Analysis

Continuous variables with a normal distribution are expressed as the mean ± standard deviation, while those with non-normal distribution are shown as the median (interquartile range). Categorical variables were summarized as numbers (percentages). Independent t-tests or the Mann–Whitney *U*-test for continuous variables and the chi-squared test for categorical variables were used to compare differences between two groups. Changes in laboratory indicators and body composition parameters from baseline (one day before LSG) to the 12-month follow-up were assessed using paired t-tests. The magnitude of change in these parameters between the MS and non-MS groups was compared using independent samples t-tests. Repeated measures ANOVA was conducted to evaluate changes between the MS and non-MS groups, post-hoc pairwise comparisons were performed with Bonferroni correction for multiple comparisons. Correlations between site-specific fat depots and laboratory indicators were assessed by calculating Spearman correlation coefficients. A two-sided p-value of < 0.05 was considered statistically significant. All statistical analyses were performed using IBM SPSS version 24.0 for Macintosh (IBM Corp).

Results

A total of 108 consecutive patients underwent baseline QCT prior to LSG during the study period. After excluding 56 patients due to missing 12-month follow-up data (n=52) or poor image quality (n=4), 52 patients with complete clinical, laboratory, and imaging data were included in the final analysis (Figure 2). A comparison of baseline characteristics between included and excluded participants showed no significant differences (Table S1). The cohort comprised 24 patients in MS group and 28 patients in the non-MS group. The ICC values for all body composition measurements were greater than 0.90, indicating excellent interobserver reliability (Table S2).

Changes in body composition and clinical data from baseline to the 12-month follow-up are summarized in Table 1. Following LSG, all patients exhibited significant reductions in all laboratory indicators (except for LDL-C and TC) and body composition measures (all P < 0.05). At baseline, the MS group had significantly higher values for all laboratory parameters (except TC and LDL-C) and LF compared to the non-MS group (all P < 0.05). At the 12-month follow-up, however, no significant differences in laboratory indicators or body composition were observed between the two groups, with the exception of LDL-C (Tables 2 and 3).

The rates of change in laboratory parameters and body composition, both within and between the MS and non-MS group, are compared in Table 4. Among the abdominal fat compartments (total, visceral, and subcutaneous), the VFA

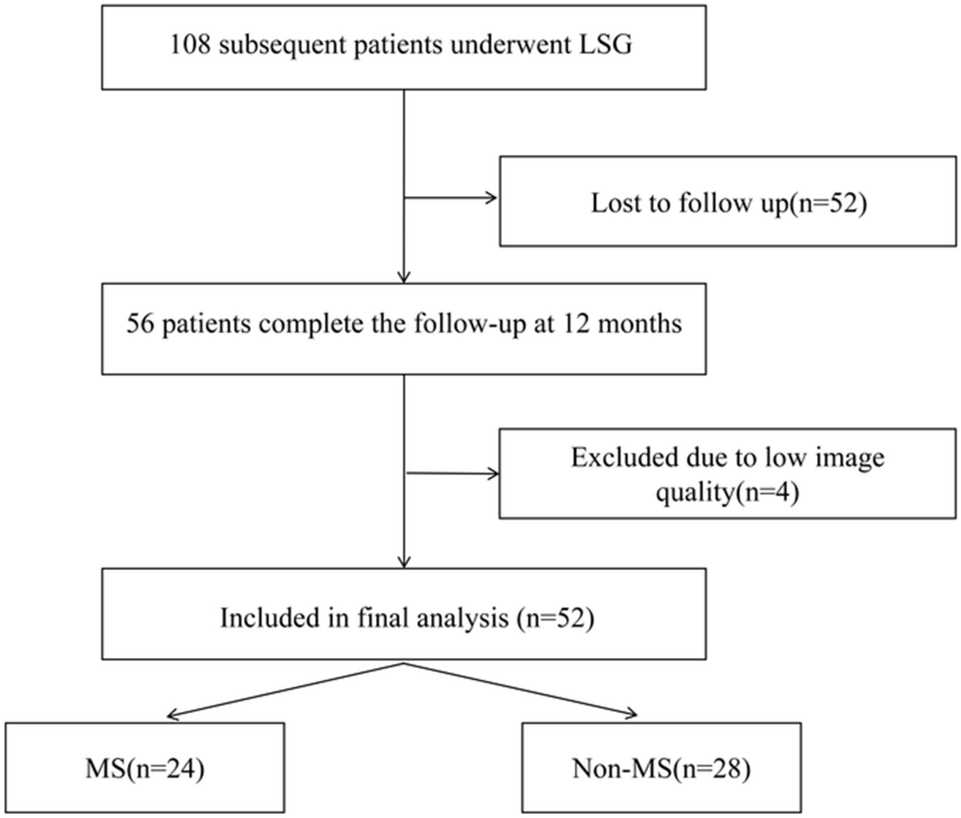

Figure 2 Flowchart showing the process used to select the study participants.
Abbreviations: LSG, laparoscopic sleeve gastrectomy; MS, metabolic syndrome.

demonstrated the highest rate of reduction (non-MS group, 0.62 [0.48, 0.70]; MS group, 0.60 [0.49, 0.67]). Similarly, among the ectopic fat depots, the LF showed the greatest change (non-MS group, 0.53 [0.24, 0.60]; MS group, 0.55 [0.28, 0.66]). The MS group exhibited more pronounced improvements in most laboratory indicators (except TC) and diastolic blood pressure. In contrast, the reduction in both BMI and PMFC was significantly greater in the non-MS group (both $P < 0.05$). Repeated measures ANOVA revealed a significant main effect of time on PMFC in the overall cohort (P for time < 0.001). In addition, a significant time × group interaction was observed (P for interaction = 0.023). Post-hoc

Table 1 Longitudinal Changes in Clinical Data and Body Composition of Obese Patients One Day Before and After LSG

Varibles	1day Before LSG (n=52)	12 Months After LSG (n=52)	P
BMI, kg/m^2	37.69±4.82	25.77±3.73	**< 0.001**
Systolic blood pressure, mmHg	133 (125, 149.5)	120 (110, 121)	**< 0.001**
Diastolic blood pressure, mmHg	83.5 (75.25, 90.75)	70 (61.25, 80)	**< 0.001**
Hypertension, %	40% (21/52)	13% (7/52)	**< 0.001**
TC, mmol/L	4.73±0.78	4.84±0.75	0.280
TG, mmol/L	1.96 (1.29, 2.68)	1.01 (0.81, 1.26)	**< 0.001**
HDL, mmol/L	1.25±0.21	1.44±0.27	**< 0.001**
LDL, mmol/L	2.98±0.68	2.94±0.67	0.683
Hyperlipidemia, %	65.3% (34/52)	11.5% (6/52)	**< 0.001**
HbA1c, %	5.6 (5.4, 6.38)	5.1 (5, 5.2)	**< 0.001**

(*Continued*)

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Table 1 (Continued).

Varibles	1day Before LSG (n=52)	12 Months After LSG (n=52)	P
Glucose, mmol/L	5.6 1 (4.87, 6.38)	5.08 (4.74, 5.19)	**< 0.001**
Diabetes, %	25% (13/52)	4% (2/52)	**0.001**
MS, %	46.1% (24/52)	0% (0/52)	**< 0.001**
TFA, cm^2	632.87±149.52	307.36±106.18	**< 0.001**
VFA, cm^2	230.38±77.60	95.58±46.40	**< 0.001**
SFA, cm^2	402.49±120.62	211.78±85.47	**< 0.001**
LF, %	20.58±7.41	9.76±2.73	**< 0.001**
PF, %	20.45 (16.15, 30.13)	15.6 (10, 19.68)	**< 0.001**
SMI, cm^2/m^2	64.72±10.96	49.9±8.78	**< 0.001**
PMFC, %	7.9 (6.02, 10.32)	5.9 (4.25, 7.83)	**< 0.001**

Notes: Descriptive statistics were expressed as mean±standard deviation or median (interquartilerange). Bolded numbers indicate statistical significance.

Abbreviations: LSG, laparoscopic sleeve gastrectomy; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MS; metabolic syndrome; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMI, skeletal muscle index; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

Table 2 Comparison of Clinical Data and Body composition Between MS and Non-MS Obese Patients 1 Day Before LSG

Varibles	Non-MS (n=28)	MS (n=24)	p
BMI kg/m^2	37.93±5.05	37.40±4.64	0.696
Age, in years	31.00±8.56	31.54±6.14	0.797
Sex (M/F), %	21.4% (6/28)	29% (7/24)	0.521
Systolic blood pressure, mmHg	130 (122, 144)	138.5 (125.75, 153.5)	0.208
Diastolic blood pressure, mmHg	80 (73, 89)	86 (75.5, 92.0)	0.274
Hypertension, %	35.7% (10/28)	45.8% (11/24)	0.458
TC, mmol/L	4.57±0.78	4.93±0.76	0.101
TG, mmol/L	1.44 (1.1, 2.1)	2.92 (1.75, 3.50)	**< 0.001**
HDL, mmol/L	1.34±0.16	1.15±0.21	**0.001**
LDL, mmol/L	2.94±0.63	3.03±0.75	0.648
Hyperlipidemia, %	43% (12/28)	92% (22/24)	**< 0.001**
HbA1c,%	5.4 (5.23, 5.58)	6.40 (5.93, 7.5)	**< 0.001**
Glucose, mmol/L	5.14 (4.65, 5.64)	6.4 (5.42, 7.22)	**< 0.001**
Diabetes, %	0% (0/28)	54% (13/24)	**< 0.001**
TFA, cm^2	629.13±164.16	637.23±133.79	0.848
VFA, cm^2	222.69±77.38	239.36±78.53	0.445
SFA, cm^2	406.44±130.56	397.87±110.47	0.801
LF,%	18.66±6.47	22.83±7.93	**0.042**
PF,%	19.75 (15.08, 27.90)	20.90 (17.95, 31.28)	0.313
SMI, cm^2/m^2	62.18±10.04	67.70±11.06	0.07
PMFC,%	8.25 (6.2, 10.55)	7.45 (5.68, 9.95)	0.186

Notes: Descriptive statistics were expressed as mean±standard deviation or median (interquartilerange), Bolded numbers indicate statistical significance.

Abbreviations: MS, metabolic syndrome; LSG, laparoscopic sleeve gastrectomy; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMI, skeletal muscle index; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

Table 3 Comparison of Clinical Data and Body Composition Between MS and Non-MS Obese Patients After LSG

Varibles	Non-MS (n=28)	MS (n=24)	p
BMI, kg/m^2	25.33±4.10	26.28±3.25	0.367
Systolic blood pressure, mmHg	120 (110, 120.75)	120 (110,121.75)	0.838
Diastolic blood pressure, mmHg	70 (65, 80)	70 (60, 79.5)	0.675
Hypertension, %	11% (3/28)	17% (4/24)	0.826
TC, mmol/L	4.74 (4.23, 4.84)	4.84 (4.56, 5.74)	0.050
TG, mmol/L	1.02±0.35	1.04±0.33	0.815
HDL, mmol/L	1.46±0.28	1.43±0.25	0.695
LDL, mmol/L	2.70±0.52	3.22±0.72	**0.005**
Hyperlipidemia, %	7% (2/28)	14% (4/28)	0.525
HbA1c,%	5.1 (5, 5.1)	5.1 (5.03, 5.3)	0.154
Glucose, mmol/L	4.93±0.32	5.13±0.69	0.175
Diabetes, %	0% (0/28)	8% (2/25)	0.208
TFA, cm^2	301.45±109.28	314.25±104.36	0.669
VFA, cm^2	87.8 (58.33, 103.48)	90.85 (72.73, 111.85)	0.521
SFA, cm^2	209.4 (133.98, 287.73)	189.6 (151.08, 269.65)	0.727
LF,%	9.30±2.81	10.29±2.60	0.194
PF,%	14.80±5.48	16.06±6.32	0.446
SMI, cm^2/m^2	47.73±8.49	52.44±8.59	0.052
PMFC,%	5.9 (4.25, 8.44)	5.7 (3.97, 7.25)	0.927

Notes: Descriptive statistics were expressed as mean ± standard deviation or median (interquartilerange). Bolded numbers indicate statistical significance.
Abbreviations: MS, metabolic syndrome; LSG, laparoscopic sleeve gastrectomy; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMI, skeletal muscle index; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

Table 4 Comparison of Change Rates in Clinical Data and Body Composition Between MS and Non-MS Obese Patients After LSG

Variables	Non-MS (n=28)	MS (n=24)	p
ΔBMI	0.33±0.07	0.30±0.06	**0.036**
ΔSystolic blood pressure	0.11±0.07	0.15±0.09	0.104
ΔDiastolic blood pressure	0.12±0.09	0.17±0.10	**0.039**
ΔTC	−0.02 (−0.14, 0.11)	−0.05 (−0.15, 0.05)	0.354
ΔTG	0.40 (0.12, 0.52)	0.66 (0.49, 0.71)	**< 0.001**
ΔHDL	−0.09±0.19	−0.24±0.21	**0.012**
ΔLDL	0.13 (−0.07, 0.20)	−0.10 (−0.16, 0.07)	**0.015**
ΔHbA1c	0.06±0.06	0.23±0.14	**< 0.001**
ΔGlucose	0.02±0.13	0.20±0.19	**< 0.001**
ΔTFA	0.52±0.12	0.51±0.12	0.714
ΔVFA	0.62 (0.48, 0.70)	0.60 (0.49, 0.67)	0.927
ΔSFA	0.49±0.13	0.45±0.15	0.259
ΔLF	0.53 (0.24, 0.60)	0.55 (0.28, 0.66)	0.533
ΔPF	0.27 (0.16, 0.45)	0.31 (0.22, 0.43)	0.388
ΔSMI	0.22 (0.11, 0.31)	0.22 (0.18, 0.28)	0.912
ΔPMFC	0.25 (0.15, 0.41)	0.18 (0.11, 0.24)	**0.041**

Notes: Descriptive statistics were expressed as mean ± standard deviation or median (interquartile range). Bolded numbers indicate statistical significance. ΔBody composition = (preoperative Body composition− postoperative Body composition) / preoperative Body composition; ΔBMI = (preoperative BMI− postoperative BMI) / preoperative BMI; ΔLaboratory indicators = (preoperative Laboratory indicators− postoperative Laboratory indicators) / preoperative Laboratory indicators.
Abbreviations: LSG, laparoscopic sleeve gastrectomy; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MS; metabolic syndrome; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMI, skeletal muscle index; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

pairwise comparisons with Bonferroni correction demonstrated significant reductions in PMFC in both groups (adjusted P <0.001).

The results of the correlation analyses for BMI, body composition, and laboratory parameters are presented in Figures 3 and 4 and the Electronic Supplementary Material (Tables S3–S6). At baseline, the MS group showed positive correlations between

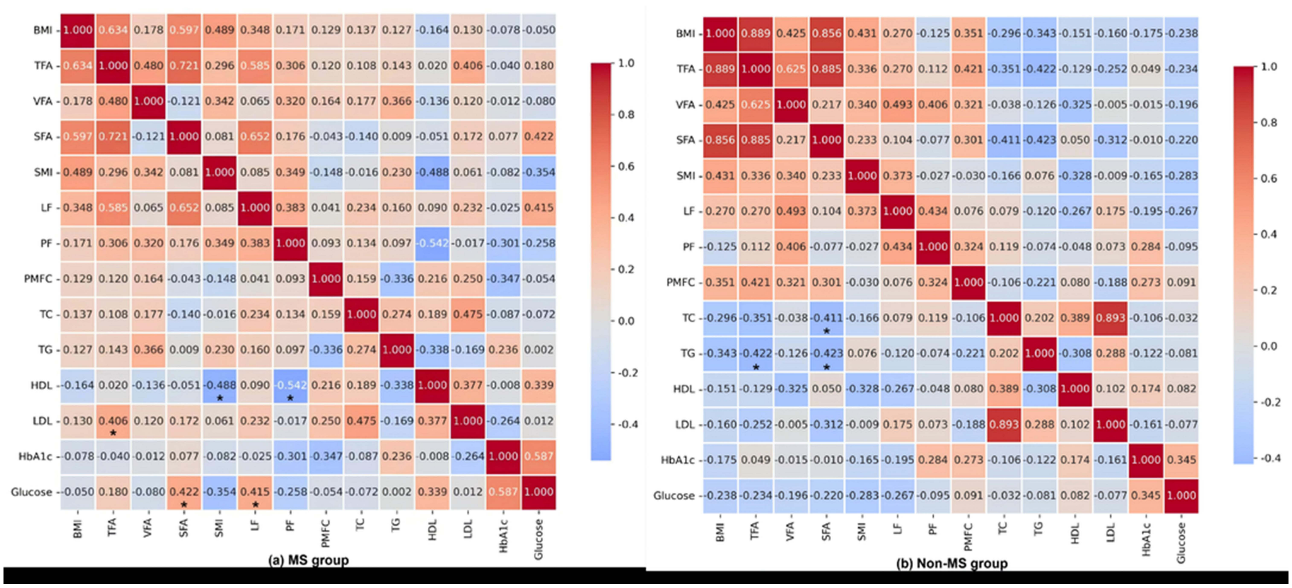

Figure 3 Heatmap showing the Spearman correlations between BMI, body composition and laboratory indicators in patients with (**a**) and without MS (**b**) at baseline. *p <0.05.

Abbreviations: BMI, body mass index; MS, metabolic syndrome; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMI, skeletal muscle index; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

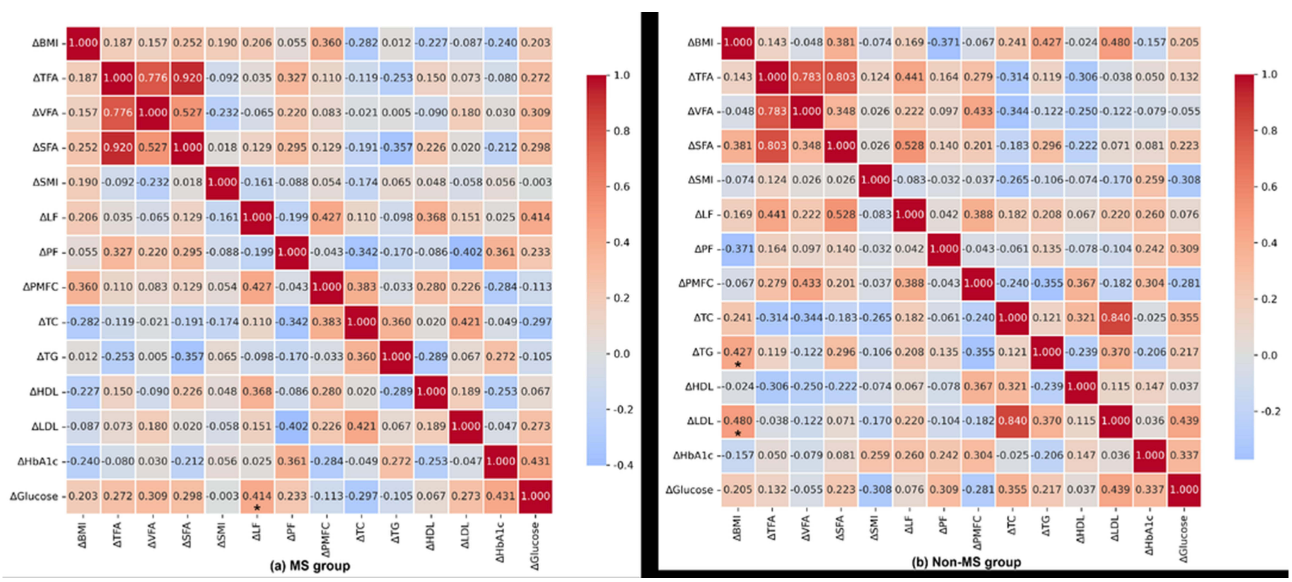

Figure 4 Heatmap showing the Spearman correlations between the rates of change in BMI, body composition and laboratory indicators in patients with (**a**) and without (**b**) MS after LSG.*p <0.05. ΔBody composition = (preoperative Body composition− postoperative Body composition) / preoperative Body composition; ΔBMI = (preoperative BMI− postoperative BMI) / preoperative BMI; ΔLaboratory indicators = (preoperative Laboratory indicators− postoperative Laboratory indicators) / preoperative Laboratory indicators.

Abbreviations: BMI, body mass index; MS, metabolic syndrome; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TFA, total fat area; VFA, visceral fat area; SFA, subcutaneous fat area; SMI, skeletal muscle index; LF, liver fat; PF, pancreas fat; PMFC, postvertebral muscle fat content.

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TFA and LDL-C (r = 0.406, P = 0.049), SFA and plasma glucose (r = 0.422, P = 0.040), and LF and plasma glucose (r = 0.415, P = 0.044), alongside negative correlations between PF and HDL-C (r = −0.542, P = 0.006) and between SMI and HDL-C (r = −0.488, P = 0.016). In the non-MS group at baseline, negative correlations were observed between TFA and TG (r = −0.422, P = 0.025), SFA and TG (r = −0.423, P = 0.025), and SFA and TC (r = −0.411, P = 0.030). Regarding postoperative changes, in the MS group, the change in LF positively correlated with the change in plasma glucose (r = 0.414, P = 0.044). In the non-MS group, the change in BMI positively correlated with changes in TG (r = 0.427, P = 0.023) and LDL-C (r = 0.480, P = 0.010).

Discussion

This study utilized QCT to evaluate changes in body composition and metabolic indicators in obese patients following LSG. Our findings provide valuable insights into the interactions between regional fat deposition and metabolic abnormalities in the context of surgical weight loss. We demonstrated that LSG effectively improves both body composition and laboratory indicators in obese patients. Notably, we observed that preoperative parameters differed between patients with and without MS, and more importantly, the magnitude of postoperative improvement was significantly influenced by metabolic status. Furthermore, the correlations between body composition and laboratory indicators, as well as the extent of their improvement after LSG, varied considerably according to metabolic status.

It is well established that LSG is one of the most effective treatments for obesity and leads to significant weight loss and metabolic improvements at multiple postoperative time points,[9,13–16] primarily because of the substantial reduction in gastric volume and changes in secretion of gastrointestinal hormones. In terms of fat loss one year after LSG, regardless of whether patients had concomitant MS, the most significant reduction was in visceral adipose tissue rather than subcutaneous adipose tissue, while reduction in LF was the most prominent among the ectopic fat compartments. This pattern is noteworthy given the antagonistic relationship between subcutaneous adipose tissue, which serves as a safe lipid depot limiting deposition of ectopic fat,[17] and visceral adipose tissue, which is more prone to lipolysis and inflammation.[18] The preferential reduction of visceral adipose tissue is particularly important, given that alleviating chronic inflammation is essential for metabolic improvement after LSG.[19] These changes have a pronounced impact on the liver, which is the central organ for metabolism of fatty acids.[20]

In this study, there were more pronounced reductions in BMI and PMFC following LSG in patients without MS. Two potential mechanisms may explain these findings. First, the sequence in which body compartments are mobilized after LSG is not uniform.[16] By one year post-surgery, the substantial contribution of abdominal and ectopic fat to metabolic dysregulation may have diminished, allowing the more subtle effects of intermuscular fat to become clinically apparent, particularly in non-MS patients whose greater baseline metabolic reserve may facilitates this later-stage fat mobilization. Second, the extent of weight loss is directly associated with the degree of improvement in multi-organ insulin sensitivity.[21,22] Specifically, an initial weight loss of 5% primarily enhances insulin sensitivity in multiple organs, while further weight reduction additionally improves sensitivity in skeletal muscle.[22] Given the close anatomical and metabolic relationship between muscle and intermuscular fat, this LSG-induced improvement in muscle insulin sensitivity may critically modulate the local metabolic impact of intermuscular fat.[23] Previous studies have indicated that intermuscular adipose tissue contributes to local muscle inflammation and impairment of insulin signaling through the secretion of pro-inflammatory cytokines and free fatty acids.[24] Therefore, the greater reduction in PMFC observed in non-MS patients may represent not merely quantitative fat loss, but also a qualitative improvement in the muscular microenvironment. This could potentially enhance muscle insulin sensitivity and help preserve long-term muscle function.

The associations between body composition and metabolic markers have been described previously.[25,26] However, there is a scarcity of literature on how these associations vary according to metabolic status and on the correlations between their improvements following LSG. In this study, we observed significant correlations of ectopic fat, abdominal fat, and skeletal muscle mass with glucose and indicators of lipid metabolism in the MS group, whereas only abdominal fat was associated with lipid metabolism in the non-MS group. In patients without MS, the association between abdominal fat and blood lipid levels is linked to the storage capacity of subcutaneous fat. Given that these patients predominantly accumulated fat in the subcutaneous compartment, this pattern may help to maintain a favorable serum lipid profile.[27,28] However, when the storage capacity of subcutaneous fat is saturated, lipids are redirected and deposited in visceral fat and ectopic tissue.[29,30] Moreover, excessive visceral fat directly releases free fatty acids and pro-inflammatory cytokines, exposing the liver to high concentrations of free fatty acids and glycerol, which may contribute to the dyslipidemia and hyperglycemia characteristic of metabolic abnormalities.[31]

Our analyses further showed a consistent association of accumulation of hepatic fat with elevated plasma glucose and that a reduction in hepatic fat after LSG was associated with improvement of plasma glucose only in patients with MS. Other studies have also reported a strong link between hepatic fat and type 2 diabetes, particularly in patients with metabolic dysfunction.[32,33] The reduction in hepatic fat after bariatric surgery likely improves insulin sensitivity in the liver,[34] thereby contributing to better glycemic control and a lower risk of diabetes. Notably, bariatric surgery leads to elevated levels of glucagon-like peptide-1, which plays an important role in hepatic glucose metabolism.[35] In addition, patients with MS often exhibit impaired β-cell function, and glucagon-like peptide-1 exerts a positive feedback effect on β-cells.[36,37] Together, these mechanisms act synergistically to improve glycemic control. However, in our patients without MS, improvements in TG and LDL-C levels were associated only with a reduction of BMI, possibly because their intact adipose tissue allowed direct correction of lipid levels by weight loss. In contrast, metabolic benefits in morbidly obese patients are mediated principally by surgery-induced hormonal and metabolic changes, in particular rapid resolution of insulin resistance, rather than by weight loss alone.[38,39] Therefore, this effect may overshadow the more linear contribution of weight loss to variations in lipid levels, attenuating the correlation observed in MS.

This study has several limitations. First, the sample size was relatively small. This was primarily because most participants were young women who experienced significant weight loss within three months after surgery, and some became pregnant, leading to loss to follow-up. Furthermore, the limited sample size may have compromised our ability to fully elucidate the complex relationships between body composition and laboratory parameters. Second, postoperative dietary intake, physical activity, and psychosocial factors were not analyzed and thus may represent unaccounted confounders affecting weight loss outcomes. Finally, the follow-up duration may have been insufficient to observe long-term changes in metabolic parameters and body composition after LSG. Future studies with larger, more diverse cohorts and longer follow-up periods are warranted to validate and extend these findings.

Conclusions

LSG significantly improves body weight, body composition, and laboratory indicators in obese patients. In this study, the most notable improvements were observed in VFA and LF, which were generally consistent across all patients. However, the reduction in PMFC was more pronounced in patients without MS. There was a correlation between reduction in LF and improved glucose metabolism in patients with MS, whereas weight loss was associated with improved lipid metabolism in those without MS.

Data Sharing Statement

The datasets used for analyses during the current study are available from the corresponding authors upon reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the First Affiliated Hospital of Wannan Medical College Ethics Committee (No. 2025-180). Given the retrospective nature of this study, the requirement for patient informed consent was waived by the Ethics Committee. All patient data were handled in strict compliance with confidentiality protocols.

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Author Contributions

Rui-Jia Yan contributed to conceptualization, formal analysis and writing-original draft. Chao Zhang contributed to investigation, writing-original draft and funding acquisition. Min-Hong Wang contributed to investigation and writing-original draft. Yun-Feng Zhou and Juan Wang contributed to conceptualization, methodology, writing—review&editing

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Disclosure

The authors declare that they have no competing interests.

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