



The Changes in Body Composition in Obese Patients with Polycystic Ovary Syndrome after Laparoscopic Sleeve Gastrectomy: a 12-Month Follow-up

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Abstract

Objectives To investigate the changes in body fat distribution and predicting factors of these changes in polycystic ovary syndrome (PCOS) patients with obesity, after laparoscopic sleeve gastrectomy (LSG).

Methods This study consecutively enrolled 153 patients with obesity aged 18–45 years (83 with PCOS and 70 control patients) who underwent LSG from May 2013 to September 2020 at the Department of Endocrinology, Shanghai Tenth People's Hospital, with a 12-month follow-up. Dual-energy X-ray absorptiometry (DEXA) was used to assess body fat distribution.

Results The percentage of fat mass loss in the visceral adipose tissue (VAT) region (55.08%) was more than that in any other body regions at 12 months post-surgery in the PCOS group yet insignificant. Homeostatic model assessment of insulin resistance (HOMA-IR) at baseline and Δ HOMA-IR were only negatively correlated with the variations in VAT mass and volume at 3 months post-surgery in the PCOS group. Logistic regression analysis showed that HOMA-IR <6.65 was an independent predictive factor for the changes in VAT mass and volume at 3 months post-surgery in the PCOS group.

Conclusions In this study, the percentage loss of fat mass was greater in the VAT region than in any other body regions in all patients. The rate of VAT decrease in the PCOS group was higher than that in the control group yet insignificant. Compared with control patients, HOMA-IR at baseline was an independent risk factor for the changes in VAT mass and volume at 3 months post-surgery in patients with PCOS.

Key Points

- The percentage loss of fat mass was greater in the VAT region than in any other body regions in all patients.
- The rate of VAT decrease in the PCOS group was higher than that in the control group yet insignificant.
- HOMA-IR at baseline was an independent risk factor for the changes of VAT mass in patients with PCOS.

Keywords Polycystic ovary syndrome · Laparoscopic sleeve gastrectomy · Body fat distribution · Visceral adipose tissue

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Introduction

Polycystic ovary syndrome (PCOS) is a reproductive endocrine disease characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphologic features [1, 2]. Premenopausal women with PCOS frequently present with a predominantly abdominal and visceral fat distribution [3–5]. Although the etiology of PCOS is unclear, visceral adipose tissue (VAT) plays an important role in the pathogenesis of PCOS. The interaction between PCOS and VAT may be bidirectional, forming a vicious circle. On the one hand, excessive androgen production could favor visceral fat deposition; on the other hand, VAT could facilitate excessive androgen

production of ovarian and/or adrenal origin through the direct effects of several autocrine, paracrine, and endocrine mediators or indirectly through the induction of insulin resistance (IR) and hyperinsulinism [6, 7].

Multiple studies have demonstrated that laparoscopic sleeve gastrectomy (LSG) is superior to nonsurgical approaches in the treatment of PCOS patients with obesity, especially those with morbid obesity [8–10]. Except for significant weight loss, LSG could improve metabolic dysfunction, reduce testosterone levels, and restore menstrual cycles and ovulation rates [11, 12]. Moreover, pregnancy and fertility rates increased in PCOS patients with morbid obesity, after LSG surgery, with few maternal and neonatal complications [13, 14]. It was reported that LSG was beneficial in the reduction of VAT mass in patients with obesity [15, 16], which may help to reduce inflammatory adipokine secretion, decrease branched-chain amino acids levels, and improve IR [17, 18]. However, limited data are available on the changes in body fat distribution (especially VAT) in PCOS patients with obesity after LSG surgery. Based on the key role of VAT in premenopausal women with PCOS, we hypothesized that changes in VAT may be most pronounced in the aforementioned group post-surgery.

Therefore, the present study aimed to investigate the effect of LSG on body fat distribution (especially VAT) in premenopausal PCOS patients with obesity over a period of 12 months and explore the predictive factors for their body compositions after LSG. This study will help us to further understand the role of VAT in the pathogenesis of PCOS and to predict the effectiveness of bariatric surgery in terms of VAT loss in PCOS patients with obesity.

Methods

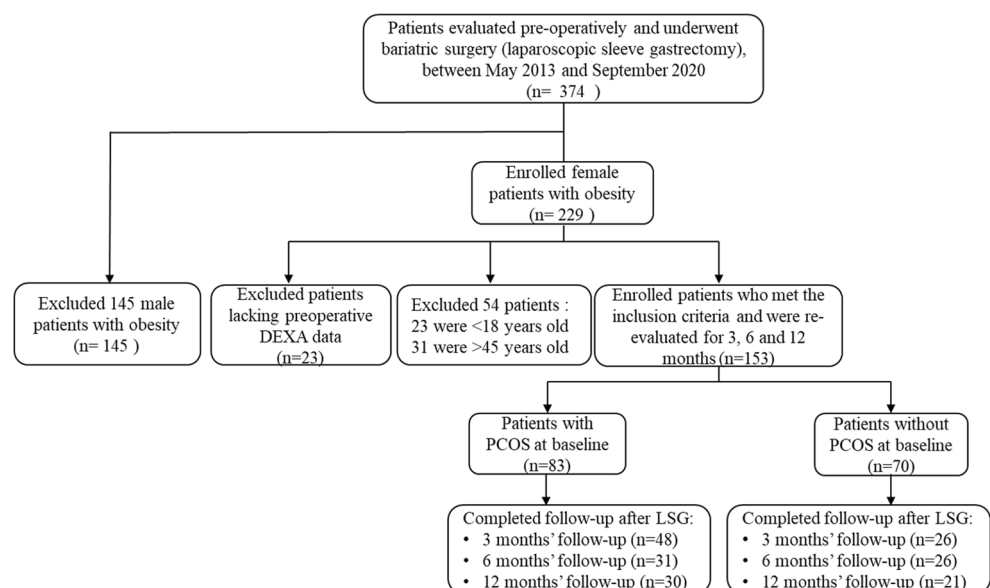
Study Design and Patients

This study consecutively enrolled 153 patients with obesity aged 18–45 years (83 with PCOS and 70 control patients) (Fig. 1). All patients underwent sleeve gastrectomy at the Shanghai Tenth People's Hospital. Diagnosis of PCOS was based on the Rotterdam diagnosis criteria (2003) [19]. Exclusion criteria were as follows: (1) age <18 years or >45 years old; (2) secondary obesity due to endocrine disorders; (3) severe hepatic, renal dysfunction, and/or heart failure; and (4) mental illnesses that caused inability to provide informed consent. The control group comprised patients with regular menstrual cycles (24–35 days) without any clinical and/or biochemical signs of hyperandrogenism. Irregular menstrual cycles were defined as oligo-amenorrhea or cycles lasting 35 days or longer, as reported by patients. All enrolled patients underwent LSG using a standardized technique. All procedures performed in studies involving human participants were in accordance with the ethical standards of the ethics committee of Shanghai Tenth People's Hospital and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Anthropometric Assessment and Laboratory Analysis

All patients were hospitalized for a comprehensive examination before surgery, and followed-up in our department by bariatric surgeons and nurses at 3, 6, and 12 months post-surgery. The clinical data were retrospectively collected from all patients by trained endocrinology specialists, including

Fig. 1 Flow diagram of female patients with obesity who completed a 12-month follow-up after bariatric surgery



height, body weight, BMI, waist circumference (WC), hip circumference (HC), Ferriman-Gallwey score, and menstrual cycle. Signs of polycystic ovaries were measured using an ultrasound scan of pelvis. The BMI and waist-to-hip ratio (WHR) were calculated as follows: BMI= body weight/(height × height) (kg/m²) and WHR=WC/HC. Blood samples were obtained from all patients in the morning after at least 10 h of overnight fasting. Fasting plasma glucose (FBG), fasting serum insulin (FINS), glycosylated hemoglobin (HbA1c), creatinine (Cr), uric acid (UA), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), and sex hormone binding globulin (SHBG) levels were measured. Postprandial plasma glucose (PBG) and postprandial insulin (PINS) were measured using the standard meal load test, followed by an examination at 120 min [20]. Luteinizing hormone (LH), follicle-stimulating hormone (FSH), and total testosterone (TT) levels were measured using an immunochemiluminometric assay (Roche Diagnostics GmbH, Cot., Sandhofer, Mannheim, Germany). Calculated free testosterone (cFT) concentration were calculated using the formula $24.00314 \times [TT] / \log [SHBG] 0.04599 \times [TT]^2$, and this formula has been widely adopted in the literature [21]. Homeostatic model assessment of IR (HOMA-IR) was calculated using the formula $[FBG (mmol/L) \times FINS (mU/L)] / 22.5$ [22]. Dual-energy X-ray absorptiometry (DEXA, APEX 4.5.0.2, Hologic, USA) was used to assess body fat distribution in all patients at baseline, 3, 6, and 12 months after LSG. The fat mass was evaluated in six different regions, including the arms, legs, trunk, head, android, and gynoid regions, subtotal fat means the sum of arm fat mass, leg fat mass, and trunk fat mass, and the android/gynoid ratio was calculated as android fat mass/gynoid fat mass.

Statistical Analysis

All statistical analyses were performed using SPSS version 25.0. All normal distribution continuous data were presented as means ± standard deviation (SD) and integrated with delta percentage ($\Delta\%$). Non-normal distribution continuous data were presented as median and interquartile range (25%, 75%). Categorical variables were presented as percentages. Continuous data were compared between the two groups using independent sample t test, while paired sample t test was used to compare the preoperative and postoperative levels of relevant indicators. The chi-squared test was used to compare categorical data between groups. Linear and logistic regression analyses were performed to explore the association between the HOMA-IR levels at baseline and changes in fat mass at different time points post-surgery. A value of $P < 0.05$ was considered statistically significant.

Results

Comparison of Clinical Characteristics and Body Fat Distribution in the PCOS and Control Groups at Baseline

The study participants in the PCOS and control groups were young (29.0 ± 4.9 and 30.0 ± 6.9 years, respectively) and obese (BMI 38.1 ± 4.6 and 36.9 ± 4.1 kg/m², respectively) at baseline. The detailed baseline information for both groups is presented at Table 1. The mean Ferriman-Gallwey score, TT, cFT, and frequency of irregular menstrual cycles were higher in the PCOS group than in the control group. The total fat, arm fat, leg fat, subtotal fat, and gynoid fat in the PCOS group were not significantly different from those in the control group at baseline, whereas the trunk fat, android fat, and visceral fat were higher in the PCOS group than in the controls.

Follow-up of All Patients at 3, 6, and 12 Months After LSG

The follow-up rates at 3, 6 and 12 months after LSG in the PCOS group vs. control group were 57.83% vs. 37.1%, 37.35% vs. 37.14%, and 36.14% vs. 30.00%, respectively (Fig. 1). Early postoperative complications were rare in all patients after LSG, including gastric fistula, anastomotic bleeding, and gastric sleeve stenosis. The most common long-term complications at 12 months after LSG were alopecia (82%), anemia (47%), and gastroesophageal reflux (5%).

Twelve months after LSG, HOMA-IR value, TT level, and Ferriman-Gallwey score in patients with PCOS significantly decreased, and their menstrual cycles improved (Supplementary Table 1). WC, HC, and WHR at 3, 6, and 12 months post-surgery significantly decreased in all patients. However, there was no significant difference in body weight or BMI change between the PCOS and control groups. The total fat, trunk fat, arm fat, leg fat, subtotal fat, android fat, gynoid fat, android/gynoid ratio, and VAT mass, volume, and area significantly decreased at 3, 6, and 12 months post-surgery in all patients (Table 2). Although the fat mass in each region of the body dramatically decreased at 12 months post-surgery in all patients, the percentage loss of fat mass was greater in the VAT region than in any other body region, with a percentage reduction of 55.08% and 49.06% in the PCOS and control groups, respectively. However, this was not statistically significant.

Correlations Between Clinical Indicators and Body Fat Distribution in Patients with PCOS

After adjusting for possible confounders (age, BMI, TG, UA, and HbA1c), linear regression analysis showed loss of VAT after LSG was associated with the changes in fasting glucose

Table 1 Clinical characteristics in obese women with PCOS and without PCOS at baseline

Items	PCOS group (n= 83)	Control group (n= 70)	P value
Age (year)	29.0±4.9	30.8±6.9	0.080
Weight (kg)	102.2±14.5	101.2±14.2	0.691
BMI (kg/m ²)	38.1±4.6	36.9±4.1	0.100
WC (cm)	115.5±11.0	111.3±9.8	0.016*
HC (cm)	120.2±10.2	119.2±9.0	0.525
WHR	0.96±0.1	0.93±0.1	0.005*
HbA1c (%)	6.1±0.9	6.3±1.3	0.366
HOMA-IR	6.8 (4.9-9.8)	5.6 (4.0-11.0)	0.684
FBG (mmol/L)	6.3±2.3	6.0±2.0	0.494
PBG (mmol/L)	10.0±4.3	9.4±4.4	0.407
FINS (mU/L)	30.3±15.9	25.4±11.7	0.135
PINS (mU/L)	180.3±137.2	126.7±97.1	0.014*
Cr (umol/L)	56.9±14.2	56.9±9.4	0.844
UA (umol/L)	408.0±81.2	360.9±83.2	0.003*
TC (mmol/L)	4.5±0.8	4.6±0.8	0.926
TG (mmol/L)	1.5 (1.2-2.0)	1.3 (1.1-1.7)	0.008*
HDL-c (mmol/L)	1.0±0.2	1.1±0.3	0.010*
LDL-c (mmol/L)	2.8±0.7	2.8±0.7	0.675
LH (IU/L)	7.4±3.8	6.5±5.2	0.216
FSH (IU/L)	4.7±1.8	5.7±5.0	0.112
TT (nmol/L)	1.6±0.8	1.1±0.6	<0.001*
cFT ((nmol/L)	9.3±12.3	3.3±5.2	0.009*
SHBG (nmol/L)	19.7±17.3	25.2±14.7	0.368
Number of menstruations per year	5.1±3.3	11.1±0.8	<0.001*
#Irregular menstruation prevalence n (%)	80(96.3%)	0(0%)	<0.001*
Ferriman-Gallway score	7.1±3.8	5.0±4.0	0.036*

Abbreviation: *WC*, waist circumference; *HC*, hip circumference; *WHR*, waist to hip ratio; *BMI*, body mass index; *HbA1c*, glycosylated hemoglobin A1c; *HOMA-IR*, homeostatic model assessment insulin resistance index; *FBG*, fasting blood-glucose; *PBG*, postprandial blood-glucose; *FINS*, fasting serum insulin; *PINS*, postprandial insulin; *Cr*, serum creatinine; *UA*, uric acid; *TC*, total cholesterol; *TG*, triglycerides; *HDL-c*, high-density lipoprotein cholesterol; *LDL-c*, low-density lipoprotein cholesterol; *LH*, luteinizing hormone; *FSH*, follicle-stimulating hormone; *TT*, total testosterone; *SHBG*, sex hormone-binding globulin; *cFT*, calculated free testosterone;# These patients had menstrual cycles >35 day or were taking oral contraceptives for management of Irregular menstruation, *Means $P < 0.05$

and HOMA-IR levels at 3 months post-surgery in the PCOS group and with the changes in TC levels at 12 months post-surgery in the control group (Table 3).

Moreover, both baseline HOMA-IR and the changes in HOMA-IR values negatively correlated with the changes in VAT mass and volume at 3 months post-surgery in patients with PCOS (Table 4). Further logistic regression analysis showed that baseline HOMA-IR <6.65 was an independent protective factor for the changes in VAT mass [OR=0.997 (0.994–1.000) $P=0.030$] and volume at 3 months [OR=0.995 (0.990–0.999) $P=0.013$] post-surgery in patients with PCOS. However, there was no significant correlation between baseline HOMA-IR, changes in HOMA-IR values and the changes in VAT mass, volume, and area post-surgery in the control group.

There was no significant association between TT, cFT and the trunk fat, android fat, gynoid fat, and android/gynoid ratio both at baseline and at 3, 6, and 12 months post-surgery in all patients. In addition, other clinical indicators (age, BMI, HbA1c, LH, FSH, SHBG, TG) showed no association with body fat distribution at baseline or 3, 6, and 12 months post-surgery in the PCOS group (data not shown).

Discussion

Many observational studies have reported that premenopausal women with PCOS frequently present with a predominantly abdominal and visceral fat distribution (5,6,7). LSG has a positive effect on the clinical, biochemical, hormonal

Table 2 Anthropometric measurements and body fat distribution at baseline and 3, 6, and 12 months after LSG in patients with obesity

Items	PCOS group				Control group				P value	
	Baseline (n= 83)	3 months after LSG (n= 48)	6 months after LSG (n= 31)	12 months after LSG (n= 30)	P Value	Baseline (n=70)	3 months after LSG (n=26)	6 months after LSG (n=26)		12 months after LSG (n=21)
Weight(kg)	102.2 ±14.5	81.6±12.1 ^a	76.1±15.8 ^b	68.9±10.3 ^c	<0.001*	101.2 ±14.2	80.1±10.6 ^a	71.9 ±10.2 ^b	70.2±9.7 ^c	<0.001*
Δ Weight(kg)	-	20.6±4.0	26.9±9.1	32.5±8.1	<0.001*	-	20.8±5.5	27.0±7.2	32.1±7.9	<0.001*
BMI (kg/m ²)	38.1±4.6	30.2±3.8 ^a	28.3±4.9 ^b	25.5±3.6 ^c	<0.001*	36.9±4.1	29.4±3.2 ^a	26.2±3.3 ^b	25.6±2.5 ^c	<0.001*
ΔBMI (kg/m ²)	-	7.8±1.5	10.1±3.2	12.2±2.7	<0.001*	-	8.0±2.1	10.3±3.2	11.2±3.1	<0.001*
WC (cm)	115.1 ±10.5	98.5±10.2 ^a	92.8±9.5 ^b	84.5±16.5 ^c	<0.001*	111.3±9.8	97.7±10.9 ^a	87.1±8.5 ^b	83.3±8.9 ^c	<0.001*
HC (cm)	119.8±9.7	107.4 ±10.5 ^a	102.5 ±12.1 ^b	96.9±8.6 ^c	<0.001*	119.2±9.0	108.4 ±10.0 ^a	100.1 ±7.0 ^b	98.8±6.8 ^c	<0.001*
WHR	0.96±0.1	0.91±0.1 ^a	0.91±0.1 ^b	0.86±0.1 ^c	0.043*	0.93±0.1	0.90±0.1 ^a	0.87±0.1 ^b	0.8±0.1 ^c	0.036*
Total body % Fat	47.2±3.4	44.0±4.6 ^a	41.1±4.1 ^b	37.5±4.8 ^c	<0.001*	46.3±3.7	43.4±4.7 ^a	38.8±4.5 ^b	37.8±4.2 ^c	<0.001*
Total fat mass (kg)	47.2±8.7	35.6±9.7 ^a	32.7±8.6 ^b	26.5±7.0 ^c	<0.001*	45.5±8.2	35.8±7.0 ^a	28.5±6.6 ^b	27.6±6.5 ^c	<0.001*
Trunk fat mass (kg)	25.0±4.8	19.1±4.8 ^a	16.6±4.9 ^b	13.6±4.0 ^c	<0.001*	23.4±4.0	18.3±4.3 ^a	14.5±3.7 ^b	14.0±3.8 ^c	<0.001*
Arm fat mass (kg)	6.7±1.5	5.7±2.5 ^a	4.5±1.3 ^b	3.6±0.9 ^c	<0.001*	6.6±1.7	5.2±2.4 ^a	4.0±1.2 ^b	3.9±1.4 ^c	<0.001*
Leg fat mass (kg)	14.1±3.9	12.0±6.3 ^a	10.2±3.1 ^b	7.8±2.2 ^c	<0.001*	14.0±3.9	11.0±2.9 ^a	8.7±2.4 ^b	8.6±2.4 ^c	<0.001*
Subtotal fat mass (kg)	45.7±8.6	35.5±8.4 ^a	31.4±8.5 ^b	25.3±6.9 ^c	<0.001*	44.3±8.2	33.7±6.9 ^a	27.2±6.5 ^b	26.4±6.5 ^c	<0.001*
Android fat mass (kg)	4.5±1.2	3.1±0.9 ^a	2.6±0.9 ^b	2.1±0.8 ^c	<0.001*	4.1±1.0	3.1±0.9 ^a	2.2±0.7 ^b	2.2±0.7 ^c	<0.001*
Gynoid fat mass (kg)	6.7±1.6	5.1±1.5 ^a	4.7±1.2 ^b	3.8±0.9 ^c	<0.001*	6.7±1.6	5.1±1.2 ^a	4.1±0.9 ^b	4.1±1.0 ^c	<0.001*
Android / Gynoid Ratio	1.2±0.1	1.2±0.1 ^a	1.1±0.1 ^b	1.1±0.1 ^c	0.004*	1.2±0.1	1.1±0.1	1.1±0.2	1.0±0.1 ^c	0.134
VAT mass (kg)	1.2±0.3	0.8±0.3 ^a	0.7±0.2 ^b	0.5±0.2 ^c	<0.001*	1.1±0.2	0.8±0.3 ^a	0.6±0.2 ^b	0.5±0.2 ^c	<0.001*
VAT Volume (cm ³)	1.3±0.3	0.9±0.3 ^a	0.7±0.2 ^b	0.6±0.2 ^c	<0.001*	1.1±0.3	0.9±0.3 ^a	0.6±0.2 ^b	0.6±0.3 ^c	<0.001*
VAT Area (cm ²)	0.24±0.06	0.19±0.15 ^a	0.14±0.04 ^b	0.11±0.03 ^c	<0.001*	0.22±0.05	0.16±0.05 ^a	0.12 ±0.04 ^b	0.11 ±0.05 ^c	<0.001*

Abbreviation: WC, waist circumference; HC, hip circumference; BMI, body mass index; WHR, waist to hip ratio; VAT, visceral adipose tissue; LSG, laparoscopic sleeve gastrectomy; ^a Means 3 months after surgery compared with baseline, *P*<0.05; ^b Means 6 months after surgery compared with baseline, *P*<0.05; ^c Means 12 months after surgery compared with baseline, *P*<0.05; ^Δ Means the changes in the different regions in obese PCOS patients before and after LSG. *Means *P*<0.05

Table 3 Correlations between the variation of VAT mass and clinical metabolic indices at 3, 6, and 12 months after LSG in patients with obesity, after adjusting for possible confounders (age, BMI, TG, UA, and HbA1c)

Items	Δ VAT mass								
	3 months after LSG			6 months after LSG			12 months after LSG		
	β	R^2	P value	β	R^2	P value	β	R^2	P value
PCOS group									
Δ HbA1c (%)	0.947	-0.033	0.107	-0.010	-0.239	0.970	-0.411	-0.045	0.667
Δ HOMA-IR	0.509	0.083	0.011*	0.255	0.174	0.271	0.246	-0.011	0.313
Δ FBG (mmol/L)	0.481	0.053	0.018*	0.447	0.288	0.051	0.147	-0.071	0.662
Δ PBG (mmol/L)	0.368	-0.024	0.094	0.476	0.376	0.098	0.253	-0.018	0.436
Δ UA (μ mol/L)	0.072	-0.128	0.757	0.105	0.112	0.664	0.597	0.127	0.076
Δ TC (mmol/L)	0.150	-0.096	0.429	-0.305	0.177	0.262	0.181	0.251	0.482
Δ TG (mmol/L)	0.855	-0.092	0.389	-0.859	0.142	0.465	1.077	-0.087	0.656
Δ HDL-c (mmol/L)	-0.107	-0.109	0.576	-0.078	0.121	0.727	0.032	-0.084	0.896
Δ LDL-c (mmol/L)	0.280	-0.039	0.147	-0.357	0.194	0.202	0.291	-0.006	0.296
Control group									
Δ HbA1c (%)	0.742	0.020	0.465	0.671	0.547	0.365	0.131	0.654	0.763
Δ HOMA-IR	0.095	0.054	0.370	-0.170	0.538	0.541	-0.426	-0.317	0.602
Δ FBG (mmol/L)	0.127	0.017	0.677	0.192	0.532	0.481	0.990	0.228	0.061
Δ PBG (mmol/L)	-0.456	0.123	0.204	0.055	0.509	0.825	0.202	0.390	0.643
Δ UA (μ mol/L)	0.278	-0.172	0.378	0.343	0.534	0.397	-0.432	-0.241	0.553
Δ TC (mmol/L)	-0.010	-0.204	0.713	-0.112	0.520	0.612	-0.809	0.365	0.044*
Δ TG (mmol/L)	0.816	-0.043	0.164	0.953	0.599	0.159	0.673	-0.287	0.701
Δ HDL-c (mmol/L)	0.082	-0.209	0.773	-0.264	0.537	0.436	-0.466	-0.006	0.219
Δ LDL-c (mmol/L)	-0.235	-0.172	0.490	-0.088	0.540	0.719	-0.438	-0.120	0.338

Abbreviation: VAT, visceral adipose tissue; HOMA-IR, homeostatic model assessment insulin resistance index; HOMA-IR, homeostatic model assessment insulin resistance index; FBG, fasting blood-glucose; PBG, postprandial blood-glucose; UA, uric acid; TC, total cholesterol; TG, triglycerides; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; LSG, laparoscopic sleeve gastrectomy; Δ Means the changes in the different regions in obese PCOS patients before and after LSG. *Means $P < 0.05$

indicators (12), and the fertility and pregnancy outcomes of patients with PCOS (13,14). However, research on the changes in body fat distribution in PCOS patients after LSG is still limited. In this study, we provided the first evidence about changes in body fat distribution in PCOS patients with obesity after LSG and explored the predictive factors for the body composition in the same population.

In our study, although the mean age and BMI were comparable in the PCOS and control groups, the mean WC and WHR of the PCOS group were higher than those of the control group, which was consistent with previous studies [23, 24]. Moreover, high levels of PINS, TG, and UA, and lower levels of HDL-C were observed in the PCOS group, which was in line with previous studies, showing that patients with PCOS have worse metabolic status than controls [25, 26]. Twelve months after LSG, the mean BMI, HOMA-IR level, TT level, and Ferriman-Gallwey score in patients with PCOS significantly decreased, and their menstrual cycles improved. The findings were supported by other studies, which have

demonstrated that bariatric surgery could be an effective therapy for PCOS patients with obesity and could improve their clinical symptoms and metabolic dysfunctions [12, 27].

Our results showed that trunk fat, android fat, and VAT levels in PCOS patients were significantly higher than those in control patients. This finding is generally in line with some studies showing that patients with PCOS are characterized by android fat patterning [28]) and have higher VAT level than controls [29, 30]. The accumulation of abdominal adiposity in these patients may be attributed to impaired lipolysis, diminished fat oxidation, and metabolic inflexibility [31, 32]. Considering the efficiency of bariatric surgery in the treatment of PCOS patients, it is extremely important to clarify the changes in body fat distribution post-surgery.

In this study, the loss of body fat mass was higher in VAT region in both groups post-surgery, with more loss in the PCOS group than in the control group (55.08% versus 49.06%). However, this difference was not statistically significant, which might be attributed to the small sample size and a

Table 4 Correlations between the variation of the HOMA-IR and body fat distribution at 3, 6, and 12 months after LSG in patients with obesity, after adjusting for possible confounders (age, BMI, TG, UA, and HbA1c)

Items	Δ HOMA-IR								
	3 months after LSG			6 months after LSG			12 months after LSG		
	β	R^2	P value	β	R^2	P value	β	R^2	P value
PCOS group									
Δ Trunk fat mass (kg)	0.027	0.101	0.869	-0.027	0.092	0.895	0.165	-0.178	0.530
Δ Android fat mass (kg)	0.129	0.120	0.431	-0.098	0.103	0.628	0.024	-0.210	0.931
Δ Gynoid fat mass (kg)	-0.124	0.118	0.457	-0.243	0.166	0.220	-0.164	-0.176	0.518
Δ Android/gynoid ratio	0.108	0.114	0.507	0.036	0.092	0.867	0.040	-0.209	0.886
Δ VAT mass (kg)	0.398	0.283	0.011*	0.262	0.152	0.271	0.275	-0.129	0.313
Δ VAT Volume (cm ³)	0.391	0.275	0.013*	0.175	0.119	0.458	0.281	-0.126	0.304
Δ VAT Area (cm ²)	0.231	0.163	0.152	0.151	0.112	0.518	0.314	-0.129	0.314
Control group									
Δ Trunk fat mass (kg)	-0.125	-0.003	0.628	0.618	0.569	0.008*	-0.261	0.745	0.258
Δ Android fat mass (kg)	-0.379	0.123	0.182	-0.119	0.175	0.627	-0.287	0.789	0.112
Δ Gynoid fat mass (kg)	-0.004	-0.024	0.989	-0.243	0.223	0.354	-0.169	0.727	0.362
Δ Android/gynoid ratio	-0.497	-0.167	0.123	0.501	0.444	0.036*	-0.085	0.697	0.687
Δ VAT mass (kg)	-0.261	0.045	0.370	-0.252	0.313	0.541	-0.096	0.702	0.602
Δ VAT volume (cm ³)	-0.260	0.044	0.372	-0.252	0.313	0.542	-0.073	0.697	0.696
Δ VAT area (cm ²)	-0.258	0.043	0.377	-0.218	0.313	0.544	-0.095	0.702	0.606

Abbreviation: VAT, visceral adipose tissue; HOMA-IR, homeostatic model assessment insulin resistance index; LSG, laparoscopic sleeve gastrectomy; Δ Means the changes in the different regions in obese PCOS patients before and after LSG. *Means $P < 0.05$

certain rate of loss to follow-up. Large sample studies are required to further clarify this issue. Moreover, loss of VAT post-surgery was associated with the changes in fasting glucose and HOMA-IR levels at 3 months post-surgery in the PCOS group and with the changes in TC at 12 months post-surgery in the control group. This finding was generally consistent with those from previous studies reporting that decreased VAT may contribute to the improvement of IR and lipid metabolism [18, 33].

Further analyses were performed to explore the factors that may predict changes in body fat distribution in PCOS patients after LSG. After adjusting for other confounding factors, changes in HOMA-IR values negatively correlated with changes in VAT mass and volume at 3 months post-surgery in patients with PCOS, whereas this correlation was not significant for the control group. The results suggested that VAT was more closely related to IR in PCOS patients, and were generally in line with a 2-year follow-up study, showing that insulin sensitivity was improved due to continued loss of VAT in PCOS patients [34]. They significantly correlated with the change in android/gynoid ratio at 3 months post-surgery in the control group. In addition, HOMA-IR at baseline was an independent risk factor for the changes in VAT mass and volume at 3 months post-surgery in the PCOS group, but not in the control group. However, the results on 3 months are not

shown at 6 and 12 months in the PCOS group, which might be explained by the small sample size and a certain rate of loss to follow-up. A large multicenter study is needed to verify this important outcome.

In our study, there was no association between androgen (TT level and cFT level) and body fat distribution in PCOS patients both at baseline and after LSG. This finding was in line with those of some studies [35, 36], but in contrast to other studies [37, 38]. Tosi et al. have reported that the levels of total fat and central fat in PCOS patients could independently predict IR, but not hyperandrogenemia (35). Taken together with the positive association between HOMA-IR and body fat distribution, our results suggest that IR, rather than androgen, may play a major role in body fat distribution in PCOS populations with obesity [39].

The main limitation of this study is the relatively short follow-up time and a certain rate of loss to follow-up, which could lead to insufficient information on the factors influencing changes in body fat distribution after LSG. More longitudinal studies are required to confirm these findings. The strength of this study is that it provides the first evidence that changes in body fat distribution PCOS patients with obesity after LSG may contribute to the knowledge of the determinants of body fat distribution.

Conclusion

In this study, the percentage loss of fat mass was greater in the VAT region than in any other body regions in all patients. Although there was no statistically significant correlation between the PCOS group and control group post-surgery, the rate of VAT decrease in the PCOS group was higher than that in the control group. Compared with control patients, HOMA-IR at baseline was an independent risk factor for the changes in VAT mass and volume at 3 months post-surgery in patients with PCOS.

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Declarations

Competing Interests The authors declare that they have no conflict of interest.

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