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ORIGINAL CONTRIBUTIONS

Evaluation of Persistent Efficacy of Diabetes Remission and Decline of Cardiovascular Risk After Laparoscopic Sleeve Gastrectomy: a Preliminary 1-Year Study

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Abstract

Background Laparoscopic sleeve gastrectomy (LSG) is a relative safe procedure in bariatric surgery. However, relatively few studies had been assessed its comprehensive efficacies. In the current study, the efficacies of LSG were comprehensively explored on glycemic control and cardiovascular disease (CVD) risk reduction.

Methods A total of 95 obese patients, who owned body mass index (BMI) of more than 35, were recruited. All of them primarily underwent LSG from 2014 to 2016. Type 2 diabetes mellitus (T2DM) remission was defined as levels of glycated hemoglobin (A1C) and fasting blood glucose (FBG) of less than 6.4% and 125 mg/dL, respectively. The further efficacies of LSG on CVD and coronary heart disease (CHD) risks were explored by using original- and recalibrated Framingham 10-year CHD risk scores and the other 3 well-established CVD risk prediction models.

Results Systolic blood pressure (SBP), serum FBG, A1C, triglyceride (TG), BMI, and body weight showed significantly declined and high-density lipoprotein-cholesterol (HDL) displayed twice higher than beginning level after LSG. The 71 of 95 patients with obesity were T2DM; 62 of them exhibited persistent DM remission until 1 year after LSG. Cardiovascular age, general cardiovascular risk (GCVR), and atherosclerotic cardiovascular disease risk (ASCVD) also showed significant decrements after LSG. We also observed significant reductions in estimated CVD and CHD risks.

Conclusion LSG resulted in a persistent T2DM remission and corrected metabolic abnormalities. Subsequently, LSG also benefits declined risks of CVD and 10-year CHD developments. LSG may be helpful for primary CVD care in obese patients with BMI of more than 35.

Keywords Laparoscopic sleeve gastrectomy · Obesity · Type 2 diabetes mellitus · Diabetic remission · Cardiovascular disease

Introduction

Obesity, diabetes mellitus, and metabolic abnormalities (ex. hypertension, hyperlipidemia, and hyperglycemia) have become pandemic worldwide [1, 2]. It had been reported that CHD, dyslipidemia, micro- and macro-vascular diseases were considered as the major comorbidities of T2DM [3–5]. In addition, risk developments of CVD and CHD are highly associated with obese degree, T2DM remission, insulin amelioration, and metabolic abnormality correction [4, 6]. Thus, an effective treatment for obesity, T2DM, and metabolic abnormality is urgently required for CVD risk reduction in Asian population. Bariatric surgery plays a major role in long-lasting T2DM remission and weight reduction. Moreover, laparoscopic Roux-en-Y gastric bypass (LRYGB) exerts a satisfactory effect on obesity reduction

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and T2DM remission that can be attributable to the incretin effect explained by the foregut or hindgut hypothesis [7, 8]. Although LRYGB is effective for weight loss and T2DM remission compared with other well-established surgical procedures (sleeve gastrectomy and laparoscopic adjustable gastric banding [LAGB]), a large amount of studies have reported inconsistent findings regarding the long-term effects of LRYGB on weight regain, T2DM remission, and comorbidity re-emergence [7, 9]. Considering the balance between long-term comorbidities and effective weight loss and T2DM remission, many novel procedures involving sleeve gastrectomy along with some intestinal bypass or alteration in the small intestine have been developed. Laparoscopic sleeve gastrectomy (LSG) was a newly and frequently used to obesity and poor glycemic control therapies in Asia–Pacific region because of its relatively lower complications and favorable efficacies [9]. Several studies have reported that compared with LRYGB, LSG is a relatively safe and acceptable effectiveness on obesity therapy and DM remission [8, 10–12].

Some literatures had been illustrated that risk development of CVD is potentially associated with obesity, T2DM, and metabolic abnormalities [2, 13, 14]. Although LSG has been reported to exert significantly early effects on T2DM remission and weight loss in obese patients with T2DM [15–19], relative few studies had explored the CVD and CHD risk changes after LSG, by using various well-established predictive models. The multi-variable Framingham's risk prediction model has been widely applied to estimate the 10-year risk of coronary heart disease (CHD) [20, 21]. However, these studies have recruited participants of European descent; thus, their results cannot be appropriately generalized to other populations without re-evaluating other ethnicities. Thus, this pilot study was proposed to explore the efficacies after LSG on weight loss and T2DM remission; furthermore it aimed to indirectly assess the results of CVD and CHD risks by using the original and recalibrated Framingham functions [22]. Additionally, the developed predictive models were also used to evaluate changes of heart and vascular physiological ages and ASCVD risk after LSG.

Material and Methods

Study Patients

This pilot study recruited 95 obese patients with BMI of more than 35 (44 men and 51 women) aged with 20 to 60 years. All of them had undergone LSG from 2014 to 2016 in the same medical institute (TMUH: Taipei Medical University Hospital, Taipei, Taiwan). The anthropometric and biochemical data of the patients were retrieved pre-operatively and 1 year after LSG. The inclusion criterion was undergoing the primary LSG procedure. The exclusion criteria were who having cancer, mental disorder, and end-organ damage. Data were retrieved at three time points: before LSG and the 6th and 12th month after LSG. Informed consent was obtained from all individual participants included in the study. This study was approved by the Institutional Ethical Review Committee of the Taipei Medical University (TMU), Taipei, Taiwan (TMU-JIRB 201203002).

Definition of T2DM Remission After LSG

Complete T2DM remission was defined as having a glycated hemoglobin (A1C) level of < 5.7% or a fasting blood glucose (FBG) level of < 100 mg/dL. Partial T2DM remission was defined as having an A1C level with 5.7–6.4% or an FBG level of 100–125 mg/dL without receiving any glycemia-lowering medication and insulin injection [23]. In this study, the definition of T2DM remission included complete and partial T2DM remission.

Anthropometric and Laboratory Data

Body weight, fat mass, muscle mass, and basal metabolic rate (BMR) were measured using segmental multi-frequency bioelectrical impedance analysis (InBody770, USA). In addition, waist circumference, BMI (body mass index), and excess weight loss percentage (EWL%) were measured. Fasting blood samples were drawn under NPO (nothing by mouth) for more than 8 h. The levels of fasting blood glucose (FBG), A1C (Roche Cobas e602 analyzer), aspartate aminotransferase, alanine aminotransferase, total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), serum urea nitrogen, and serum creatinine were analyzed. The serum hemoglobin concentration was analyzed using a Beckman Coulter DxH1601 machine.

Surgical Procedure

LSG was performed using a three-port approach. One 12-mm trocar was inserted through the umbilicus, and two 5-mm trocars were inserted into the bilateral upper abdomen. Vertical transection of the stomach, starting from 5 cm proximal to the pylorus on the greater curvature and proximally extending to the angle of His, was performed using a linear stapler under the guidance of a 36-Fr orogastric tube placed along the lesser curvature. Subsequently, the staple line was reinforced with a 3–0 Vicryl suture to prevent leakage and hemorrhage.

Risk Prediction Models for the Primary Care of CVD

Currently, well established risk predictive models used in the primary care of CVD include multivariable Framingham

functions for predicting the absolute 10-year CHD risk [24–27], recalibrated Framingham functions for predicting the 10-year CHD risk in the Chinese population [22], the atherosclerotic CVD (ASCVD) multiple model for predicting the 10-year risk of heart disease or stroke [28], the sex-specific multivariable model for predicting general CVD risk [29], and the heart or vascular age prediction model derived from the Framingham study [29].

Statistical Analysis

Data are expressed as the mean ± standard deviation. Categorical variables are indicated as the frequency or percentage and were analyzed using the chi-square test. Continuous variables were analyzed using analysis of covariance (ANCOVA) after adjustment for sex and age. The paired-*t* test and McNemar's chi-square test were performed to compare paired variables and the status of T2DM remission between the 6th- and 12th-month after LSG, respectively. Non-parametric analysis was performed to examine the relationship between two groups for non-normally distributed data. Trend analysis was performed to examine cardiovascular age, ASCVD, CVD, and CHD risks within 1 year after LSG by using the repeated analysis of variance analysis. A p-value of < 0.05 was considered statistically significant. All statistical analyses were performed using SAS (version 9.4).

Results

A total of 95 obese patients (44 men and 51 women) aged between 18 and 60 years were recruited. The mean ages of the male and female patients were 40.5 and 42.9 years, respectively, and the mean BMI was 42.5 ± 7.0 kg/m^2. The pre-operative FBG and A1C levels were 196.0 ± 49.1 mg/dL and 8.1% ± 1.6%, respectively. In terms of T2DM status, 70.6% and 29.4% of the patients had 6.5% < A1c < 9% and A1c ≥ 9%, respectively (data not shown). The pre-operative levels of serum TG, TC, and LDL-C were 216.4 ± 171.8, 185.7 ± 40.4, and 122.5 ± 39.1 mg/dL, respectively (Table 1). Most of the parameters initially showed significant reductions at the 6th month after LSG.

Table 1 Changes in the anthropometric data and serum profiles of patients with obesity undergoing LSG preoperatively and 12 months after LSG

Variables	Pre-LSG	6th Month post-LSG	12th Month post-LSG
Anthropometric data			
Sex distribution(M/F, *n*)	44/51		
Age(M/F, y)	40.5 ± 8.3/42.9 ± 9.1	-	-
BMI (kg/m^2)	42.5 ± 7.0	32.8 ± 5.7*	31.1 ± 5.2*
Body weight (kg)	118.3 ± 24.3	91.8 ± 19.7*	86.4 ± 17.7*
Fat mass (%)	45.0 ± 6.5	36.6 ± 7.2*	34.0 ± 7.2*
WC (cm)	125.3 ± 15.7	104.1 ± 13.8*	99.7 ± 13.9*
EWL (%)	-	40.0 ± 17.2	56.4 ± 16.7$^+$
BMR (kcal)	1760.2 ± 302.2	1608.9 ± 247.6	1588.3 ± 240.0*
SBP (mmHg)	140.9 ± 18.7	131.3 ± 18.6*	132.5 ± 16.9*
DBP (mmHg)	88.3 ± 13.6	83.4 ± 12.2	85.1 ± 12.5
Serum profiles			
FBG(mg/dL)	196.0 ± 49.1	103.7 ± 29.9*	98.2 ± 23.7*
A1c (%)	8.1 ± 1.6	6.0 ± 1.1*	5.9 ± 0.8*
UA (mg/dL)	6.4 ± 1.7	6.2 ± 1.4	5.9 ± 1.4
TG (mg/dL)	216.4 ± 171.8	107.8 ± 48.3*	99.0 ± 49.0*
TC (mg/dL)	185.7 ± 40.4	184.6 ± 39.5	190.1 ± 42.6
LDL-C (mg/dL)	122.5 ± 39.1	122.2 ± 37.4	125.8 ± 37.7
HDL-C	21.1 ± 33.2	40.8 ± 16.7*	44.4 ± 18.1*

Data are presented as the mean ± standard deviation. Sex distribution was analyzed using the chi-square test

WC waist circumference, *BMI* body mass index, *WC* waist circumference, *EWL%* excess weight loss percentage, *BMR* basal metabolic rate, *DBP* diastolic blood pressure, *SBP* systolic blood pressure, *FBG* fasting blood glucose, *A1c* glycated hemoglobin, *UA* uric acid, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol

*Significant difference with $p < 0.05$ between pre-LSG and 6th month post-LSG determined using the paired-*t* test

+Significant difference with $p < 0.05$ between the 6th and 12th month post-LSG determined using the paired-*t* test

Comparing with pre-LSG, most of the parameters showed significant differences at the beginning of the 6th month of post-LSG; the similar outcomes were kept to the end of the 12th month, inclusive of BMI, body weight, fat mass percentage, SBP, FBG, A1c, TG, and HDL-C. The excess weight loss percentage (EWL%) exhibited substantial differences between the 6th and 12th months post-LSG, Table 1.

The T2DM remission profiles at the 6th and 12th months, respectively, after LSG of the 71 of the 95 obese with T2DM patients (by the definition of American Diabetes Association) (Table 2) were examined. Between the 6th- and the 12th-month after LSG, the BMI (32.4 ± 6.8 vs. 30.0 ± 4.5, $p < 0.05$), body weight (90.1 ± 17.0 kg vs. 84.1 ± 14.1 kg, $p < 0.05$), and EWL% (41.8 ± 17.0% vs. 58.6 ± 16.0%, $p < 0.05$) of the T2DM remission group showed significant differences; the non-T2DM remission group exhibited significant changes on EWL% (43.8 ± 16.2% vs. 52.7 ± 15.7%, $p < 0.05$), TC (213.3 ± 38.1 mg/dL vs. 181.3 ± 30.5 mg/dL, $p < 0.05$) and A1C (8.7 ± 0.8% vs. 7.1 ± 1.5%, $p < 0.05$) (Table 2). The non-T2DM remission group exhibited substantial declines in serum A1C and TC levels and a significant increment in EWL% at the 12th month compared with the 6th month after LSG. Comparing with the non-remission group, the T2DM remission group exhibited significant declines in systolic blood pressure (SBP), FBG levels, and A1C after LSG at both the 6th and 12th months. However, body weight, fat mass percentage, and serum TG level in the remission group emerged and marked decreases only at the 12th month. Although the BMI of the remission group did not reach a significant level compared with that of the non-remission group both at the 6th and 12th months after LSG; borderline significance was noted (32.4 ± 6.8 vs. 35.1 ± 7.0, $p = 0.087$ at the 6th month and 30.0 ± 4.5 vs. 34.9 ± 6.1, $p = 0.056$ at the 12th month). The ANCOVA results revealed that pre-surgical FBG level and pre-surgical A1C affected post-surgical T2DM remission profiles, by adjusting for age, sex, and BMI reduction ($p = 0.002$ and 0.014 for pre-surgical FBG and A1C at the 6th month and $p < 0.0001$ and $p = 0.009$ at the 12th month, respectively; data not shown).

Table 2 Comparison of anthropometric data and blood profiles of 71 obese patients with T2DM who experienced and did not experience T2DM remission at the 6th and 12th months after LSG

Variables	6th Month post-LSG			12th Month post-LSG		
	Remission $N=63$	Non-remission $N=8$	p value	Remission $N=65$	Non-remission $N=6$	p value
Age (y/o)	42.3 ± 9.0	44.6 ± 7.0	0.389	42.6 ± 9.0	42.5 ± 7.3	0.424
Sex M/F (n)	29/34	6/2	0.096	31/34	4/2	0.323
BMI (kg/m^2)	32.4 ± 6.8	35.1 ± 7.0	0.087	30.0 ± 4.5$^+$	34.9 ± 6.1	0.056
Body weight (kg)	90.1 ± 17.0	95.0 ± 14.2	0.351	84.1 ± 14.1$^+$	94.2 ± 12.7	**0.018**
WC (cm)	104.6 ± 12.5	102.2 ± 11.2	0.864	99.1 ± 12.7	99.0 ± 12.3	0.339
EWL (%)	41.8 ± 17.0	43.8 ± 16.2	0.747	58.6 ± 16.0$^+$	52.7 ± 15.7*	0.173
BMR (kcal)	1598.0 ± 216.3	1711.7 ± 174.2	0.518	1578.7 ± 208.0	1701.6 ± 181.9	0.206
Muscle mass (%)	35.0 ± 5.2	37.1 ± 7.6	0.754	36.9 ± 5.2	35.2 ± 4.7	0.130
Fat mass (%)	34.6 ± 6.9	36.7 ± 8.8	0.161	33.1 ± 7.1	36.7 ± 7.7	**0.050**
SBP (mmHg)	130.8 ± 17.7	145.0 ± 26.6	**0.016**	130.5 ± 17.8	152.2 ± 16.3	**0.008**
DBP (mmHg)	84.8 ± 12.2	82.0 ± 13.2	0.831	84.6 ± 13.1	90.8 ± 15.9	0.332
FBG (mg/dL)	97.1 ± 13.6	179.4 ± 51.0	**< 0.001**	95.0 ± 13.9	160.0 ± 50.1	**< 0.001**
A1C (%)	5.8 ± 0.8	8.7 ± 0.8	**< 0.001**	5.8 ± 0.6	7.1 ± 1.5*	**< 0.001**
UA (mg/dL)	6.2 ± 1.3	6.4 ± 1.4	0.309	5.9 ± 1.4	6.8 ± 1.7	0.111
TC (mg/dL)	187.0 ± 42.1	213.3 ± 38.1	0.949	189.9 ± 41.9	181.3 ± 30.5*	0.120
HDL-C (mg/dL)	41.2 ± 17.1	40.0 ± 25.0	0.581	45.0 ± 20.2	41.3 ± 18.0	0.950
LDL-C (mg/dL)	124.1 ± 39.2	115.0 ± 37.6	0.595	125.0 ± 36.2	143.3 ± 38.7	0.247
TG (mg/dL)	108.4 ± 49.9	131.4 ± 62.9	0.384	94.2 ± 44.4	140.3 ± 80.3	**0.013**
Hgb (mg/dL)	14.1 ± 1.6	14.9 ± 1.8	0.167	13.8 ± 1.8	14.8 ± 2.4	0.196

Of the 95 patients with obesity, 71 were diagnosed as having type 2 diabetes before LSG. All data are presented as the mean ± standard deviation. The chi-square test was performed to examine sex distribution. p value: comparison between T2DM remission and non-remission groups through analysis of covariance (ANCOVA) after adjustment for age, sex, and pre-surgical factors (e.g., BMI, FBG, and A1c). Considering collinearity, matched pre-surgical variables were excluded from variable analysis by ANCOVA

BUN blood urea nitrogen, *Cre* creatinine

*Significant difference ($p < 0.05$) in the non-T2DM remission group between the 6th and 12th months after LSG determined using the Mann–Whitney *U* test

+Significant difference ($p < 0.05$) in the T2DM remission group between the 6th and 12th months after LSG determined using the paired-*t* test

The status of T2DM remission between the 6th and 12th months after LSG is shown in Table 3. In the 71 obese patients with T2DM, the 63 and 65 individuals experienced T2DM remission (including partial- and complete-T2DM remissions) at the 6th and 12th months, respectively, after LSG. No significant differences in the T2DM remission ratio ($p = 0.501$, paired chi-square test) and sex distribution ($p = 0.317$, Fisher's exact test) were observed between the 6th and 12th months after LSG. After LSG, the 62 of the 71 obese patients with T2DM (29 men and 33 women) experienced persistent DM remission from the 6th month to the 12th month; only 3 patients had DM remission at the 12th month after LSG; 5 patients were consistently on non-remission, and only one patient exhibited inconsistent DM remission between the 6th and 12th months after LSG. The present findings indicated that the success rate of T2DM remission was relatively high (65/71 = 91.55%) within 1 year of post-LSG; the 62 of 71 patients (87.32%) start own T2DM remission at the 6th month of post-LSG and kept that remission up to 1 year after LSG.

Figure 1 presents the risk changes of CVD and CHD in cardiovascular primary care after LSG. The Framingham 10-year CHD risk estimates exhibited that no significant change had shown in both of male and female after LSG. However, the recalibrated Framingham 10-year CHD risk estimator which was based on the Chinese population exhibited significant declines after LSG (Fig. 1a; p for trends: 0.011 for female and 0.048 for male). The overall CVD risk considerably decreased from 32.4 to 17.2% in the men and from 15.8 to 5.8% in the female, respectively, after LSG (p for trend < 0.001; Fig. 1b). The estimated 10-year primary risk for ASCVD, heart and vascular age substantially showed decreased trends within 1 year after LSG. The estimated 10-year primary risk for ASCVD showed reductions from 7.6 to 2.98% and from 5.08 to 1.72% in the male and female patients after LSG, respectively (p values for trend < 0.001; Fig. 1c). The estimated heart and vascular age also displayed significantly lower within 1 year after LSG (decreased from 70.1 to 57.6 years old in the female and from 61.8 to 44.4 years old in the male; Fig. 1d).

Discussion

An increasing amount of evidence has indicated that bariatric surgery is more beneficial for T2DM remission compared with gastric banding [30]. Though LRYGB owns a relatively higher effectivity in terms of weight loss and the remission of obesity-related comorbidities [3–5, 31] than those of LSG; however, LSG might be the first consideration in Asia because of its simplicity, fairly safe, and lower malnutrition risk [19, 32]. The results of this study indicated that the EWL% of the patients with obesity was 56.4% ± 16.7% at 12 months after LSG (Table 1); this percentage was higher than that reported in a study including the Taiwanese population because of the higher baseline BMI of the patients enrolled in this study [33]. Although LSG is the primary procedure used to achieve weight loss and improve obesity-related metabolic abnormalities, the persistent efficacy of T2DM remission after LSG remains a controversy [34–36]. This pilot study also explored that issue in obese Taiwanese with T2DM after LSG. The results revealed simultaneous significant reductions in BMI, fat mass percentage, waist circumference, and TG and HDL-C levels and T2DM remission at the 6th month after LSG; this finding is similar to those of other studies [19, 30, 37–40]. Compared with other studies, this study demonstrated more considerable improvements in metabolic abnormalities and reduction in CVD risk. T2DM remission after LSG might occur through the fluctuation and secretion of incretins and through *FXR* gene interaction instead of the foregut or hindgut hypothesis [40]. LSG involves the removal of the majority of the stomach, resulting in decreased ghrelin and leptin secretions that induce weight loss and affect food intake, thus possibly resulting in diabetic remission [40, 41]. The results of this study indicated that T2DM remission corresponded to the decrements in body weight, BMI, and fat mass at the 6th month; thus we supposed that T2DM remission may be in response to an improved insulin activity through the reductions of waist-circumference and fat mass [19, 35]. However, comprehensive information regarding the *FXR* gene; insulin secretion; and bile acid, leptin, and ghrelin levels was lacking in this study.

Table 3 Change of T2DM remission in the total of 71 obese patients with diabetes mellitus between the 6th and 12th months after LSG

		Remission at the 6th month		The change of remission ratio between 6 and 12th months ($p = 0.501$)
	N (M/F)	Yes	No	*p* value for sex distribution
Remission at the 12th month	Yes	62 (29/33)	3 (2/1)	0.371
	No	1 (0/1)	5 (4/1)	

McNemar's paired chi-square test for the change in DM remission status between the 6th and 12th months after LSG. Fisher's exact test was performed to examine the effect of sex on changes in DM remission 6 and 12 months after LSG

M male, *F* female

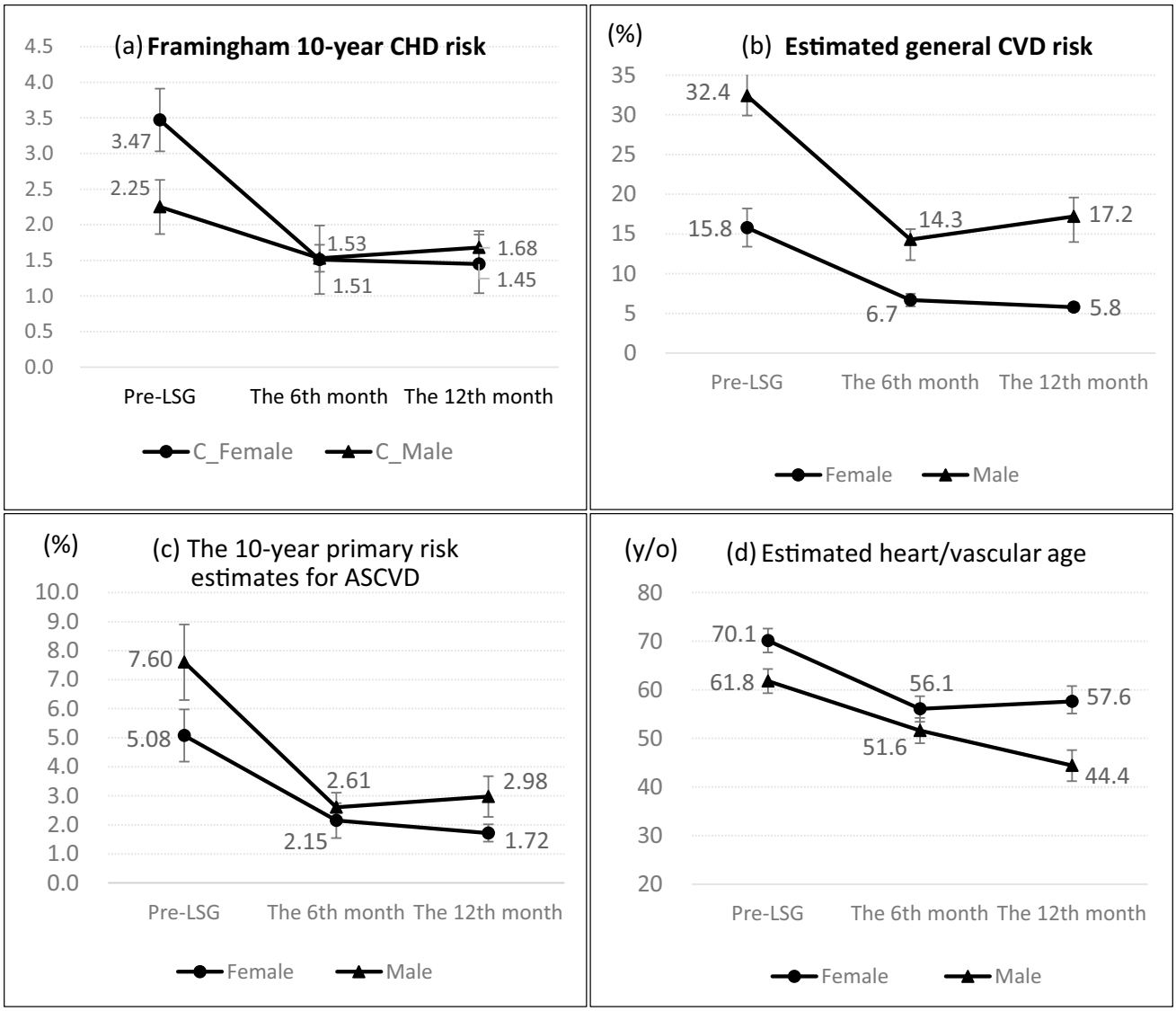

Fig. 1 Changes in quantified estimators of cardiovascular disease risk in 95 patients with obesity after LSG. The *P* value for trend determined using the repeated general linear model is labeled on each panel. All data are expressed as the mean and standard error and labeled on the two sides of each sign. Framingham estimates of the 10-year CHD risk. C_Female and C_Male indicate estimates derived from the recalibrated Framingham 10-year CHD risk functions in the Chinese population. General CVD risk estimates from the Framingham study. The 10-year primary risk estimates for ASCVD. Estimates of heart or vascular age determined using the CVD risk prediction model

Additional studies should re-examine the synergistic action of the *FXR* gene and LSG. Lee et al. reported that older age is a negative predictor of T2DM remission after bariatric surgery [34]. However, the ANCOVA results revealed that pre-surgical FBG level and A1C might predominantly affect the success rate of T2DM remission more than age after LSG (Table 2) (data not shown). Thus, the synergistic effect of pre-surgical factors (e.g., pre-surgical C-peptide level, homeostasis model sssessment [HOMA]-insulin resistance index [HOMA-IR], HOMA-beta, A1C, FBG level, and DM duration) might effect T2DM remission and its persistent capacity after LSG. In addition, insulin and C-peptide are secreted in response to elevated blood glucose; the pre-surgical HOMA-IR index and C-peptide level might play the other roles in T2DM [42] because the HOMA-IR index and C-peptide level might exhibit different responses among individuals with varying physiological statuses and T2DM durations [42]. Their levels above the normal range indicate a compensatory response in the initial stage of insulin resistance under normal beta-cell function, whereas lower levels are noted in late-stage T2DM because of the progressive destruction of beta-cell function [40]. Although this study demonstrated an association of the pre-surgical HOMA-IR index and C-peptide level with T2DM remission at the 6th

month after LSG (data not shown), we did not explore the associations between T2DM remission and the HOMA-IR index or C-peptide level because of the limited physiological data and imprecision in the self-reported T2DM duration. Although numerous models related to T2DM remission after bariatric surgery have been proposed, several predictors of DM remission after LSG remain controversial [15, 18, 31, 42]. Thus, subsequent studies including a larger sample size should elucidate the underlying mechanism of T2DM remission after LSG.

Relatively rare studies have focused on the persistence of T2DM remission and risk changes of CVD and CHD after LSG in primary cardiovascular disease care. This study indicated that 63 of the 71 obese patients with T2DM experienced DM remission before the 6th month after LSG, and 62 of the 63 patients kept a persistent DM remission up to 1 year. Unfortunately, T2DM recurrence was noted in one patient at the 12th month because of surgical complication managed by parental nutritional support, which resulted in a poor glycemic control. In addition, 3 (2 men and 1 woman) of the 71 patients were observed to have T2DM remission in the interval between the 6th and 12th months after LSG (the totally success rate of T2DM remission reached to 91.55% at the post-surgical 1 year); this finding is similar to those of other studies conducted in Taiwan [18, 35]. The results of this study indicated that most of the patients experienced T2DM remission before the 6th month after LSG and that the remission persisted until the 12th month after LSG.

The risks of CVD and CHD are close to obese degree, T2DM severity, and metabolic abnormalities [43]. Several LSG studies have only focused on T2DM remission instead of simultaneously exploring the risk reductions of CVD and CHD. This study also evaluated whether the risks of CVD and CHD decreased after LSG by using various proven predictive models. Multi-variable Framingham function is a prominent prediction model for 10-year CHD risk [24–27]. We applied this model to estimate the 10-year CHD risk after LSG. This is the first attempt to predict the risks of CVD and CHD in patients with obesity after LSG. By using validated multi-variable Framingham functions, we observed that all of the 95 patients with obesity did not exhibit a significant reduction in the 10-year risk of CHD after LSG [24]. Liu et al. reported that multi-variable Framingham functions caused a bias in the prediction of the 10-year CHD risk in Chinese population and therefore developed a recalibrated multivariable Framingham function based on the Chinese population [22]. We re-evaluated the 10-year CHD risk by applying the recalibrated Framingham functions because of the genetic similarity. The results showed that LSG not only considerably improved metabolic abnormalities but also declined the 10-year CHD risk in obese Taiwanese with BMI more than 35 (Fig. 1a). SBP is associated with the risks of CVD, CHD, heart failure, stroke, and ASCVD. In addition, SBP is related to the all-cause mortality of general CVD [44–48]. We noted that the overall CVD risk (Fig. 1) appeared to synchronously decrease with weight loss, improvement in metabolic abnormalities, and reduction in diastolic blood pressure; this finding is similar to those of other studies [44–46, 48–54]. Although whether SBP alone can predict general CVD risk is unknown [55]; most studies have indicated that SBP is a predominant indicator on the primary care of general CVD [44, 45, 48]. A new Framingham model [29] and the ASCVD multiple function [28] have been developed to predict the 10-year risk of CVD and stroke for a broader field on the primary care of total CVD cases. Figure 1b and d also reveal that LSG effectively reduced the 10-year risk of general CVD and heart and vascular age. The LSG exerted a reduction on CVD risk possibly derived not only from weight loss but also from DM remission, SBP, and blood lipid reduction. Our preliminary data revealed that LSG owns beneficial therapeutic effects on obesity and metabolic abnormalities (blood pressure disorder, high lipid, and glucose levels) and that synergistic effect potentially reduces heart and vascular physiological ages, general CVD, and CHD risks.

Subsequent studies with a larger sample size should collect data regarding additional biomarkers and precise DM duration to elucidate the detailed mechanism through which LSG reduces cardiovascular risk.

Conclusion

T2DM remission at post-surgical 6 months had been observed, and this phenomenon remained persistent up to 1 year. The LSG evidently resulted in weight loss, T2DM remission, improved metabolic abnormalities, and subsequently, showed beneficial effectiveness for the primary cares of CVD and CHD in obese patients.

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Declarations

Conflict of Interest The authors declare no competing interests.

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