Dynamic changes in aortic stiffness after substantial weight loss by laparoscopic sleeve gastrectomy in patients with obesity: a 1-year follow-up study

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Accepted 9 March 2021

To cite: Gul M, Inci S, Aktas H, *et al*. *J Investig Med* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/jim-2021-001778

ABSTRACT

Arterial stiffness has been identified as a powerful and independent risk factor for cardiovascular disease. Obesity is associated with an increased risk of aortic stiffness (AS) and adverse cardiovascular events. Herein, we aimed to evaluate the effects of weight loss after laparoscopic sleeve gastrectomy (LSG) on AS in individuals with morbid obesity by using the transthoracic echocardiography (TTE).

A total of 53 patients with obesity (17 males, 36 females) who underwent LSG and did not have any known heart disease were included in the study. The AS parameters were measured with TTE. The demographic and echocardiographic data of the patients were studied before, at 1 month and 12 months after surgery.

The mean age of the study group was 34.41±11.62, 68% of whom were female. There were no significant differences in terms of the standard echocardiography and Doppler measurements as compared with preoperative values (all p>0.05). When the elastic parameters of the aorta were compared, no significant differences were detected regarding aortic strain (%) ((16.28±4.11) vs (16.68±4.56), p=(0.998)), distensibility (cm^2/dyn) ((6.74±1.78) vs (7.03±2.31), p=(0.997)) and Aortic Stiffness Index values ((10.73±3.84) vs (10.63±3.34), p=0.998) between baseline and first month after surgery. In the 12-month follow-up, it was determined that the aortic strain ((16.28±4.11) vs (22.74±5.79), p≤0.001) and distensibility ((6.74±1.78) vs (10.34±3.059), p<0.001)) values increased at significant levels.

Weight loss by LSG improves arterial stiffness parameters in patients with obesity over a 1-year follow-up.

Significance of this study

What is already known about this subject?

- Aortic strain is used as a measure for assessing the early stages of arterial stiffening.
- It has been reported that reductions in the aortic strain and distensibility were considered as an early marker of vascular stiffness in humans.
- Furthermore, weight reduction in individuals with obesity has been associated with increased aortic distensibility.

What are the new findings?

- In our study, the aortic stiffness data of the patients with obesity who underwent laparoscopic sleeve gastrectomy surgery were evaluated by using the transthoracic echocardiography method; and improvements were shown in the mid-term follow-up after surgical procedure.

How might these results change the focus of research or clinical practice?

- This technique appears as a simple, reliable and applicable to evaluate the routine follow-up of aortic stiffness, which is an important predictor for cardiovascular diseases.

INTRODUCTION

Obesity is a prevalent and growing public health problem worldwide. According to data from WHO, 39% of the global population above 18 years of age are overweight and of these, 13% have obesity.[1] Thirty-one per cent of patients with obesity suffer from the metabolic syndrome, which doubles the risk for coronary disease and stroke, and increases total mortality by 1.5-fold.[2–4] Moreover, it has been identified as an independent risk factor for cardiovascular morbidity and mortality.[5] Many studies have suggested that obesity is associated with an increased risk of aortic stiffness (AS).[6–8] Indeed, the presence of arterial stiffness has been shown in individuals with obesity, even if they are as young as 20–30 years.[8] Therefore, evaluation of AS is recommended in the management of patients with obesity by the European Society of Obesity Studies and the European Society of Hypertension.[9]

The elasticity of the aorta could be explained as dilating by increasing pressure in systole and recoiling slowly to its initial shape when blood pressure falls in diastole.[10] Arterial stiffness describes the rigidity of the arterial wall, which is primarily determined by structural components of the arterial wall, and vascular smooth muscle tone. Large arteries lose their elasticity over time due to elastin degeneration, collagen accumulation and thickening of the arterial wall.[10] As the aortic wall stiffening, its buffering capacity decreases gradually, which leads to an increase in cardiac afterload, reduction in diastolic coronary flow and damage microcirculation in high-flow organs such as the kidneys and brain.[11] It is well known that large arterial stiffening contributes to the development of incident hypertension, stroke and cardiovascular events.[12] AS has also been associated with vascular aging, which is considered an independent predictor of cardiovascular mortality and morbidity.[13]

Bariatric surgery produces significant weight loss and reduces mortality and morbidity in patients with established cardiovascular disease.[14] Laparoscopic sleeve gastrectomy (LSG), a safe and effective bariatric surgical procedure, is expected to have a favorable effect on many systems, especially on the cardiovascular system.[15] Although several studies have already revealed favorable modulation of the LSG on the cardiovascular system,[16] there is limited data regarding the effect of LSG on AS. Herein, we aimed to evaluate the short-term and mid-term effects of substantial weight loss on AS in individuals with morbid obesity. We hypothesized that AS would be improved increasingly and continuously after bariatric surgery.

METHODS

Design and study population

This study was conducted as a prospective manner between August 2017 and February 2019 in a single tertiary healthcare center. The eligibility criteria were age superior to 18 years, body mass index (BMI) ≥40 kg/m^2 without comorbidities or a BMI ≥35 kg/m^2 with additional comorbidity such as type 2 diabetes mellitus (DM), hypertension and obstructive sleep apnea. Exclusion criteria were the presence of coronary heart disease, decompensated heart failure, systolic dysfunction (ie, left ventricular ejection fraction <50%), moderate or severe valvular heart disease, chronic kidney disease, active malignancy, low-quality image or other metabolic diseases and the possibility of endangering safe anesthesia (moderate or severe obesity hypoventilation syndrome included).

Risk factors for atherosclerosis such as hyperlipidemia, DM and hypertension were analyzed. Hyperlipidemia was defined as fasting total cholesterol ≥200 mg/dL or low-density lipoprotein cholesterol ≥130 mg/dL or fasting triglycerides ≥200 mg/dL. DM was defined as a history of DM or a fasting glucose ≥126 mg/dL. Hypertension was defined as systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg, or both, in at least three measurements, or a history of hypertension.

Patients with obesity who had previously failed to weight loss with conservative methods (diet, exercise schedules, pharmacological agents and so on) underwent LSG. A total of 57 patients who underwent LSG were examined. Those two patients with poor image quality were excluded from the study. Two patients did not come to follow-up visits. Finally, data for the remaining 53 patients were analyzed. Demographic, clinical, laboratory and echocardiographic measurements of the patients were recorded before, as well as 1 and 12 months after the surgery. The main objective was to evaluate the early and mid-term impact of LSG on the AS.

Our study followed the Strengthening the Reporting of Observational Studies in Epidemiology guidelines.[17]

Transthoracic echocardiography

Echocardiographic examinations were performed by EPIQ 7 digital ultrasound scanner (Philips Medical System, USA) in the left lateral decubitus position from multiple windows. All measurements were taken by two experienced cardiologists who were blinded to the clinical status of the patients. A single-derivation ECG was simultaneously recorded during the examination. Standard echocardiographic images (parasternal long-axis, parasternal short-axis, apical four-chamber and apical two-chamber views) were captured and evaluated by motion mode (M-mode), two-dimensional (2D), continuous stream Doppler and tissue Doppler imaging according to the American Echocardiography Association's criteria.[18] Left trial diameter (LA; mm), left ventricle end-diastolic diameter (LVEDD; mm), left ventricle end-systolic diameter (LVESD; mm), interventricular septum diameter (IVS; mm) at end-diastole and posterior wall diameter (PW) at end-diastole were obtained from the M-mode echocardiographic tracing under the guide of 2D. Left ventricular ejection fraction (EF; %) was calculated with Simpson's method as (diastolic volume−systolic volume)/(diastolic volume).

Diastolic functions were evaluated by measurement of transmitral flow parameters including the early (E) and late (A) diastolic filling velocities, and the E/A ratio from an apical four-chamber view with the sample volume sited at the tip of the mitral leaflet. Tissue Doppler velocities, a useful echocardiographic technique for the assessment of regional and global myocardial function, were measured from the septal and lateral annulus in apical four-chamber view. The peak systolic velocity (Sm), early diastolic myocardial peak velocity (Em), late diastolic myocardial peak velocity (Am) and E/Em ratio (as the ratio between the E transmitral flow velocity and mean of the lateral and septal walls Em velocity) were measured. The values were determined by averaging three subsequent beats.

M-mode rod was placed so that it passed through the aortic region and was 3 cm distal to the aortic valve, systolic and diastolic diameters of the ascending aorta were obtained from the aortic trace. Systolic diameter was measured from the location of the aortic trace in which maximum forward movement was observed, while diastolic diameter was measured from the location that corresponded to the R spike of the ECG. To determine the pulse pressure (PP), which was necessary for calculating the relevant parameters, systolic blood pressure and diastolic blood pressure were concurrently measured using a mercury sphygmomanometer; the difference between the two blood pressure values was recorded as the pulse pressure. AS was non-invasively calculated with TTE based on the relationship between changes in aortic diameter and pressure with each cardiac

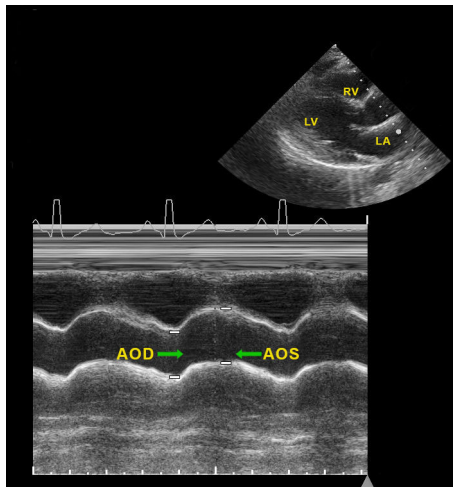

Figure 1 Systolic and diastolic diameter measurements of the ascending aorta via M-mode echocardiography. AOD, aortic diastolic diameter; AOS, aortic systolic diameter; LA, left atrium; LV, left ventricle; RV, right ventricle.

pulse. Mean systolic and diastolic measurements were calculated based on three consecutive measurements. AS was assessed using the established aortic elasticity parameters, consisting of aortic distensibility, aortic strain and Aortic Stiffness Index (ASI)[19] (figure 1). These parameters were formulated as:

$$\text{Arotic strain}(\%) = (\text{systolic aortic diameter} - \text{diastolic arotic diameter}) \times 100/\ \text{diastolic diameter}$$

$$\text{Distensibility}\ (\text{cm}^2/\text{dyn}) = 2(\text{aortic strain}) / (\text{systolic pressure} - \text{diastolic pressure})$$

$$\text{ASI} = \text{In}(\text{systolic blood pressure}/\text{diastolic blood pressure})/ \left[\text{aortic systolic diameter} - \text{aortic diastolic diameter}/\text{aortic diastolic diameter}\right]$$

To investigate changing parameters, deltas were calculated by subtraction of postoperative values from preoperative ones. The absolute value of the AS parameters were:

Delta ASI: post-LSG ASI value–pre-LSG ASI value.

Delta aortic strain: post-LSG aortic strain value–pre-LSG aortic strain value.

Delta distensibility: post-LSG distensibility value–pre LSG distensibility value.

Statistical analysis

Kolmogrov-Smirnov and Shapiro-Wilk tests were used to test normality. According to results, non-parametric tests were preferred. Continuous variables such as IVS, PW and the others were compared using Friedman's test among baseline and the postoperative first month and the year. Dunn's post hoc multiple comparison tests were used to know which time points differ from which others.[20] General descriptive statistics are summarized as mean±SD for continuous variables. And 'p' value of <0.05 was considered statistically significant. The parameters such as Δaortic strain, Δaortic distensibility, ΔASI and Δweight were calculated by subtracting the first measurement (baseline) from the last measurement (first year). And the Spearmen's correlation coefficients were obtained and summarized with respective p values. In addition, intraobserver and interobserver agreement of the measurements of 15 randomly selected patients was assessed by two-way random model of intraclass correlation (ICC) and given by their 95% CIs. SPSS V.11.5 for Windows were used for all these statistical analyses.

RESULTS

A total of 53 patients (17 males and 36 females) who underwent LSG and completed at least 12 months postoperative follow-up visit were included for analysis. The mean age of the study group was 34.41±11.62, 68% of whom were female. The mean BMI was 44.11±2.66 kg/m^2, and the initial mean weight was 133.32±15.62 kg before the LSG. Among them, 10 patients (19%) had hypertension, 8 patients (15%) had DM and 19 patients (35%) had dyslipidemia. Furthermore, 13 (%25) patients were current smokers at the time of inclusion. Although there was no surgical morbidity or mortality, only three patients had wound infection that was fully recovered by medical treatment. No major complications were detected during the 1-year follow-up of the patients. There was a substantial improvement in terms of weight loss, aortic strain, distensibility and ASI in which absolute delta values were 33.11±10.84 kg, 6.50%±3.20%, 3.59±1.99 cm^2/dyn and 3.16±2.32, respectively. The prevalence of hypertension decreased from 19% to 9% (p=0.003) with highly significant decrease in systolic blood pressure (p=0.004) and diastolic blood pressure (p=0.026). Similarly, the prevalence of DM, dyslipidemia and smoking was also significantly decreased (p<0.05) after surgery. Considerable improvement of all lipid subfractions was observed during follow-up (p<0.05) (table 1).

Of note, there were no significant differences in the conventional echocardiographic measurements (IVS, LVEDD, LVESD, LA, PW, left ventricular EF and diastolic filling velocities (E, A and E/A ratios) as compared with preoperative values (p>0.05) (table 2). Similarly, there was also no significant difference in tissue Doppler parameters (Sm, Em, Am, E/Em ratio) that were measured from the lateral mitral annulus.

Although there was a non-significant increase in aortic strain at 1 month ((16.28±4.11) vs (16.68±4.56), p=(0.998)), a statistically significant increase was observed at 1-year follow-up compared with baseline ((16.28±4.11) vs (22.74±5.79), p≤0.001) (table 2). The distensibility parameters were measured as 6.74±1.78 (cm^2/dyn) at baseline, and 7.03±2.31 (cm^2/dyn) 1-month follow-up (p=0.997). Similar to the aortic strain measurements, aortic distensibility was also increased significantly in the 1-year follow-up compared with baseline ((6.74±1.78) vs (10.34±3.059, p<0.001)). After LSG, the ASI decreased non-significantly at 1 month ((10.73±3.84) vs (10.63±3.34), p=0.998), but a statistical significant decrease was observed at 1-year follow-up ((10.73±3.84) vs (7.56±2.19), p<0.001) (table 2).

In the correlation analysis, delta aortic strain exhibited a positive and significant correlation with delta weight (r=0.66, p<0.001). Similarly, a positive correlation was

Table 1 Clinical, demographic and laboratory characteristics of the study population at baseline and follow-up after surgery

	Baseline	12 months	P value
Age (years, mean±SD)	34.41±11.62	35.64±11.57	0.968
Sex, female (n/%)	36 (68%)	–	–
Weight (kg)	133.32±15.62	101.46±11.27	<0.001
BMI (kg/m^2)	44.11±2.66	31.92±5.78	<0.001
Hypertension prevalence (n/%)	10 (19%)	5 (9%)	0.003
Diabetes mellitus (n/%)	8 (15%)	5 (9%)	0.041
Dyslipidemia (n/%)	24 (50%)	13 (24%)	<0.001
Smoking (n/%)	13 (27.1%)	4 (8%)	0.024
SBP (mm Hg)	129.90±11.38	109.24±11.23	0.004
DBP (mm Hg)	88.94±8.14	74.56±4,62	0.026
Glucose (mg/dL, mean±SD)	112.62±38.95	88.64±66.84	0.001
Low-density lipoprotein cholesterol (mg/dL, mean±SD)	121.43±31.39	99.21±24.97	0.001
High-density lipoprotein cholesterol (mg/dL, mean±SD)	45.66±11.76	56.24±10.41	0.001
Triglycerides (mg/dL, mean±SD)	167.85±49.59	95.41±42.17	0.023

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.

detected between delta distensibility and delta weight (r=0.76, p<0.001). Furthermore, a negative and significant correlation was found between the ΔASI and Δweight (r=−0.32, p<0.018) (figure 2, table 3).

For the variables of aortic strain, distensibility and ASI, the ICCs were calculated according to the two-way random and absolute agreement model. Intraobserver and interobserver reliability statistics obtained for the measurement of 15 randomly selected patients are given in the table 4.

DISCUSSION

The main finding of the present study is that weight loss via LSG reduces AS in patients with morbid obesity. Furthermore, a significant and positive correlation was found between delta weight and aortic elasticity properties during the 1-year follow-up.

Aortic elastic properties consisting of aortic strain, distensibility and stiffness index are used as a measure for assessing the early stages of arterial stiffening.[10] It has been reported that reductions in the aortic strain and distensibility were considered as an early marker of vascular stiffness in humans.[21] Furthermore, weight reduction in individuals with obesity has been associated with increased aortic distensibility.[22] Also, the ASI, which is a good characteristic of aortic elasticity, was increased in patients with obesity compared with normal subjects.[23] In our study, the aortic strain and distensibility increased at 1-year follow-up, whereas the ASI was decreased significantly. In this regard, our study findings revealed that aortic elasticity parameters significantly improved 12 months after LSG, correlating with weight loss.

Many factors have been suggested to explain the relationship between AS and the cardiovascular system. AS is associated with increased systolic blood pressure, which leads to an increase in left ventricular afterload, ventricular hypertrophy and oxygen demand of the heart.[24 25] Left ventricular hypertrophy is a well-known risk factor for congestive heart failure and cardiovascular adverse events.[24 25] As a result of the decreased diastolic blood pressure due to AS, coronary perfusion deteriorates gradually and leads to subendocardial ischemia. Despite the increase in oxygen demand, decreased coronary blood flow causes an ischemia in the myocardia. Multiple studies have specifically correlated that increased

Table 2 Echocardiographic and aortic elasticity characteristics of the study population at baseline and at follow-up after surgery

	Baseline	First month	First year	P value*	P1†	P2†	P3†
IVS (mm)	12.31±1.35	12.04±1.21	11.94±1.02	0.890			
PW (mm)	12.11±0.80	12.12±0.77	12.13±0.78	1.000			
LVEDD (mm)	46.79±3.38	46.03±3.69	46.39±3.38	0.531			
LVESD (mm)	29.62±3.12	29.45±3.13	28.73±2.92	0.424			
LA (mm)	36.54±8.25	36.25±7.97	35.45±7.22	0.120			
EF (%)	59.81±3.96	59.82±3.94	59.85±3.95	1.000			
E (m/sn)	72.73±8.63	72.67±8.13	72.32±8.09	0.399			
A (m/sn)	61.00±6.26	61.39±5.83	61.75±5.35	0.120			
E/A	1.20±0.17	1.17±0.16	1.16±0.16	0.257			
Sm (cm/s)	12.75±1.81	12.56±1.79	12.66±1.50	0.323			
Em (cm/s)	11.18±1.54	11.13±1.30	11.43±1.26	0.131			
Am (cm/s)	9.55±1.20	9.58±1.02	9.49±1.11	0.151			
E/Em	6.81±1.31	6.74±1.22	6.49±1.00	0.139			
Aortic strain (%)	16.28±4.11	16.68±4.56	22.74±5.79	**<0.001**	0.998	**<0.001**	**<0.001**
Aortic distensibility (cm^2/dyn)	6.74±1.78	7.03±2.31	10.34±3.05	**<0.001**	0.997	**<0.001**	**<0.001**
Aortic Stiffness Index	10.73±3.84	10.63±3.34	7.56±2.19	**<0.001**	0.998	**<0.001**	**<0.001**

Results are shown as mean±SD. P1=baseline vs 1st month; P2=1st month vs 12th month; P3=baseline vs 12th month.
Statistically significant p values are shown in bold.
*Friedman's test.
†Dunn's post hoc multicomparison test result.
A, late; Am, late diastolic myocardial peak velocity; E, early; E/Em ratio, ratio between the E transmitral flow velocity and mean of the lateral and septal walls Em velocity; EF, ejection fraction; Em, early diastolic myocardial peak velocity; IVS, interventricular septum; LA, left atrium; LVEDD, left ventricle end-diastolic diameter; LVESD, left ventricle end-systolic diameter; PW, posterior wall; Sm, peak systolic velocity.

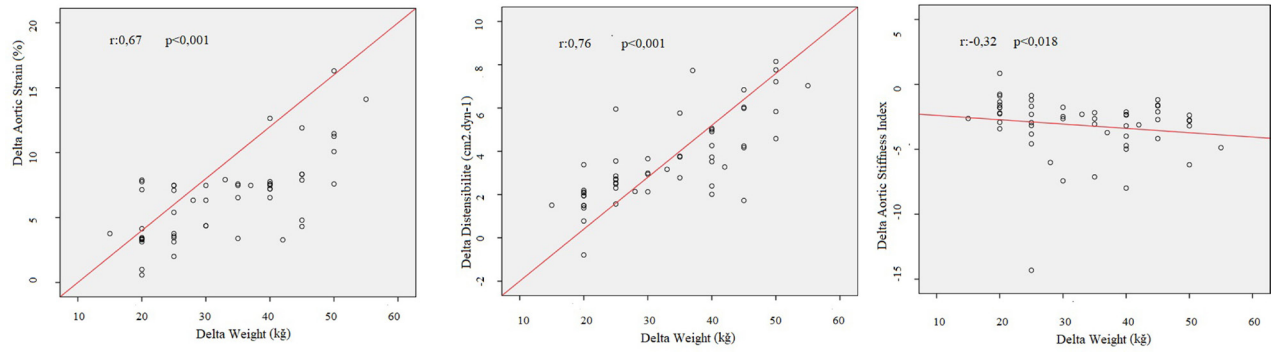

Figure 2 Scatter plots of significance correlations between delta weight and aortic stiffness parameters.

arterial stiffness with atherosclerosis.[26 27] For these reasons, AS is considered a strong predictor for adverse cardiovascular events.

The pathophysiological mechanisms of the relationship between obesity and arterial stiffness remain unclear. Seals and Gates[28] speculated that obesity may induce oxidative stress due to the presence of excessive adipose tissue. In this case, extracellular matrix proteins lead to changes in vascular structure and functions by an increase in vasoconstrictive, vasodilator molecules and vascular muscle cells. These molecular changes may contribute to the development of arterial stiffness. Another critical point is that insulin resistance, most of which accompany obesity, results in vascular endothelial dysfunction and vasoconstriction in obese people.[29 30] Increased insulin levels may also be associated with vascular effects like proliferation in vascular muscle cells and increased collagen production.[31 32] It has been shown that the high glucose levels that occur in insulin resistance stimulate collagen synthesis and bind to protein fibers.[33]

Adipocytes and preadipocytes have been identified as a source of proinflammatory cytokines, including tumor necrosis factor-α, interleukin (IL)-1 and IL-6; thus, obesity is considered a state of chronic inflammation.[34] We know that inflammation has a potent role in the pathogenesis of arterial stiffness.[35] Similarly, increased production of angiotensin 2 levels from the adipose tissue may contribute to inflammation and vascular deterioration.[36] High angiotensin 2 levels may cause increased blood volume in the circulation because of sodium involvement and vascular changes with shear stress change.[37] It has been revealed that arterial diameter and wall thickness increased with obesity,[38] which can also lead to increased arterial stiffness.

Increased arterial stiffness (regardless of age, gender and blood pressure) was closely associated with abdominal fat deposition and general adiposity.[39] In these studies, the abdominal visceral fat tissue was measured with tomography and was shown as a strong predictor of AS.[40 41] Wildman *et al*[42] showed that there was a strong association between weight gain and AS progression in healthy young individuals in 2 years follow-up. Mahfauz *et al*[43] found similar results in accordance with Wildman *et al*. However, Benetos *et al*[44] failed in showing this relationship. Although this situation is confusing, the fact that the patient group of the study conducted by Benetos *et al* was elderly may explain the situation partly. Furthermore, it has been shown that there is a difference between arterial stiffness in male and female populations, especially in the postmenopausal period.[45] On the other hand, the fact that most of our study patients were in the premenopausal state may have affected the study results.[45] Iancu *et al*[46] found that the basal aortic elasticity parameters correlated with preoperative BMI, waist circumference, systemic hypertension, blood glucose levels and aortic elastic parameters. We could hypothesize that AS, which is impaired by many mechanisms in patients with obesity, would be improved after LSG follow-up.

Although there are many studies on AS in patients with obesity, there are limited data regarding the effect of weight loss after bariatric surgery. Rider *et al*[47] showed that significant improvements were in AS as a result of losing weight via diet. The effect of surgical weight loss by LSG on AS

Table 3 Correlation coefficients of aortic stiffness parameters with weight loss (Spearman's rho)

	ΔAortic strain (%)	ΔDistensibility (cm^2/dyn)	ΔAortic Stiffness Index
ΔWeight (kg)			
Rho (r)	0.666	0.766	0.324
P value	**<0.001**	**<0.001**	**0.018**
N	53	53	53

Statistically significant p values are shown in bold.

Table 4 Reliability demonstrated by intraclass correlation coefficient (ICC) and 95% CIs for the aortic stiffness parameters

	ICC	95% CI		P value
		Lower bound	Upper bound	
Interobserver				
Aortic strain (%)	0.84	0.65	0.99	**0.002**
Aortic distensibility (cm^2/dyn)	0.88	0.69	0.97	**0.001**
Aortic Stiffness Index	0.82	0.58	0.91	**0.011**
Intraobserver				
Aortic strain (%)	0.97	0.88	0.99	**<0.001**
Aortic distensibility (cm^2/dyn)	0.98	0.86	0.97	**<0.001**
Aortic Stiffness Index	0.94	0.78	0.99	**<0.001**

Statistically significant p values are shown in bold.

parameters remains unclear. Many studies have revealed a significant improvement in inflammation, sympathetic activity and endothelial dysfunction after LSG[48 49] in patients with obesity. Also, Iancu *et al*[46] showed that there were significant improvements in the aortic elastic parameters at 6-month and 1-year follow-up after LSG. In the light of these data, it may be considered that many pathophysiological conditions that are caused by obesity may recover after weight loss. Endothelial functions are likely to return normal with the activation of the nitric oxide system, which is one of the main components of endothelial dysfunction.[50] Increased plasma levels of endothelial activation markers in individuals with obesity reflect the positive association between cardiovascular disease and obesity.[51] Nijhuis *et al* monitored patients' endothelial cell activation markers for 2 years after bariatric surgery and observed that bariatric surgery can reduce endothelial activation in the long-term.[51] The decrease in inflammation, which is one of the crucial pathophysiological components can be considered as one of the leading causes of this improvement.

Bariatric surgery increases the concentrations of adiponectin, an anti-apoptotic and anti-inflammatory cytokine, whereas decreases the levels of resistin, which has been shown to cause atherogenesis.[52] Although the cross-linking of collagen and protein is irreversible, we have demonstrated a reduction in obesity-related comorbidities. These improvements may have a positive effect on cardiovascular morbidity and mortality with LSG. The fact that the correlation between weight change and stiffness parameters during the follow-ups confirms the relationship between adipose tissue and AS reported in previous studies. Weight loss after LSG is an important parameter for the recovery of AS. Motivation and therapies to the continuation of lifestyle changes after LSG can positively contribute to the AS improvement in these patients.

Obesity is closely associated with numerous comorbidities such as hypertension, dyslipidemia, ischemic heart disease, stroke and DM.[5] Both obesity and its comorbidities are well-known risk factors for the development of AS.[6] And it is well known that effective management of atherosclerotic risk factors are an important goal for protection from arterial stiffness.[53] Bariatric surgery provides effective weight loss and leads to significant improvements in cardiovascular outcomes. It has also been shown that LSG reduces the long-term estimated risk of coronary heart disease in patients with obesity.[54] We revealed a significant improvement in comorbidities that are independent predictors of AS, which was compatible with the findings of previous reports.[46] We believe that bariatric surgery will enhance future cardiovascular health for individuals with obesity.

LIMITATIONS

This study should be evaluated in light of some limitations. First, our study was conducted as a single-centered and not randomized. Second, our study population was relatively small, further clinical randomized trials with greater participation can offer strong statistical data. Also, our study cohort was not homogenous in terms of gender, so results of this study may not be directly applicable to the whole population. Third, we did not have the opportunity to use the pulse wave rate in measuring arterial stiffness since this method is not used routinely in our clinical practice. Finally, although our study was a follow-up study, only 1-year follow-up was carried out, so studies with longer follow-up periods may be a need.

CONCLUSION

In our study, the AS data of the patients with obesity who underwent LSG surgery were evaluated by using the transthoracic echocardiography method; and improvements were shown in the mid-term follow-up after surgical procedure. In addition, this improvement was found to be correlated with weight loss change. This technique appears as a simple, reliable and applicable to evaluate the routine follow-up of AS, which is an important predictor for cardiovascular diseases. Further prospective and long-term studies are needed in this respect.

Clinicians should be aware of bariatric surgery benefits and encourage patients to bariatric surgery along with strict dietary and exercise recommendations to achieve sustained weight loss and improve cardiovascular risk.

Contributors Conception and design: MG, SI and NO. Data collection and processing: MG, SI, OY, HA and YA. Analysis and interpretation: NO, SI, YA and OY. Literature review: MG, SI, HA and OY. Writer: MG and SI. Critical review: MG, SI, OY, HA, YA and NO.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not required.

Ethics approval All the procedures were in accordance with the ethical standards of our institutional, and national research committee, and with the 1964 Helsinki Declaration. This study was approved by the local ethics committee (Necmettin Erbakan University, No: 2019 84/1756, March 1, 2019). The informed and signed consents were obtained from all patients.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement No data are available.

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REFERENCES

1 WHO. Risk factors. Available: https://www.who.int/gho/ncd/risk_factors/overweight/en/2020 [Accessed 30 May 2020].
2 Mohammedi K, Compaoré A, Potier L, *et al*. Outpatient measurement of arterial stiffness in patients with type 2 diabetes and obesity. *J Diabetes* 2017;9:237–42.
3 Engin A. The definition and prevalence of obesity and metabolic syndrome. *Adv Exp Med Biol* 2017;960:1–17.
4 Kim HM, Kim DJ, Jung IH, *et al*. Prevalence of the metabolic syndrome among Korean adults using the new international diabetes Federation definition and the new abdominal obesity criteria for the Korean people. *Diabetes Res Clin Pract* 2007;77:99–106.
5 Zhang C, Rexrode KM, van Dam RM, *et al*. Abdominal obesity and the risk of all-cause, cardiovascular, and cancer mortality. *Circulation* 2008;117:1658–67.
6 McEniery CM, Spratt M, Munnery M, *et al*. An analysis of prospective risk factors for aortic stiffness in men: 20-year follow-up from the Caerphilly prospective study. *Hypertension* 2010;56:36–43.
7 Johansen NB, Vistisen D, Brunner EJ, *et al*. Determinants of aortic stiffness: 16-year follow-up of the Whitehall II study. *PLoS One* 2012;7:e37165.
8 Wildman RP, Mackey RH, Bostom A, *et al*. Measures of obesity are associated with vascular stiffness in young and older adults. *Hypertension* 2003;42:468–73.
9 Jordan J, Nilsson PM, Kotsis V, *et al*. Joint scientific statement of the European association for the study of obesity and the European Society of hypertension: obesity and early vascular ageing. *J Hypertens* 2015;33:425–34.

10 Nemes A, Geleijnse ML, Forster T, *et al*. Echocardiographic evaluation and clinical implications of aortic stiffness and coronary flow reserve and their relation. *Clin Cardiol* 2008;31:304–9.
11 Cavalcante JL, Lima JAC, Redheuil A, *et al*. Aortic stiffness: current understanding and future directions. *J Am Coll Cardiol* 2011;57:1511–22.
12 Ben-Shlomo Y, Spears M, Boustred C, *et al*. Aortic pulse wave velocity improves cardiovascular event prediction: an individual participant meta-analysis of prospective observational data from 17,635 subjects. *J Am Coll Cardiol* 2014;63:636–46.
13 Townsend RR, Wilkinson IB, Schiffrin EL, *et al*. Recommendations for improving and standardizing vascular research on arterial stiffness: a scientific statement from the American heart association. *Hypertension* 2015;66:698–722.
14 Alsabrook GD, Goodman HR, Alexander JW. Gastric bypass for morbidly obese patients with established cardiac disease. *Obes Surg* 2006;16:1272–7.
15 Ozeki Y, Masaki T, Yoshida Y, *et al*. Relationships between computed tomography-assessed density, abdominal fat volume, and glucose metabolism after sleeve gastrectomy in Japanese patients with obesity. *Endocr J* 2019;66:605–13.
16 Christou NV, Sampalis JS, Liberman M, *et al*. Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. *Ann Surg* 2004;240:416–24.
17 Moher D, Schulz KF, Simera I, *et al*. Guidance for developers of health research reporting guidelines. *PLoS Med* 2010;7:e1000217.
18 Quiñones MA, Otto CM, Stoddard M, *et al*. Recommendations for quantification of Doppler echocardiography: a report from the Doppler quantification Task force of the nomenclature and standards Committee of the American Society of echocardiography. *J Am Soc Echocardiogr* 2002;15:167–84.
19 Seyfeli E, Duru M, Saglam H, *et al*. Association of left ventricular diastolic function abnormalities with aortic elastic properties in asymptomatic patients with type 2 diabetes mellitus. A tissue Doppler echocardiographic study. *Int J Clin Pract* 2008;62:1358–65.
20 Dunn OJ. Multiple comparisons among means. *J Am Stat Assoc* 1961;56:52–64.
21 Redheuil A, Yu W-C, Wu CO, *et al*. Reduced ascending aortic strain and distensibility: earliest manifestations of vascular aging in humans. *Hypertension* 2010;55:319–26.
22 Miyaki A, Maeda S, Yoshizawa M, *et al*. Effect of weight reduction with dietary intervention on arterial distensibility and endothelial function in obese men. *Angiology* 2009;60:351–7.
23 Nemes A, Gavallér H, Csajbók E, *et al*. Obesity is associated with aortic enlargement and increased stiffness: an echocardiographic study. *Int J Cardiovasc Imaging* 2008;24:165–71.
24 Mitchell GF. Arterial stiffness and hypertension. *Hypertension* 2014;64:13–18.
25 Safar ME, Czernichow S, Blacher J. Obesity, arterial stiffness, and cardiovascular risk. *J Am Soc Nephrol* 2006;17:S109–11.
26 Palombo C, Kozakova M. Arterial stiffness, atherosclerosis and cardiovascular risk: pathophysiologic mechanisms and emerging clinical indications. *Vascul Pharmacol* 2016;77:e7
27 Oberoi S, Schoepf UJ, Meyer M. Progression of arterial stiffness and coronary atherosclerosis: longitudinal evaluation by cardiac CT, AJR am. *J Roentgenol* 2013;798:e804.
28 Seals DR, Gates PE. Stiffening our resolve against adult weight gain. *Hypertension* 2005;45:175–7.
29 Jia G, Aroor AR, DeMarco VG, *et al*. Vascular stiffness in insulin resistance and obesity. *Front Physiol* 2015;6:231.
30 Brillante DG, O'Sullivan AJ, Howes LG. Arterial stiffness in insulin resistance: the role of nitric oxide and angiotensin II receptors. *Vasc Health Risk Manag* 2009;5:73–8.
31 Anfossi G, Russo I, Doronzo G, *et al*. Contribution of insulin resistance to vascular dysfunction. *Arch Physiol Biochem* 2009;115:199–217.
32 Zhang Z-W, Guo R-W, Lv J-L, *et al*. MicroRNA-99a inhibits insulin-induced proliferation, migration, dedifferentiation, and rapamycin resistance of vascular smooth muscle cells by inhibiting insulin-like growth factor-1 receptor and mammalian target of rapamycin. *Biochem Biophys Res Commun* 2017;486:414–22.
33 Spencer M, Yao-Borengasser A, Unal R, *et al*. Adipose tissue macrophages in insulin-resistant subjects are associated with collagen VI and fibrosis and demonstrate alternative activation. *Am J Physiol Endocrinol Metab* 2010;299:E1016–27.
34 Fonseca-Alaniz MH, Takada J, Alonso-Vale MIC, *et al*. Adipose tissue as an endocrine organ: from theory to practice. *J Pediatr* 2007;83:S192–203.
35 Peyster E, Chen J, Feldman HI, *et al*. Inflammation and arterial stiffness in chronic kidney disease: findings from the CRIC study. *Am J Hypertens* 2017;30:400–8.
36 du Toit EF, Nabben M, Lochner A. A potential role for angiotensin II in obesity induced cardiac hypertrophy and ischaemic/reperfusion injury. *Basic Res Cardiol* 2005;100:346–54.
37 Ward MR, Pasterkamp G, Yeung AC, *et al*. Arterial remodeling. mechanisms and clinical implications. *Circulation* 2000;102:1186–91.
38 Wildman RP, Mehta V, Thompson T, *et al*. Obesity is associated with larger arterial diameters in Caucasian and African-American young adults. *Diabetes Care* 2004;27:2997–9.
39 Orr JS, Gentile CL, Davy BM, *et al*. Large artery stiffening with weight gain in humans: role of visceral fat accumulation. *Hypertension* 2008;51:1519–24.
40 Resnick LM, Militianu D, Cunnings AJ, *et al*. Direct magnetic resonance determination of aortic distensibility in essential hypertension: relation to age, abdominal visceral fat, and in situ intracellular free magnesium. *Hypertension* 1997;30:654–9.
41 Sutton-Tyrrell K, Newman A, Simonsick EM, *et al*. Aortic stiffness is associated with visceral adiposity in older adults enrolled in the study of health, aging, and body composition. *Hypertension* 2001;38:429–33.
42 Wildman RP, Farhat GN, Patel AS, *et al*. Weight change is associated with change in arterial stiffness among healthy young adults. *Hypertension* 2005;45:187–92.
43 Mahfouz RA, Abdulmoneim A, Abduo M, *et al*. The relation of aortic stiffness and in-stent restenosis in patients undergoing percutaneous coronary stenting. *Echocardiography* 2013;30:582–7.
44 Benetos A, Adamopoulos C, Bureau J-M, *et al*. Determinants of accelerated progression of arterial stiffness in normotensive subjects and in treated hypertensive subjects over a 6-year period. *Circulation* 2002;105:1202–7.
45 Seeland U, Demuth I, Regitz-Zagrosek V, *et al*. Sex differences in arterial wave reflection and the role of exogenous and endogenous sex hormones: results of the Berlin aging study II. *J Hypertens* 2020;38:1040–6.
46 Iancu ME, Copăescu C, Şerban M, *et al*. Favorable changes in arterial elasticity, left ventricular mass, and diastolic function after significant weight loss following laparoscopic sleeve gastrectomy in obese individuals. *Obes Surg* 2014;24:364–70.
47 Rider OJ, Francis JM, Ali MK, *et al*. Beneficial cardiovascular effects of bariatric surgical and dietary weight loss in obesity. *J Am Coll Cardiol* 2009;54:718–26.
48 Owan T, Avelar E, Morley K, *et al*. Favorable changes in cardiac geometry and function following gastric bypass surgery: 2-year follow-up in the Utah obesity study. *J Am Coll Cardiol* 2011;57:732–9.
49 Kemaloğlu Öz T, Ünal Dayı Şennur, Seyit H, *et al*. The effects of weight loss after sleeve gastrectomy on left ventricular systolic function in men versus women. *J Clin Ultrasound* 2016;44:492–9.
50 Behrendt D, Ganz P. Endothelial function. from vascular biology to clinical applications. *Am J Cardiol* 2002;90:40–8.
51 Nijhuis J, van Dielen FMH, Fouraschen SMG, *et al*. Endothelial activation markers and their key regulators after restrictive bariatric surgery. *Obesity* 2007;15:1395–9.
52 Reilly MP, Lehrke M, Wolfe ML, *et al*. Resistin is an inflammatory marker of atherosclerosis in humans. *Circulation* 2005;111:932–9.
53 Elias MF, Crichton GE, Dearborn PJ, *et al*. Associations between type 2 diabetes mellitus and arterial stiffness: a prospective analysis based on the Maine-Syracuse study. *Pulse* 2018;5:88–98.
54 Iancu M, Copăescu C, Şerban M, *et al*. Laparoscopic sleeve gastrectomy reduces the predicted coronary heart disease risk and the vascular age in obese subjects. *Chirurgia* 2013;108:659–65.