ORIGINAL ARTICLE

The impact of weight loss after laparoscopic sleeve gastrectomy on early markers of atherosclerotic vascular disease: a prospective study

Bedri C. Kaya[1], Hasan Elkan[2]

1 Clinic of Cardiology, Mehmet Akif Inan Training and Research Hospital, Sanliurfa, Turkey
2 Clinic of General Surgery, Sanliurfa Training and Research Hospital, Sanliurfa, Turkey

KEY WORDS
ankle-brachial index, aortic propagation velocity, carotid intima media thickness, epicardial fat thickness, sleeve gastrectomy

EDITORIALS
by Casella and Castagneto-Gissey, see p. 655 and Ministrini, Mondovecchio, Lupattelli, see p. 657

ABSTRACT

BACKGROUND Considering the emerging role of aortic propagation velocity (APV) in determining the burden of the coronary artery disease, we hypothesized that laparoscopic sleeve gastrectomy (LSG) could improve APV in morbidly obese patients.

AIMS The aim of this study was to investigate the impact of LSG on surrogate markers of atherosclerotic vascular disease such as APV, carotid intima-media thickness (CIMT), epicardial fat thickness (EFT), and ankle-brachial index (ABI) in patients with morbid obesity.

METHODS We prospectively enrolled 71 patients who were scheduled for LSG for standard indications between December 2018 and June 2019 with accordance to the international guidelines. All patients underwent transthoracic echocardiography and carotid ultrasonography. Differences in the variables measured (Δ) were calculated by subtracting 6-month follow-up results from the baseline results.

RESULTS Laparoscopic sleeve gastrectomy led to a significant reduction in body weight, and at 6-month follow-up, there was a reduction in systolic and diastolic blood pressure as well as in levels of triglycerides and low-density lipoprotein cholesterol. Moreover, a reduction in EFT and CIMT as well as an increase in ABI and APV were noted at 6-month follow-up compared with the baseline measurements. The change in APV at 6-month follow-up was correlated with systolic blood pressure, diastolic blood pressure, EFT, ABI, and CIMT.

CONCLUSIONS LSG leads to a significant improvement in BMI as well as CIMT, EFT, ABI, and APV, which are the surrogate markers of atherosclerotic vascular disease, in morbidly obese patients at 6-month follow-up after the procedure. The improvement in APV is correlated with the improvement in BMI, CIMT, EFT, and ABI.

INTRODUCTION Coronary artery disease (CAD) is by far the most common cause of heart failure and mortality in developed countries. Age, family history of CAD, diabetes, smoking, high blood pressure, hyperlipidemia, and obesity are well-established risk factors of CAD.[1,2] Obesity has been shown to lead to CAD due to high blood pressure and diabetes. It is also an independent risk factor for increased prevalence of CAD.[3-5] A recent meta-analysis demonstrated that obesity is associated with an increased all-cause mortality rate, with an odds ratio of 1.29 for those with a body mass index (BMI) of 35 or higher, but no such relation was observed in patients with BMI between 30 and 35.[6] A 10 kg increment in body weight has been shown to increase the risk of CAD by 12%.[7] Therefore, weight loss is crucial not only in the primary prevention of CAD in high-risk patients, but also in the secondary prevention in those with established CAD. Moreover, patients with CAD who intentionally lost weight have a significantly lower risk of adverse clinical outcomes.[8]

Given the direct and indirect impact of obesity on CAD, several approaches, including bariatric surgery, have been developed to facilitate weight loss in obese patients. Sustained weight loss achieved with Roux-en-Y gastric bypass has

Correspondence to:
Bedri C. Kaya, MD, Clinic of Cardiology, Mehmet Akif Inan Training and Research Hospital, Esentepe Mah, Ertugrul Cad. 132-A, 63 040 Sanliurfa, Turkey, phone: +90 5055701303, email: bckaya23@gmail.com
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WHAT'S NEW?

Given the impact of obesity on the development of coronary artery disease, several approaches, including bariatric surgery, have been developed to facilitate weight loss in obese patients. Despite the improvement in weight loss after bariatric surgery, the impact of this procedure on markers of the coronary artery disease burden has not been studied yet. We demonstrated that in morbidly obese patients, laparoscopic sleeve gastrectomy not only reduced total body weight but also led to a significant improvement in surrogate markers of atherosclerotic burden, including aortic propagation velocity, carotid intima-media thickness, ankle-brachial index, and epicardial fat thickness at 6-month follow-up after the procedure.

been shown to be associated with less coronary calcification independent of changes in low-density lipoprotein (LDL) cholesterol.[9] Bariatric surgery has also been reported to improve peripheral endothelial function and coronary microvascular function at 4-year follow-up in morbidly obese patients.[10] Moreover, several reports indicated that bariatric surgery reduces the incidence of myocardial infarction as well as the need for coronary revascularization in morbidly obese patients.[11]

Laparoscopic sleeve gastrectomy (LSG), a relatively new technique of bariatric surgery, has been shown to lead to a weight loss of more than 58.4% of initial body weight at 5-year follow-up.[12] Despite the improvement achieved in weight loss with LSG, the impact of this procedure on markers of the CAD burden has not been studied yet.

Given the emerging role of the aortic propagation velocity (APV) in the determination of the CAD burden, we hypothesized that LSG could improve APV in morbidly obese patients.[13] The present study aimed to investigate the impact of LSG on surrogate markers of atherosclerotic vascular disease such as APV, carotid intima-media thickness (CIMT), epicardial fat thickness (EFT), and ankle-brachial index (ABI) in patients with morbid obesity.

METHODS **Patient selection** The present prospective cohort study was conducted in patients with morbid obesity who were scheduled for LSG at Mehmet Akif Inan Training and Research Hospital in Sanliurfa, Turkey between December 2018 and June 2019. Indications for bariatric surgery were based on criteria from international guidelines on the diagnosis and treatment of obesity, including BMI higher than 40 kg/m^2 or BMI higher than 35 kg/m^2 in the presence of comorbidities in which surgically induced weight loss is expected to improve the patient condition (type 2diabetes, cardiorespiratory disease, severe joint disorders, and severe psychological problems related to obesity).[14] Patients were scheduled for LSG when nonsurgical approaches (diet, exercise programs, and pharmacological therapy) failed to decrease weight. Patients aged between 18 and 65 years were enrolled if they met the aforementioned criteria for bariatric surgery.

Exclusion criteria were as follows: known atherosclerotic vascular disease, history of statin use in the preceding year, previous revascularization, uncontrolled diabetes (HbA_{1c} >7%), a mild or more severe kidney or liver disease, a mild or more severe degree valvular dysfunction, reduced left ventricular ejection fraction (<50%), permanent pacemaker, severe mental disorders, and binge eating disorder. Whether the patients complied with the exclusion criteria or not was evaluated based on patient self-reports. Then, patient records were assessed with regard to these criteria and the statements obtained from the patients were confirmed. Those with low quality images on echocardiography were also not included in the study. Out of those undergoing LSG, a total of 71 patients met the inclusion criteria and completed the follow-up period.

All patients had systolic and diastolic blood pressure measured. A standard aneroid sphygmomanometer was used on the right arm of a seated patient. Two separate blood pressure measurements were performed and the mean value was recorded. Blood samples were collected in the fasting state to analyze the levels of blood glucose, total cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides. For each leg, ABI was calculated by taking the higher pressure of the 2 arteries at the ankle and dividing by the brachial arterial systolic pressure. The higher of the 2 brachial systolic pressure measurements was used in the calculation of the ABI. Blood sampling and blood pressure measurements were performed before and 6 months after LSG. Differences (Δ) were calculated by subtracting 6-month results from the baseline results.

Aortic flow propagation velocity and epicardial fat thickness All patients underwent a physical examination, electrocardiography, and transthoracic echocardiography before and 6 months after LSG. Conventional echocardiographic parameters, APV, and EFT were measured in the left lateral decubitus position using the same ultrasound system (VividS5, GE Vingmed Ultrasound, Horten, Norway), interfaced with a 2.5 to 3.5 MHz phased array probe, by 2 sonographers who were not involved in clinical follow-up and were blinded to the clinical data at all times. The biplane modified Simpson method was used in the measurement of ejection fraction. Color M-mode Doppler recordings from the suprasternal view were used for the evaluation of APV. For this purpose, the cursor was placed parallel to the direction of the flow in the descending thoracic aorta, the Nyquist limit was set at 30 to 50 cm/s (sweep rate of 200 mm/s), and an M-mode

spatiotemporal velocity map with the shape of a flame was displayed. In patients with an unclear slope of the flame, baseline shifting was used to change the aliasing velocity until a clear appearance of the isovelocity slope. APV was calculated by dividing the distance between points corresponding to the beginning and end of the propagation slope with the duration between corresponding time points in cm/s.[15] Epicardial fat thickness was measured at end systole from the 2-dimensional long-axis view on the right ventricular free wall perpendicular to the aortic annulus. Epicardial fat thickness ranged between 1 and 23 mm. The median EFT was 7 mm for men and 6.5 mm for women undergoing transthoracic echocardiography for standard clinical indications.[16] All echocardiographic measurements were performed on 3 consecutive cardiac cycles, and average values were recorded. Measurements of APV and EFT were performed before and 6 months after LSG.

Intima-media thickness of the carotid artery Carotid intima-media thickness is utilized to estimate the early atherosclerosis in those with and without risk factors for atherosclerotic vascular disease.[17] CIMT values of more than 0.9 mm are considered abnormal. In this study, all CIMT measurements were performed by the same sonographer using a high-frequency (7.0–13.0 MHz) linear ultrasound scanning probe (Siemens Healthineers, Erlangen Germany) in the supine position with the neck extended and the head tilted away from the side being examined. Multiple longitudinal planes were imaged to obtain the clearest resolution of the intima-media thickness. The left and the right common carotid arteries were imaged proximal to the bulb. The mean IMT was obtained by manually tracing the intima-media in the far wall of the artery. CIMT measurements were performed at end-diastole on 3 consecutive cardiac cycles, and average values were recorded. CIMT measurement was performed before and 6 months after LSG. The intraobserver variability for CIMT measurements was obtained from 30 random patients and was 2%.

Primary outcome The change in BMI, APV, CIMT, EFT, ABI, and cholesterol (HDL, LDL) and triglyceride levels obtained by comparing baseline (preoperative) and 6-month follow-up values was the primary outcome measure of the study. The relation between weight loss and changes in APV, CIMT, EFT, ABI was the secondary outcome measure.

Statistical analysis All analyses were performed on SPSS v21 (SPSS Inc., Chicago, Illinois, United States). The Shapiro–Wilk test was used to determine whether variables were distributed normally or not. The homogeneity of variances was assessed with the Levene test. Data are presented as mean (SD) and frequency (percentage) for categorical variables. A paired-samples *t* test was used to compare changes in variables from baseline to 6-month follow-up. Pearson correlation coefficients were calculated for the assessment of the relationships between the change in BMI and selected echocardiography parameters. A *P* value of less 0.05 was considered statistically significant.

Ethics The study was approved by the institutional review board and was conducted in accordance with the Helsinki declaration. Informed consent was obtained from all individual participants included in the study.

TABLE 1 **Demographic features, baseline laboratory measurements, and clinical characteristics of the study population**

Variable	Value (n = 71)
Age, y	37.6 (11.2)
Male sex, n (%)	48 (67.6)
Hypertension, n (%)	23 (32.3)
Diabetes, n (%)	26 (36.6)
Smoking, n (%)	18 (25.3)
Leukocyte count, × 10^3/mm^3	8.5 (2.3)
AST, U/l	24.2 (9.2)
ALT, U/l	27.8 (10.6)
Creatinine, mg/dl	0.71 (0.16)

Data are presented as mean (SD) unless otherwise indicated.

SI conversion factors: to convert AST and ALT to nmol/(s•l), multiply by 16.667; creatinine to mmol/l, by 0.08845.

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase

TABLE 2 The comparison of the clinical, laboratory, and echocardiographic parameters at baseline and 6-month follow-up

Variable	Baseline	Follow-up	*P* value
Weight, kg	129 (31)	100 (21)	<0.001
BMI, kg/m^2	47.7 (6.5)	36.9 (5.4)	<0.001
FPG, mg/dl	104.6 (23.2)	98.6 (28.9)	0.4
MPV, fl	7.4 (1.5)	7.9 (1.3)	0.07
NLR	1.6 (0.8)	1.7 (0.6)	0.17
HDL-C, mg/dl	40.8 (10.5)	41.7 (11.3)	0.38
TG, mg/dl	226 (72)	164 (70)	<0.001
LDL-C, mg/dl	108 (27)	91 (16)	0.001
EF, %	62.3 (2.4)	62.6 (2.3)	0.36
SBP, mm Hg	136 (15)	122 (14)	<0.001
DBP, mm Hg	78 (9)	73 (6)	0.004
HR, bpm	82 (15)	73 (10)	<0.001
EFT, cm	0.65 (0.14)	0.58 (0.13)	<0.001
ABI	0.85 (0.08)	0.95 (0.05)	<0.001
CIMT, mm	1.07 (0.06)	0.99 (0.13)	<0.001
APV, cm/s	44.2 (4.8)	50.1 (6.6)	<0.001

Data are presented as mean (SD).

Abbreviations: ABI, ankle-brachial index; APV, aortic propagation velocity; BMI, body mass index; CIMT, carotid intima-media thickness; DBP, diastolic blood pressure; EF, ejection fraction; EFT, epicardial fat thickness; FPG, fasting plasma glucose; HDL-C, high--density lipoprotein cholesterol; HR, heart rate; LDL-C, low-density lipoprotein cholesterol; NLR, neutrophil to lymphocyte ratio; MPV, mean platelet volume; SBP, systolic blood pressure; sPAP, systolic pulmonary artery pressure; TG, triglyceride

TABLE 3 Correlation analysis between markers of atherosclerotic vascular disease and various clinical variables

Variable	ΔEFT		ΔABI		ΔCIMT		ΔAPV	
	r	*P* value	*r*	*P* value	*r*	*P* value	*r*	*P* value
ΔBMI	0.338	0.011	0.046	0.74	0.481	0.001	−0.370	0.005
ΔFPG	0.255	0.058	−0.082	0.55	0.133	0.33	−0.166	0.22
ΔHDL-C	0.231	0.086	0.124	0.36	0.028	0.84	−0.189	0.16
ΔTG	0.096	0.48	−0.077	0.57	0.026	0.85	0.110	0.42
ΔLDL-C	0.166	0.220	0.107	0.43	0.239	0.076	−0.050	0.71
ΔMPV	0.132	0.39	−0.068	0.66	−0.145	0.34	−0.151	0.32
ΔNLR	−0.005	0.97	0.047	0.73	0.032	0.93	0.030	0.83
ΔSBP	−0.034	0.80	−0.443	0.001	0.339	0.011	−0.297	0.026
ΔDBP	0.099	0.47	−0.136	0.32	0.262	0.053	−0.410	0.002
ΔEFT	–	–	0.212	0.12	0.478	<0.001	−0.369	0.005
ΔABI	0.212	0.12	–	–	0.183	0.18	0.365	0.006
ΔCIMT	0.478	<0.001	0.183	0.18	–	–	−0.356	0.007
ΔAPV	−0.369	0.005	0.365	0.006	−0.356	0.007	–	–

Differences (Δ) were calculated by subtracting 6-month results from the baseline results.

Abbreviations: see TABLE 2

TABLE 4 Linear regression analysis demonstrating the predictors of changes in markers of atherosclerotic vascular disease

Variable	APV			CIMT			ABI			EFT		
	β coefficient	95% CI	*P* value	β coefficient	95% CI	*P* value	β coefficient	95% CI	*P* value	β coefficient	95% CI	*P* value
ΔSBP	–0.254	–0.216 to 0.038	0.17	0.010	–0.003 to 0.004	0.95	–0.272	–0.004 to 0.001	0.14	–0.272	–0.004 to 0.001	0.14
ΔDBP	0.149	–0.126 to 0.299	0.42	–0.031	–0.007 to 0.005	0.86	0.068	–0.003 to 0.005	0.71	0.068	–0.003 to 0.005	0.71
ΔBMI	0.338	0.101–1.706	0.028	0.447	0.012–0.052	0.002	0.092	–0.009 to 0.017	0.53	0.092	–0.009 to 0.017	0.53
ΔLDL-C	–0.150	–0.077 to 0.023	0.29	0.156	–0.001 to 0.002	0.24	0.165	0.000–0.001	0.25	0.165	0.000–0.001	0.25
ΔTG	0.163	–0.007 to 0.026	0.26	0.098	0.000–0.001	0.47	–0.067	0.000–0.000	0.64	–0.067	0.000–0.000	0.64
ΔHR	–0.031	–0.106 to 0.084	0.82	0.076	–0.002 to 0.004	0.55	–0.138	–0.003 to 0.001	0.32	–0.138	–0.003 to 0.001	0.32

Differences (Δ) were calculated by subtracting 6-month results from the baseline results.

Abbreviations: see TABLE 2

RESULTS We recruited 71 patients who underwent LSG (mean [SD] age, 37.6 [11.2] years; male sex, 67.6%). A total of 23 patients were diagnosed with hypertension, out of which 12 have been using ramipril and 11, candesartan. Demographic variables, baseline laboratory measurements, and clinical characteristics of the study population are presented in TABLE 1.

The comparisons of clinical, laboratory, and echocardiographic parameters between baseline and 6-month follow-up are shown in TABLE 2. LSG led to a significant reduction in body weight (P <0.001) and BMI (P <0.001) at 6-month follow-up. There was a significant decrease in systolic and diastolic blood pressure, heart rate, and in triglyceride and LDL cholesterol levels. Ejection fraction at 6-month follow-up was similar to the preoperative value. However, significant reductions in EFT (P <0.001) and CIMT (P <0.001) as well as significant increases in AIB (P <0.001) and APV (P <0.001) were noted at 6-month follow-up compared with the baseline measurements.

Correlation between the change in EFT, CIMT, ABI, and APV with the change in selected clinical and laboratory parameters during the follow-up (Δ) are presented in TABLE 3. The change in APV was significantly correlated with systolic blood pressure (r = –0.297, P = 0.026), diastolic blood pressure (r = –0.410, P = 0.002), EFT (r = –0.369, P = 0.005), ABI (r = 0.365, P = 0.006), and CIMT (r = –0.356, P = 0.007).

The multiple linear regression analysis demonstrated that ΔBMI was a significant predictor for both ΔAPV (β coefficient, 0.338; 95% CI, 0.101–1.706; P = 0.028) and ΔCIMT (β coefficient, 0.447; 95% CI, 0.012–0.052; P = 0.002) (TABLE 4).

DISCUSSION The present study clearly demonstrated that in morbidly obese patients, LSG not only reduces total body weight and BMI but also leads to a significant improvement in surrogate markers of atherosclerotic burden, including APV, CIMT, ABI, and EFT at 6-month follow-up. In addition, a significant reduction was observed in systolic and diastolic blood pressure as well as in levels of triglycerides and LDL-cholesterol at 6-month follow-up. Moreover, the improvements in APV and CIMT are significantly correlated with the reduction in BMI and blood pressure. As shown in the linear regression analysis, BMI is a significant predictor for both ΔAPV and ΔCIMT.

Obesity causes a major burden on the healthcare system due to its association with numerous complications, among which type 2 diabetes and cardiovascular disease (CVD) are the most important ones. Cardiovascular disease is the major contributor to the reduced life expectancy in obese patients. Chronic inflammation, insulin resistance, and prothrombotic environment in obese patients increase the risk

for atherothrombotic events. Although obesity has been established as an independent risk factor for atherosclerotic CVD, clustering of cardiovascular risk factors in obese subjects such as type 2 diabetes, hypertension, and dyslipidemia may also give rise to the development of atherosclerotic disease.[18]

The loss of excess weight should therefore theoretically lead to a reduction in the prevalence of atherosclerotic CVD. This consideration was confirmed by the decline in CVD mortality and death from diabetes as a consequence of the population-wide weight loss of an average of 4 to 5 kg during the Cuban economic crisis of the early- to mid-1990s.[19] Several diets and lifestyle interventions, which lead to a weight loss of between 3% and 10% of the total body weight, have also been associated with improvements in cardiovascular risk factors, including the lipid profile, blood pressure, suppresion of inflammation, and improvement in insulin resistance.[20] Favorable changes in lipid profile and insulin resistance were also reported in studies investigating the effects of agents such as orlistat, naltrexone / bupropion combination, lorcaserin, phentermine / topiramate combination, and glucagon-like peptide-1 receptor agonists on weight loss.[21-24] However, despite the promising changes in the lipid profile, blood pressure, and inflammation obtained with some of the agents, data concerning the improvement in CVD outcomes were lacking.

Among all strategies assisting patients to lose weight, bariatric surgery provides more significant and sustainable weight loss, particularly for morbidly obese individuals, compared with nonsurgical treatment approaches. A meta-analysis of 11 studies with 796 obese individuals and a follow-up period of 6 months or longer showed that bariatric surgery leads to an additional weight loss of 26 kg compared with the nonsurgical treatment while blood pressure and cholesterol concentrations were not significantly different.[25] The prospective controlled SOS (Swedish Obese Study) showed that weight loss achieved with bariatric surgery was preserved even after 20 years of the surgery.[26] Bariatric surgery has been shown to improve CVD risk factors with reduced rates of hypertension and dyslipidemia and corresponding improvement in CVD risk scores.[27-29] Blood concentrations of the inflammatory markers, including C-reactive protein, interleukin 6, and adipokines, have also been reported to decrease following bariatric surgery.[30] The SOS study demonstrated that the number of cardiovascular deaths and cardiovascular events of patients allocated to bariatric surgery was significantly lower than that of subjects receiving nonsurgical treatment for obesity after a mean follow-up period of 14.7 years.[31] A systematic review and meta-analysis of 14 studies, including 29 208 patients who underwent bariatric surgery, reported valuable data regarding the relation between weight loss and cardiovascular outcomes.[32] Following a follow-up ranging between 2 to 14.7 years, the overall mortality decreased by 50% in addition to reductions in the incidence of myocardial infarction and stroke. However, more current data are required to address the impact of bariatric surgery on cardiovascular outcomes. Furthermore, there is still a debate concerning the most efficacious surgical procedure for obese individuals.

Laparoscopic sleeve gastrectomy has emerged as a safe and valid technique in the surgical management of patients with excess body weight as a stand-alone procedure due to its relative surgical ease and low risk of complications.[33,34] Previous reports have indicated that LSG can provide an excess weight loss of up to 82.9%.[35] LSG provides a significant weight loss not only by reducing the gastric capacity but also by increasing GLP-1 hormone and decreasing ghrelin levels, which is produced in the gastric fundus and shown to increase appetite.[36,37] Recently, Altin et al[38] published results of their study in 106 patients with morbid obesity who underwent LSG for standard indications. The authors have reported that LSG was associated with a significant reduction in EFT and CIMT in addition to the improvements in BMI, insulin resistance, blood lipids, and systolic and diastolic blood pressure.

In this study, we hypothesized that the weight loss achieved with LSG would provide improvement in APV, which has been indicated as a novel surrogate of the CAD burden. Our findings demonstrated that LSG was associated with improvements not only in CIMT, EFT, and ABI, which have been shown to indicate atherosclerotic burden, but also with an increase in APV. To the best of our knowledge, the present study is the first to demonstrate an improvement in APV and ABI following LSG. Favorable changes in EFT and CIMT observed in our study population is consistent with data provided by Altin et al.[38] Our findings also demonstrated that APV exerts a significant correlation with CIMT, EFT, ABI, and with the reduction in BMI. From this point of view, our findings empower the limited evidence published by Altin et al[38] which shows improvement in surrogate markers of atherosclerotic vascular disease following LSG. Moreover, given the significant correlation of the APV with CIMT, EFT, and ABI, our results also indicate that APV can be used as a simple and readily available tool in the monitorization of the CAD burden in patients undergoing sleeve gastrectomy.

There are some limitations concerning the results of the present study. First, we could not provide data regarding pre- and postoperative insulin resistance. Second, echocardiographic image quality was poor in some patients. This might have influenced EFT and APV measurements. Third, blood pressure measurements were performed during office visits; thus, they

may not reflect the blood pressure profile of the patients. Fourth, 6-month follow-up after LSG may not be appropriate for evaluating the changes in CIMT. However, previous evidence in patients with diabetes indicated that CIMT may respond to optimization of diabetes treatment even in 2 weeks.[39] Finally, EFT was measured from the free wall of the right ventricle. These results therefore need to be interpreted with caution.

Conclusion In conclusion, LSG leads to significant improvements in BMI, and CIMT, EFT, ABI, and APV, which are the surrogate markers of atherosclerotic vascular disease, in morbidly obese patients at 6-month follow-up after the procedure. The improvement in APV is correlated with the improvement in BMI, CIMT, EFT, and ABI.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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