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Authors: Bedri Caner Kaya, Hasan Elkan

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Impact of weight loss achieved with laparoscopic sleeve gastrectomy on early markers of atherosclerotic vascular disease: a prospective study

Short title: Early markers of atherosclerotic vascular disease

Bedri Caner Kaya¹, Hasan Elkan²

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

Corresponding Author: Bedri Caner Kaya

: +905055701303

: bckaya23@gmail.com

: Clinic of Cardiology, Mehmet Akif Inan Training And Research Hospital, Esentepe Mah, Ertugrul Cad. 132-A, 63040 Sanliurfa Merkez/Haliliye/Sanliurfa, Turkey

Conflict of interest

None declared
**What’s new?**

Given the effect of obesity in the development of coronary artery disease, several approaches, including bariatric surgery, have been developed to facilitate weight loss in these patients. Despite the improvement achieved in weight loss with bariatric surgery, the impact of this procedure on markers of the coronary artery disease burden has not been studied yet.

The present study clearly demonstrates that in morbidly obese subjects laparoscopic sleeve gastrectomy not only reduces total body weight but also leads 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 postoperative 6 months. In addition, a significant reduction occurred in systolic and diastolic blood pressures, triglyceride, and LDL-cholesterol levels from baseline to the postoperative 6 months.
ABSTRACT

**Background:** Considering the emerging role of the aortic propagation velocity (APV) in the determination of the coronary artery disease (CAD) burden, we hypothesized that laparoscopic sleeve gastrectomy (LSG) could provide improvement in APV in morbidly obese subjects.

**Aims:** This study purposed to investigate the impact of LSG on surrogate markers of the atherosclerotic vascular disease such as APV, carotid intima-media thickness (CIMT), epicardial fat thickness (EFT) and ankle-brachial index (ABI) in subjects with morbid obesity.

**Methods:** Seventy-one subjects who were scheduled for LSG for standard indications stated by the international guidelines were prospectively enrolled in the study. All subjects underwent transthoracic echocardiography, carotid ultrasonography. Delta (Δ) values were obtained by subtracting sixth-month values from the baseline values.

**Results:** LSG led to a significant reduction in body weight and at postoperative 6 months. There was a significant reduction in systolic and diastolic blood pressure and in triglyceride and LDL cholesterol levels. Moreover, significant reductions in EFT, and CIMT and significant increases in ABI and APV were noted at postoperative six months compared to the baseline measurements. The change in APV was significantly correlated with systolic blood pressure, diastolic blood, EFT, ABI and CIMT.

**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 subjects at postoperative 6 months. The improvement in APV is correlated with the improvement in BMI, CIMT, EFT, and ABI.

**Keywords:** Ankle-brachial index; Aortic propagation velocity; Carotid intima media thickness; Epicardial fat thickness; Sleeve gastrectomy
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 for the development of CAD [1,2]. Obesity has been shown to lead to the development of the CAD by its close relationship with high blood pressure, and diabetes, or by directly increasing the prevalence of CAD as an independent risk factor [3-5]. A recent meta-analysis demonstrated that obesity is associated with increased all-cause mortality rates, with an odds ratio of 1.29 for those with a body mass index (BMI) of ≥ 35, but no such relation was observed for subjects with a BMI between 30 and 35 [6]. A 10 kg increment in body weight has been shown to increase the risk of the CAD by 12% [7]. Therefore, weight loss is critical not only in primary prevention of the subjects with high risk for CAD but also in secondary prevention of patients with established CAD. In subjects with CAD, intentional weight loss has been shown to be associated with a significantly lower risk of adverse clinical outcomes [8].

Given the direct and indirect effect of obesity in the development of CAD, several approaches, including bariatric surgery, have been developed to facilitate weight loss in these patients. Sustained weight loss achieved with Roux-en-Y gastric bypass surgery has been shown to be associated with less coronary calcification independent of changes in LDL-cholesterol [9]. Bariatric surgery has also been reported to improve peripheral endothelial function and coronary microvascular function at a 4-year follow-up of the morbidly obese subjects [10]. Moreover, several reports indicate 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 for bariatric surgery, has been shown to lead to a weight loss of more than 58.4% of initial body weight in 5-years
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 provide improvement in APV in morbidly obese subjects [13]. Within this context, the present study aimed to investigate the impact of LSG on surrogate markers of the atherosclerotic vascular disease such as APV, carotid intima-media thickness (CIMT), epicardial fat thickness (EFT) and ankle-brachial index (ABI) in subjects with morbid obesity.

METHODS

Patient selection

The present prospective cohort study was conducted on patients with morbid obesity who were scheduled for LSG in Sanliurfa Mehmet Akif Inan Research and Training Hospital between December 2018 and June 2019. Indications for bariatric surgery were based on the criteria stated in international obesity diagnosis and treatment guidelines; including body mass index (BMI) > 40 kg/m2 or BMI > 35 kg/m2 in the presence of co-morbidities in which surgically induced weight loss is expected to improve the disorder (Type 2 diabetes, cardio-respiratory disease, severe joint disorders, severe psychological problems related to obesity) [14]. Subjects were scheduled for LSG as long as the non-surgical approaches (diet, exercise programs, and pharmacological therapy) were failed to improve weight loss.

Subjects 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 for last one year, previous revascularization, uncontrolled diabetes (HbA1c > 7%), more than mild kidney or liver disease, more than mild degree valvular dysfunction, reduced 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 the patient's self-reports. Then, patients' records were examined in terms of these criteria and the statements were confirmed. Those with low image quality were also not included in the study. Among those undergoing LSG, the number of the subjects meeting the inclusion criteria and completing the follow-up period was 71.

All subjects underwent measurement of the systolic and diastolic blood pressure using a standard aneroid sphygmomanometer on the right arm of the seated subject. Two separate measurements were performed, and the mean value of the two blood pressure measurements was recorded. Fasting blood samples were collected for analyses of blood glucose, total cholesterol, HDL-cholesterol, and triglycerides. An ABI, determined by taking the higher pressure of the two arteries at the ankle, divided by the brachial arterial systolic pressure, was calculated for each leg. The higher of the two brachial systolic pressure measurements was used in the calculation of the ABI. Blood sampling and blood pressure measurements were performed before and six months after the LSG.

**Aortic Flow Propagation Velocity and Epicardial fat thickness**

All subjects underwent a physical examination, electrocardiography, and transthoracic echocardiography before and six months after LSG. Conventional echocardiographic parameters and APV, and epicardial fat thickness (EFT) was measured in left lateral decubitus position using the same ultrasound system (VividS5, GE Vingmed Ultrasound, Horten, Norway), interfaced with a 2.5–3.5 MHz phased-array probe, by two sonographers who were not involved in clinical follow-up and were blinded to the clinical data at all times. Biplane-modified Simpson’s method was used in the measurement of the ejection fraction. Color M-mode Doppler recordings from suprasternal view were used for evaluation of the APV. For this purpose, the cursor was placed parallel to the flow of direction in the descending thoracic aorta, Nyquist limit was set at 30–50 cm/s (sweep rate of 200 mm/s), and an M-mode spatio-
temporal velocity map with the shape of a flame is displayed. In subjects 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 a two-dimensional long-axis view on the right ventricular free wall perpendicular to the aortic annulus. Epicardial fat thickness has been shown to range between 1mm and 23 mm. Median EFT has been found 7mm for men and 6.5 mm for women undergoing transthoracic echocardiography for standard clinical indications [16]. All echocardiographic measurements were performed on three consecutive cardiac cycles, and average values were recorded. APV and EFT measurements were performed before and 6 months after the LSG.

The Carotid artery intima-media thickness

Carotid intima-media thickness is utilized to estimate the early atherosclerosis in subjects 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 carotid artery intima-media thickness (CIMT) measurements were performed by the same sonographer using a high-frequency (7.0–13.0 MHz) linear ultrasound scanning probe (Siemens Helthineers, 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 three consecutive cardiac cycles, and average values were recorded. CIMT measurement was performed before
and six months after the LSG. The intraobserver variability for the CIMT measurements were obtained from random 30 patients and was found as 2%.

**Primary outcome**

The change in BMI, AFPV, CIMT, EFT, ABI, and cholesterol (HDL, LDL) and triglyceride levels from baseline (preoperative) to the postoperative sixth month following the LSG was the primary outcome measure of this study. The relation of the weight loss with the changes AFPV, CIMT, EFT, ABI was the secondary outcome measure.

**Statistical analysis**

All analyses were performed on SPSS v21 (SPSS Inc., Chicago, IL, USA). 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 (standard deviation) and frequency (percentage) for categorical variables. Paired samples t-test was used in the comparison of the change in the variables from baseline to the postoperative six months. Pearson Correlation Coefficients were calculated for the assessment of the relationships between the change in BMI and selected echocardiography parameters. P < 0.05 values accepted as statistically significant results.

**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.

**RESULTS**

Seventy-one patients who underwent LSG were recruited (mean age 37.6 [11.2] years, 67.6% male). Of the 23 patients diagnosed with hypertension, 12 have been using ramipril and 11
have been using candesartan. Demographic features, baseline laboratory measurements, and clinical characteristics of the study population are presented in Table 1.

Table 2 shows the comparison of the clinical, laboratory, and echocardiographic parameters from baseline to the postoperative 6 months. LSG led to a significant reduction in body weight ($P = 0.0001$) and BMI ($P < 0.001$) at postoperative 6 months. There was a significant reduction in systolic and diastolic blood pressure, heart rate and in triglyceride and LDL cholesterol levels. Ejection fraction at postoperative 6th months was similar to the preoperative value. However, significant reductions in EFT ($P < 0.001$), and CIMT ($P < 0.001$) and significant increases in AIB ($P < 0.001$) and APV ($P < 0.001$) were noted at postoperative six months compared to the baseline measurements.

Table 3 demonstrates the level of correlation between the change in EFT, CIMT, ABI, and APV with the change in selected clinical and laboratory parameters during the follow-up ($\Delta$). 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 ($r = -0.356, P = 0.007$).

Multiple linear regression analysis revealed that $\Delta$ BMI was a significant predictor for both $\Delta$ APV (Coefficient-$\beta$: 0.338, 95% CI: 0.101-1.706, $P = 0.028$) and $\Delta$ CIMT (Coefficient-$\beta$: 0.447, 95% CI: 0.012-0.052, $P = 0.002$) (Table 4).

DISCUSSION

The present study clearly demonstrates that in morbidly obese subjects 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 postoperative 6 months. In addition, a significant reduction occurred in systolic and diastolic blood pressures, triglyceride, and LDL-cholesterol levels from baseline to the postoperative 6 months.
Moreover, the improvements in APV and CIMT are significantly correlated with the reduction in BMI and blood pressure. As shown in linear regression analysis, BMI is a significant predictor for both Δ APV and Δ CIMT.

Obesity causes a major burden on the healthcare system due to its association with numerous complications, among which type 2 diabetes and cardiovascular disease are the most important ones. Cardiovascular disease is the major contributor to the reduced life expectancy in obese subjects. Chronic inflammatory state, insulin resistance, and pro-thrombotic environment existing in obese subjects increase the risk for atherothrombotic events. Although obesity has been established as an independent risk factor for atherosclerotic cardiovascular disease, 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 cardiovascular disease. This consideration was confirmed by the decline in cardiovascular disease mortality and death from diabetes as a consequence of the population-wide weight loss of an average of 4–5 kg during the Cuban economic crisis of the early to mid-1990s [19]. Several diets and lifestyle interventions, which lead to a weight loss 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, amelioration of the inflammatory state and improvement in insulin resistance [20]. Improvement in lipid profile and insulin resistance were also reported in studies investigating the effects of pharmacotherapy 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 results on the lipid profile and blood pressure
and inflammatory state obtained with some of the pharmacotherapy agents, data concerning the improvement in CVD outcomes is lacking.

Among all strategies assisting patients to lose weight, bariatric surgery provides more significant and sustainable weight loss, particularly for morbidly obese individuals, compared to non-surgical treatment approaches. A meta-analysis of 11 studies with 796 obese individuals and a follow-up period ≥ 6 months have revealed that bariatric surgery leads to an additional weight loss of 26 kg compared to the non-surgical treatment while blood pressure and cholesterol concentrations were not significantly different [25]. The prospective, controlled Swedish Obese Study (SOS) has shown that the weight loss achieved with the bariatric surgery was preserved even after 20 years of the surgery [26]. Bariatric surgery has been shown to improve cardiovascular disease 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 reduce following bariatric surgery [30]. The SOS study has revealed that the number of cardiovascular deaths and cardiovascular events of the subjects allocated to bariatric surgery was significantly lower than that of the subjects receiving non-surgical 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 subjects who underwent bariatric surgery, have reported valuable data regarding the relation of weight loss and cardiovascular outcomes [32]. Following a follow-up ranging between 2-14.7 years, the overall mortality reduced by 50% in addition to the reductions in the incidence of myocardial infarction and stroke. However, more actual data is required to address the impact of bariatric surgery on cardiovascular outcomes. Furthermore, there still 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 the subjects 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 the levels of the ghrelin, which is produced in the gastric fundus and shown to increase appetite [36,37]. Recently, Altin and colleagues published their results of 106 subjects 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 pressures [38].

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 demonstrate that LSG was associated with improvements not only in CIMT, EFT, and ABI, which has 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 the improvement in APV and ABI following LSG. The improvements in EFT and CIMT observed in our study population are consistent with the data provided by Altin et al. Our findings also demonstrate 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. which shows improvement in surrogate 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 monitorisation of the CAD burden in subjects undergoing SLV.
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 of the subjects. 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 months duration following the LSG may not be appropriate for evaluating the changes in CIMT. However, previous evidence in subjects with diabetes indicates that CIMT may respond to optimization of diabetes treatment even in 2 weeks period [39]. These results therefore need to be interpreted with caution. Finally, the 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 subjects at postoperative 6 months. The improvement in APV is correlated with the improvement in BMI, CIMT, EFT, and ABI.
REFERENCES


Table 1. Demographic features, baseline laboratory measurements, and clinical characteristics of the study population

<table>
<thead>
<tr>
<th></th>
<th>n = 71</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>37.6 (11.2)</td>
</tr>
<tr>
<td>Gender, male</td>
<td>48 (67.6%)</td>
</tr>
<tr>
<td>Hypertension, n</td>
<td>23 (32.3%)</td>
</tr>
<tr>
<td>Diabetes, n</td>
<td>26 (36.6%)</td>
</tr>
<tr>
<td>Smoking, n</td>
<td>18 (25.3%)</td>
</tr>
<tr>
<td>Leukocyte count, 10^3/mm³</td>
<td>8.5 (2.3)</td>
</tr>
<tr>
<td>AST, U/L</td>
<td>24.2 (9.2)</td>
</tr>
<tr>
<td>ALT, U/L</td>
<td>27.8 (10.6)</td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td>0.71 (0.16)</td>
</tr>
</tbody>
</table>

Data are presented as mean (standard deviation) for continuous variables and as frequency (%) for categorical variables.

ALT = Alanine aminotransferase, AST = Aspartate aminotransferase
Table 2. The comparison of the clinical, laboratory, and echocardiographic parameters from baseline to the postoperative 6 months

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Postoperative 6 months</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>129 (31)</td>
<td>100 (21)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Body mass index, kg/m2</td>
<td>47.7 (6.5)</td>
<td>36.9 (5.4)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Fasting plasma glucose, mg/dl</td>
<td>104.6 (23.2)</td>
<td>98.6 (28.9)</td>
<td>0.40</td>
</tr>
<tr>
<td>Mean platelet volume, fl</td>
<td>7.4 (1.5)</td>
<td>7.9 (1.3)</td>
<td>0.070</td>
</tr>
<tr>
<td>NLR</td>
<td>1.6 (0.8)</td>
<td>1.7 (0.6)</td>
<td>0.17</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dl</td>
<td>40.8 (10.5)</td>
<td>41.7 (11.3)</td>
<td>0.38</td>
</tr>
<tr>
<td>TG, mg/dl</td>
<td>226 (72)</td>
<td>164 (70)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LDL cholesterol, mg/dl</td>
<td>108 (27)</td>
<td>91 (16)</td>
<td>0.001</td>
</tr>
<tr>
<td>EF, %</td>
<td>62.3 (2.4)</td>
<td>62.6 (2.3)</td>
<td>0.36</td>
</tr>
<tr>
<td>SBP</td>
<td>136 (15)</td>
<td>122 (14)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>DBP</td>
<td>78 (9)</td>
<td>73 (6)</td>
<td>0.004</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>82 (15)</td>
<td>73 (10)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>EFT, cm</td>
<td>0.65 (0.14)</td>
<td>0.58 (0.13)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>ABI</td>
<td>0.85 (0.08)</td>
<td>0.95 (0.05)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>CIMT, mm</td>
<td>1.07 (0.06)</td>
<td>0.99 (0.13)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>APV</td>
<td>44.2 (4.8)</td>
<td>50.1 (6.6)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Data are presented as mean (standard deviation)

ABI = Ankle-brachial index, APV = aortic propagation velocity, CIMT = Carotid intima-media thickness, DBP = Diastolic blood pressure, EF = Ejection fraction, EFT = Epicardial fat thickness, HDL = High-density lipoprotein, LDL = Low-density lipoprotein,
NLR = Neutrophil to lymphocyte ratio, SBP = Systolic blood pressure, sPAP = Systolic pulmonary artery pressure, TG = Triglyceride
Table 3. Correlation analysis between Δ as aortic propagation velocity, Δ epicardial fat, Δ carotid intima media thickness, Δ ankle-brachial index and various clinical variables

<table>
<thead>
<tr>
<th></th>
<th>Δ EFT</th>
<th></th>
<th>Δ ABI</th>
<th></th>
<th>Δ CIMT</th>
<th></th>
<th>Δ APV</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>P</td>
<td>r</td>
<td>P</td>
<td>r</td>
<td>P</td>
<td>r</td>
<td>P</td>
</tr>
<tr>
<td>Δ BMI</td>
<td>0.338</td>
<td>0.011</td>
<td>0.046</td>
<td>0.74</td>
<td>0.481</td>
<td>0.001</td>
<td>-0.370</td>
<td>0.005</td>
</tr>
<tr>
<td>Δ FPG</td>
<td>0.255</td>
<td>0.058</td>
<td>-0.082</td>
<td>0.55</td>
<td>0.133</td>
<td>0.33</td>
<td>-0.166</td>
<td>0.22</td>
</tr>
<tr>
<td>Δ HDL</td>
<td>0.231</td>
<td>0.086</td>
<td>0.124</td>
<td>0.36</td>
<td>0.028</td>
<td>0.84</td>
<td>-0.189</td>
<td>0.16</td>
</tr>
<tr>
<td>Δ TG</td>
<td>0.096</td>
<td>0.48</td>
<td>-0.077</td>
<td>0.57</td>
<td>0.026</td>
<td>0.85</td>
<td>0.110</td>
<td>0.42</td>
</tr>
<tr>
<td>Δ LDL</td>
<td>0.166</td>
<td>0.220</td>
<td>0.107</td>
<td>0.43</td>
<td>0.239</td>
<td>0.076</td>
<td>-0.050</td>
<td>0.71</td>
</tr>
<tr>
<td>Δ MPV</td>
<td>0.132</td>
<td>0.39</td>
<td>-0.068</td>
<td>0.66</td>
<td>-0.145</td>
<td>0.34</td>
<td>-0.151</td>
<td>0.32</td>
</tr>
<tr>
<td>Δ NLR</td>
<td>-0.005</td>
<td>0.97</td>
<td>0.047</td>
<td>0.73</td>
<td>0.032</td>
<td>0.93</td>
<td>0.030</td>
<td>0.83</td>
</tr>
<tr>
<td>Δ SBP</td>
<td>-0.034</td>
<td>0.80</td>
<td>-0.443</td>
<td>0.001</td>
<td>0.339</td>
<td>0.011</td>
<td>-0.297</td>
<td>0.026</td>
</tr>
<tr>
<td>Δ DBP</td>
<td>0.099</td>
<td>0.47</td>
<td>-0.136</td>
<td>0.32</td>
<td>0.262</td>
<td>0.053</td>
<td>-0.410</td>
<td>0.002</td>
</tr>
<tr>
<td>Δ EFT</td>
<td>-</td>
<td>-</td>
<td>0.212</td>
<td>0.12</td>
<td>0.478</td>
<td>&lt;0.001</td>
<td>-0.369</td>
<td>0.005</td>
</tr>
<tr>
<td>Δ ABI</td>
<td>0.212</td>
<td>0.12</td>
<td>-</td>
<td>-</td>
<td>0.183</td>
<td>0.18</td>
<td>0.365</td>
<td>0.006</td>
</tr>
<tr>
<td>Δ CIMT</td>
<td>0.478</td>
<td>&lt;0.001</td>
<td>0.183</td>
<td>0.18</td>
<td>-</td>
<td>-</td>
<td>-0.356</td>
<td>0.007</td>
</tr>
<tr>
<td>Δ APV</td>
<td>-0.369</td>
<td>0.005</td>
<td>0.365</td>
<td>0.006</td>
<td>-0.356</td>
<td>0.007</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Delta (Δ) values were obtained by subtracting sixth-month values from the baseline values.

BMI = Body mass index, FPG = Fasting plasma glucose, MPV = Mean platelet volume,

For abbreviations of ABI, APV, CIMT, DBP, EFT, HDL, LDL, NLR, SBP and TG - see Table 2
**Table 4.** Linear regression analysis demonstrating the predictors of the change in APV, CIMT, ABI, and EFT

<table>
<thead>
<tr>
<th></th>
<th>APV</th>
<th>CIMT</th>
<th>ABI</th>
<th>EFT</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔSBP</td>
<td>-0.254</td>
<td>-0.216-0.038</td>
<td>0.17</td>
<td>0.010</td>
</tr>
<tr>
<td>ΔDBP</td>
<td>0.149</td>
<td>-0.126-0.299</td>
<td>0.42</td>
<td>-0.031</td>
</tr>
<tr>
<td>ΔBMI</td>
<td>0.338</td>
<td>0.101-1.706</td>
<td><strong>0.028</strong></td>
<td>0.447</td>
</tr>
<tr>
<td>ΔLDL</td>
<td>-0.150</td>
<td>-0.077-0.023</td>
<td>0.29</td>
<td>0.156</td>
</tr>
<tr>
<td>ΔTG</td>
<td>0.163</td>
<td>-0.007-0.026</td>
<td>0.26</td>
<td>0.098</td>
</tr>
<tr>
<td>ΔHR</td>
<td>-0.031</td>
<td>-0.106-0.084</td>
<td>0.82</td>
<td>0.076</td>
</tr>
</tbody>
</table>

Delta (Δ) values indicate the change in selected variables from baseline to the postoperative 6 months.

For the abbreviations of ABI, APV, CIMT, DBP, EFT, LDL, SBP and TG - see Table 2, for BMI - see Table 3.