Cardiovascular effects of laparoscopic sleeve gastrectomy

Laparoskopik sleeve gastrektominin kardiyovasküler etkileri

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In this issue of the Archives of The Turkish Society of Cardiology, Simsek et al.[1] have evaluated the short-term (3 months) effects of laparoscopic sleeve gastrectomy (LSG) on body weight and echocardiographic diastolic function. Briefly, 41 obese patients [mean age: 42.8 years; 51.1% female; mean body mass index (BMI): 44.9 kg/m²] underwent LSG. The body weight, BMI, and body surface area (BSA) were significantly reduced following the procedure: 26.6±10.9 kg, 8.8±3.9 kg/m², and 0.27±0.12 m², respectively (p<0.001 for all comparisons). The mean total weight loss percentage (TWL%) was 20.1±6.7% and the mean excess weight loss percentage (EWL%) was 48.3±21.1%.1 The TWL% was similar between genders, whereas the EWL% was significantly greater in male patients compared with females (56.3±23.4 vs. 40.7±15.5%, respectively; p=0.036). In terms of echocardiographic parameters, the E/A ratio was significantly increased in 95% of the patients, whereas the E/e ratio was significantly reduced in 73%, as was the left atrial volume (LAV), the LAV index, and the left ventricle mass (LVM) in 95%, 68%, and 75% of the patients, respectively.1 A significant moderate association was found between EWL% and ΔLAV (r=0.39; p=0.012). No other correlations between changes in body weight measurements and parameters of diastolic function were observed.1

The findings of Simsek et al.[1] highlight the potential short-term benefits of LSG on weight loss and diastolic cardiac function. Of note, LAV has been suggested as an independent predictor of cardiovascular (CV) outcomes.2 The left atrial volume (LAV) has been suggested as an independent predictor of cardiovascular (CV) outcomes, and thus its reduction, induced by LSG, can minimize CV risk. As mentioned by the authors,[1] there are limited data on the effects of LSG on cardiac function. In this context, Iancu et al.[3] reported significant improvements in left ventricular (LV) hypertrophy and diastolic function up to 12 months after LSG in 34 obese patients (mean age: 39 years; 35.2% male; mean BMI: 43.6 kg/m²). Similarly, Leung et al.[4] found significant improvements in both systolic and diastolic myocardial function in 8 obese patients with type 2 diabetes mellitus (T2DM) (mean age: 56 years; 2 males; mean BMI: 44 kg/m²) at 9 months following LSG. Furthermore, Kemaloglu et al.[5] demonstrated that LSG beneficially affected LV systolic function and reversed LV remodelling in 53 obese patients.
Apart from weight reduction and improvements in cardiac function, LSG may beneficially affect other cardiometabolic parameters, such as blood pressure, glycated hemoglobin A1C (HbA1C), lipids (total cholesterol, triglycerides, and high-density lipoprotein), and visceral adiposity. Especially in obese T2DM patients, LSG can lead to complete remission of T2DM, although T2DM can relapse in the long-term. Subclinical atherosclerosis (assessed by flow-mediated dilation and carotid intima-media thickness) may also improve after LSG.

Obstructive sleep apnea syndrome (OSAS) and non-alcoholic fatty liver disease (NAFLD) have been associated with increased CV risk. LSG has been shown to improve/reverse OSAS and liver steatosis/fibrosis. It has also been suggested that NAFLD could represent an indication for LSG since liver steatosis can be completely resolved following LSG in up to 90% of cases. Furthermore, in obese adolescents, LSG has been reported to be more effective than lifestyle measures in reducing biopsy-proven non-alcoholic steatohepatitis and hepatic fibrosis at 1 year. Interestingly, in a 3-year follow-up study, remission after LSG was observed for the following comorbidities: insulin resistance (in 89.4% of the patients), NAFLD (in 84.6%), hypertension (in 75%), and dyslipidemia (in 52%). Another study showed that LSG was more effective at decreasing circulating liver test activity than Roux-Y-gastric bypass in obese T2DM patients. Further research is needed to clarify decision-making on the most appropriate bariatric procedure for such patients.

Increased epicardial fat is related to CV risk. LSG can significantly decrease epicardial adiposity. However, data are scarce and further studies are needed. Since excessive fat depositions could occur in other organs, apart from the liver and heart, further increasing CV risk, the effects of LSG on these fat deposits need to be evaluated.

Blanco et al. reported that LSG at 12 months significantly decreased CV risk as assessed using the atherosclerotic CV disease and Framingham risk scores in 360 obese patients. Bariatric surgery, including LSG, has been shown to have a protective effect against myocardial infarction incidence at 4 years as well as against macrovascular events and coronary artery disease at 4.6 years.

Current (2016) European Society of Cardiology and European Atherosclerosis Society guidelines mention that bariatric surgery may be beneficial for reducing the risk of CV events. The American Diabetes Association has recommended (in 2019) bariatric surgery (now named “metabolic surgery”) as an option to treat obesity in obese T2DM patients with a BMI ≥30 kg/m². The European Association for the Study of Obesity guidelines state that bariatric surgery should be considered in patients aged between 18 and 60 years with i) a BMI between 35.0 and 39.9 kg/m² and co-morbidities (such as T2DM, severe joint disease, cardiorespiratory disease, and severe obesity-related psychological problems) or ii) a BMI ≥40.0 kg/m².

In conclusion, LSG has been reported to have a beneficial effect not only on body weight, but also other cardiac function indices, cardiometabolic parameters (such as cardiac function, blood pressure, HbA1c, lipids, visceral adiposity, NAFLD, epicardial fat and OSAS), as well as subclinical atherosclerosis, thus minimizing CV risk. Larger studies are needed to establish the role of such effects, especially in the long-term.

**Declaration of interest**

NK has given talks, attended conferences and participated in trials sponsored by Amgen, Angelini, Astra Zeneca, Boehringer Ingelheim, MSD, Novartis, NovoNordisk, Sanofi and WinMedica. DPM has given talks and attended conferences sponsored by MSD, AstraZeneca and Libytec.

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