

Improvement of left ventricular diastolic function and left heart morphology in young women with morbid obesity six months after bariatric surgery

Katarzyna Kurnicka¹, Justyna Domienik-Karłowicz¹, Barbara Lichodziejewska¹, Maksymilian Bielecki², Marta Kozłowska¹, Sylwia Goliszek¹, Olga Dzikowska-Diduch¹, Wojciech Lisik³, Maciej Kosieradzki³, Piotr Pruszczyk¹

¹Department of Internal Medicine and Cardiology, Medical University of Warsaw, Poland

²Department of Psychology, SWPS University of Social Sciences and Humanities, Warsaw, Poland

³Department of General Surgery and Transplantology, Medical University of Warsaw, Poland

Abstract

Background: Obesity contributes to left ventricular (LV) diastolic dysfunction (LVDD) and may lead to diastolic heart failure. Weight loss (WL) after bariatric surgery (BS) may influence LV morphology and function. Using echocardiography, this study assessed the effect of WL on LV diastolic function (LVDF) and LV and left atrium (LA) morphology 6 months after BS in young women with morbid obesity.

Methods: Echocardiography was performed in 60 women with body mass index ≥ 40 kg/m², aged 37.1 ± 9.6 years prior to and 6 months after BS. In 38 patients, well-controlled arterial hypertension was present. Heart failure, coronary artery disease, atrial fibrillation and mitral stenosis were exclusion criteria. Parameters of LV and LA morphology were obtained. To evaluate LVDF, mitral peak early (E) and atrial (A) velocities, E-deceleration time (DcT), pulmonary vein S, D and A reversal velocities were measured. Peak early diastolic mitral annular velocities (E') and E/E' were assessed.

Results: Mean WL post BS was 35.7 kg (27%). A postoperative decrease in LV wall thickness, LV mass (mean 183.7 to 171.5 g, $p = 0.001$) and LA parameters (area, volume) were observed. LVDD was diagnosed in 3 patients prior to and in 2 of them subsequent to the procedure. An improvement in LVDF Doppler indices were noted: increased E/A, D and E' lateral, and decreased S/D and lateral E/E'. None of the patients showed increased LV filling pressure. No significant correlations between hypertension and echo-parameters were demonstrated.

Conclusions: Six months after BS weight loss resulted in the improvement of LVDF and left heart morphology in morbidly obese women. (Cardiol J 2018; 25, 1: 97–105)

Key words: morbid obesity, bariatric surgery, echocardiography, left ventricular diastolic function, left atrium

Introduction

The role of obesity as an independent risk factor for the development of cardiovascular diseases such as arterial hypertension, coronary artery disease and heart failure (HF) has been well documented in the literature [1]. The risk of

developing HF increases by over 5% with every 1 kg/m² increase in the body mass index (BMI) [2]. Obesity predisposes to HF, mainly due to the impairment of left ventricular (LV) relaxation caused by myocardial hypertrophy as well as due to an increase in circulating blood volume which influences preload and leads to higher stroke volume.

Address for correspondence: Katarzyna Kurnicka, MD, PhD, Department of Internal Medicine and Cardiology, Medical University of Warsaw, ul. Lindleya 4, 02–005 Warszawa, Poland, tel: +48 22 5021144, fax: +48 22 5022142, e-mail: kkurnicka@yahoo.pl

Received: 25.09.2016

Accepted: 18.01.2017

Left ventricular diastolic dysfunction (LVDD) is often found in obese patients, and obesity is one of the most common causes of HF with preserved ejection fraction [3, 4]. Bariatric surgery (BS) is associated with both reduced cardiovascular mortality and lower incidence of cardiovascular events in obese adults [5]. A new meta-analysis by Aggarwal et al. [6] supported the effects of BS on cardiac structure assessed by echocardiography and cardiac magnetic resonance, and suggested a significant improvement in cardiac morphology and function after bariatric procedures. There is still limited evidence with respect to an improvement in LV diastolic function (LVDF) in patients with severe obesity after body mass (BM) reduction [4, 7].

The aim of this study was to assess the impact of BM reduction on LVDF and LV and left atrium (LA) morphology 6 months after BS with the use of echocardiography in young women with morbid obesity.

Methods

Patients

The study population consisted of patients qualified for BS in the referenced department and was characterized by high prevalence of women participants. Sixty women with class III obesity according to the World Health Organization (WHO) criteria, aged 37.1 ± 9.6 years, with $\text{BMI} \geq 40 \text{ kg/m}^2$ underwent cardiological evaluation followed by BS. Patients with coronary artery disease, atrial fibrillation, LV systolic dysfunction (left ventricular ejection fraction [LVEF] $< 50\%$), HF or mitral stenosis, prosthetic mitral valves or surgical rings, significant aortic valve disease, chronic kidney disease and estimated glomerular filtration rate $< 60 \text{ mL/min/1.73 m}^2$, uncontrolled diabetes or systemic diseases were not included in the study. This study was approved by the Ethics Committee of the Medical University of Warsaw. All patients participating in the study gave written consent.

Clinical evaluation was performed at the time of qualification, in the majority of patients within 1 month prior to surgery and 6 months after BS. The preoperative evaluation was performed when patients were in stable clinical condition and receiving optimal medical treatment. Anthropometric parameters such as BM, BMI, body surface area (BSA) and blood pressure (BP) along with the a list of currently used medications were recorded. Weight loss results were expressed in kilograms and relative measure — percentage excess weight loss (% EWL) and percent weight loss (% WL). At

least 50% EWL was the criterium of successfully performed BS.

Bariatric surgery

Roux-en-Y gastric bypass was performed in 24 patients and vertical banded gastroplasty in another 24 patients. Twelve patients underwent laparoscopic sleeve gastrectomy. The type of surgery was chosen depending on patients' BMI and co-morbidities.

Echocardiography

Patients underwent transthoracic echocardiography with two-dimensional (2D), M-mode and Doppler assessments (iE33 Philips MS, Andover, MA). Despite the presence of severe obesity, echocardiographic image quality was adequate for the analysis in all patients. A standardized examination was performed with patients placed in the left-lateral decubitus position, with constant monitoring of a single electrocardiogram (ECG) lead. The findings were interpreted by an experienced physician according to the recommendations of the Echocardiography Working Group of the Polish Cardiac Society, based on the guidelines of the European Association of Cardiovascular Imaging [8, 9]. A follow-up evaluation was performed 6 months after surgery and the sonographer was blinded to the results of the previous examination.

Left ventricle

Left ventricular end-diastolic diameter (LVEDd), interventricular septum (IVS) and posterior (inferolateral) wall (PW) thickness were recorded at end-diastole using 2D and M-mode presentations in the parasternal long axis view. LV dimensions were recorded by placing the ultrasound beam maximally perpendicularly to its long axis. Measurements acquired in the M-mode were later verified using 2D visualizations. Wall thickness $\geq 13 \text{ mm}$ was defined as at least moderate LV hypertrophy (LVH), and severe hypertrophy when the wall thickness was $\geq 16 \text{ mm}$, respectively [9].

Left ventricular mass (LVM) was calculated using the following Devereux regression equation: $\text{LVM} = \{0.8 \times 1.04 \times [(\text{IVS} + \text{LVEDd} + \text{PW})^3 - \text{LVEDd}^3] + 0.6\}$ [10]. Adjusting LVM for BSA was performed to obtain the LV mass index (LVMI-BSA). The upper limits for LVM and LVMI-BSA were 162 g and 95 g/m^2 , respectively. Moderate hypertrophy was defined as LVM ranging $187\text{--}210 \text{ g}$ and LVMI-BSA ranging $109\text{--}121 \text{ g/m}^2$, whereas severe hypertrophy was defined as $\text{LVM} > 210 \text{ g}$

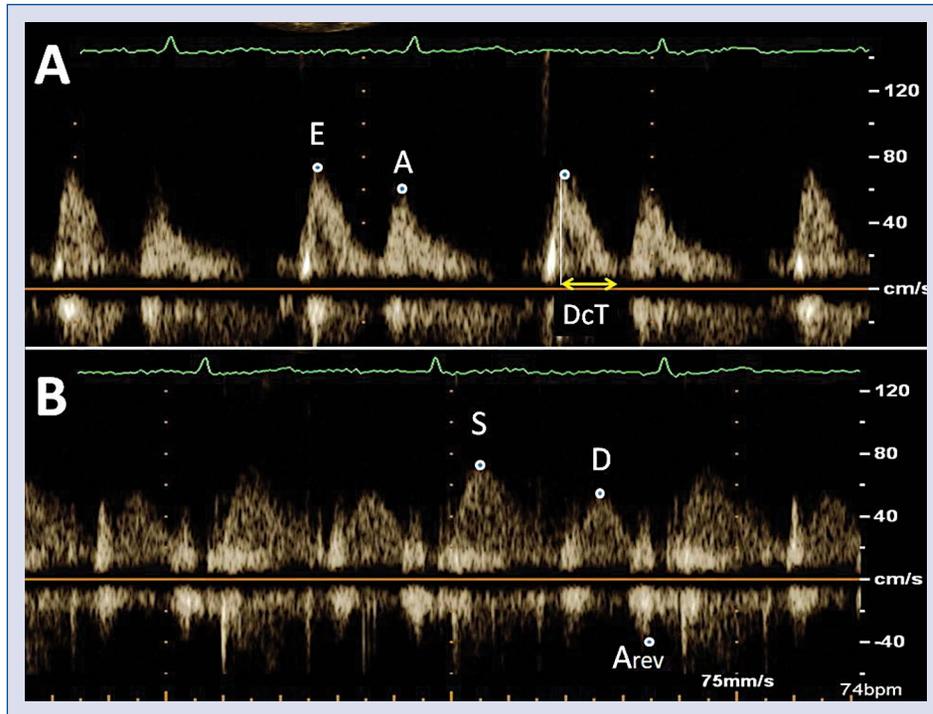


Figure 1. Mitral valve inflow; E — maximal velocity of the early wave; A — maximal velocity of the atrial wave; DcT — E-wave deceleration time (A). Pulmonary venous inflow. S — maximal velocity of the of systolic wave; D — maximal velocity of the diastolic wave; Arev — maximal velocity of the retrograde flow wave during atrial contraction (B).

and LVMI-BSA $> 121 \text{ g/m}^2$ [9]. LVMI was additionally adjusted to height raised to the power of 2.7 (LVMI-height) and defined LVH as the LVMI-height $\geq 47 \text{ g/m}^{2.7}$ [9, 11]. To evaluate LV geometry, LV relative wall thickness (RWT) using the following formula: $\text{RWT} = 2 \times \text{PW} / \text{LVEDd}$ [9] was assessed.

The biplane method of disks summation (modified Simpson's rule) to calculate LVEF was used [9].

Left ventricular diastolic function

Left ventricular diastolic function was assessed using Doppler mitral valve inflow (MVI) and pulmonary venous inflow (PVI) parameters, early diastolic mitral annular velocity in tissue Doppler imaging (TDI), and measurements of the LA [9, 12].

Using the sampling gate positioned in the mitral valve orifice, at the level of the leaflet tips, the MVI pattern was registered and the maximal velocity (V_{max}) of the early wave (E) and the atrial wave (A) were measured. The E/A ratio was calculated followed by the measurement of the E-wave deceleration time (DcT) (Fig. 1A). After placing the Doppler gate at the level of the right upper pulmonary vein, the spectrum of PVI was registered, V_{max} of systolic (S) and diastolic (D) waves were measured,

S/D ratio was calculated, which was followed by the measurement of V_{max} of retrograde flow wave into pulmonary veins (Arev) during atrial contraction (Fig. 1B). Moreover, the duration of A (AD) and Arev (ArevD) were measured. Using TDI, the early diastolic velocities of the lateral (E'lat) and septal (E' sept) parts of the mitral annulus were recorded (Fig. 2). The lateral, septal and average E/E' ratios were then derived and the LV filling pressure (LVFP) was estimated. Doppler measurements reflected an average of 3 cardiac cycles.

Left atrium morphology

Planimetry and volume measurements of the LA in 2D imaging were performed during late ventricular systole, immediately before mitral valve opening, when LA size is the largest. The maximal LA anterior-posterior dimension (LAd) was obtained in the long axis in the parasternal view — the upper limit for women was defined as 38 mm. The LA was traced in the 4-chamber apical view to calculate LA area (LAa) (Fig. 3). Mild enlargement was defined as $\text{LAa} \geq 20 \text{ cm}^2$ [13].

The LA volume (LAV) was estimated using the modified Simpson's method based on disk summation acquired after tracing the outline of LA in two

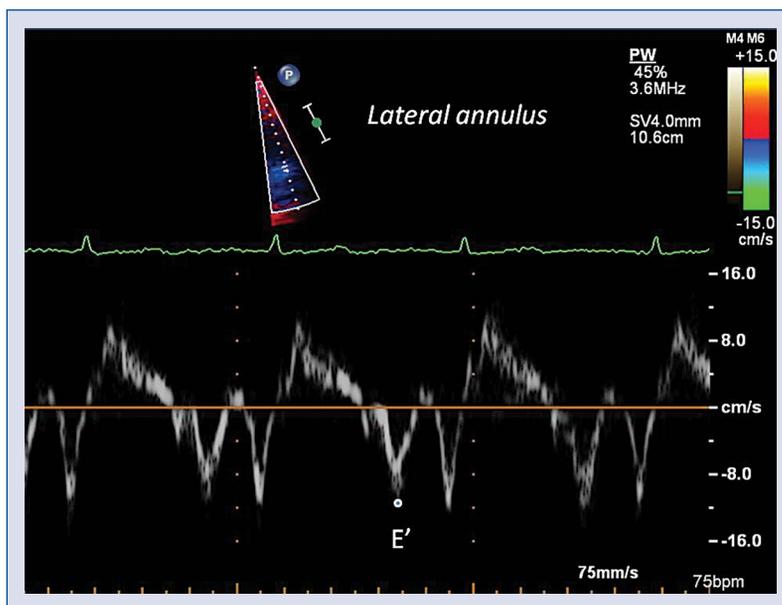


Figure 2. Tissue Doppler imaging of lateral mitral annulus; E' — early diastolic velocity.

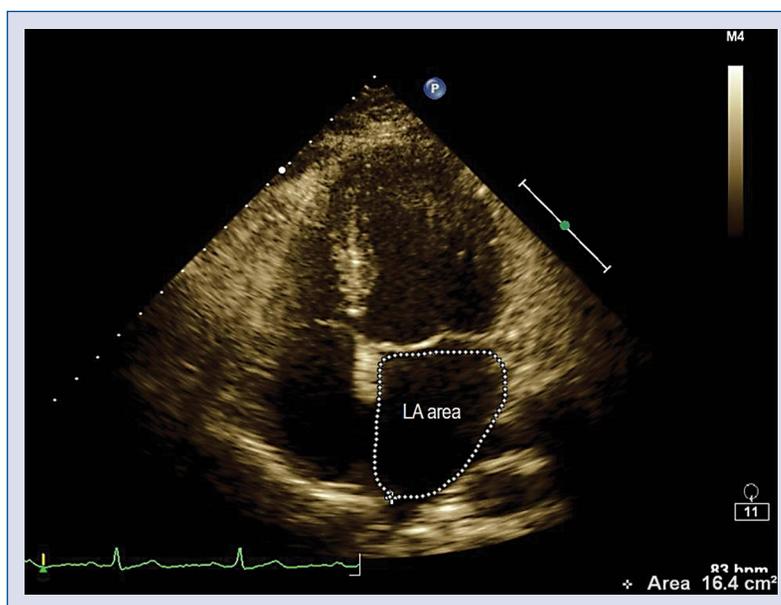


Figure 3. Four chamber view: calculation of left atrial (LA) area.

planes in 4- and 2-chamber apical views, excluding pulmonary vein orifices and LA appendage (Fig. 4). Mitral annulus was used as the lower LA border. The LA volume index (LAVI) was obtained by indexing the LAV for BSA (mL/m^2). Mildly abnormal values were considered when $\text{LAV} \geq 53 \text{ mL}$ and $\text{LAVI} \geq 29 \text{ mL}/\text{m}^2$ and moderately abnormal when $\text{LAV} \geq 63 \text{ mL}$ and $\text{LAVI} \geq 34 \text{ mL}/\text{m}^2$ [9, 13].

Left ventricular diastolic dysfunction

Left ventricular diastolic dysfunction was diagnosed, when in TDI, $E'_{\text{lat}} < 10 \text{ cm/s}$ or $E'_{\text{sept}} < 8 \text{ cm/s}$ and at least two of the following were present: $E/A < 0.8$, $\text{DcT} > 200 \text{ ms}$ or predominant S wave during PVI ($S/D > 1$). An average $E/E' \leq 8$ was defined as normal whereas $E/E' > 13$ as increased LVFP, respectively. In cases with an

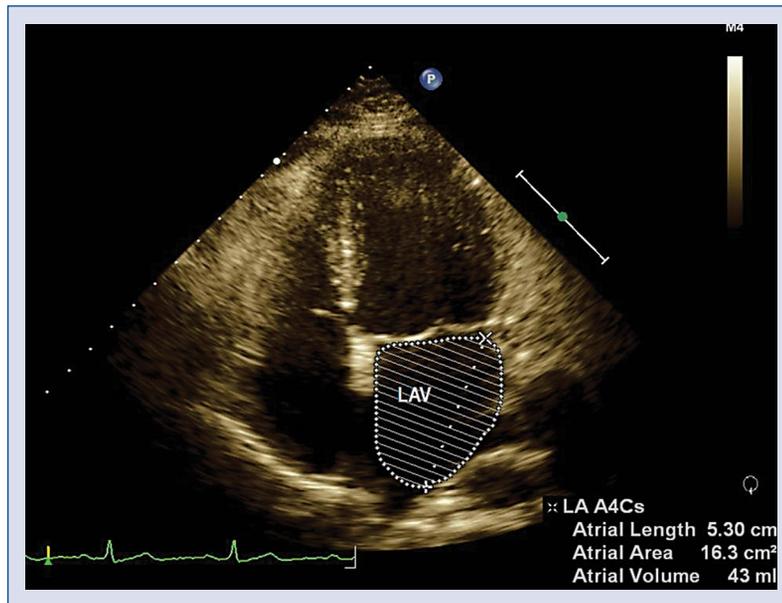


Figure 4. Four chamber view: calculation of left atrial volume (LAV).

average E/E' between 9 and 13, $A_{\text{revD-AD}} \geq 30$ ms and $LAVI \geq 34$ mL/m² was additionally needed to diagnose increased LVFP [12].

Statistical analysis

Descriptive statistics for continuous variables are expressed as means and standard deviations or medians and range. Comparisons between measurements obtained before and after surgery for continuous variables were conducted with the use of dependent samples t-test or Wilcoxon signed-rank test, depending on the normality of the distribution of difference scores (assessed with the Shapiro-Wilk test). Comparisons between dichotomous variables were performed with the McNemar's χ^2 test. All correlations were computed using the Spearman's rho coefficient. Data were considered significant at p value < 0.05.

Results

Group characteristics

Sixty women aged 37.1 ± 9.6 years, with class III obesity according to the WHO (median BMI 47.0 kg/m², IQR 12.5, mean BSA 2.1 ± 9.6 m²) were included in the study. Thirty-eight patients were diagnosed with arterial hypertension prior to the surgery (63.3%) (Table 1). These patients received standard treatment with angiotensin converting enzyme inhibitor (enalapril), diuretics (furosemide) and in a few cases (6 patients) with

beta-adrenolytics or calcium-blockers (bisoprolol or amlodipine). More than one antihypertensive drug was used by 23 patients. Nine (15.0%) patients had type 2 diabetes, which was treated with metformin in 8 cases and with insulin in 1 case.

Clinical evaluation

Six months after BS, the median WL was 35.7 kg, with values ranging from 13.4 kg to 61.9 kg. The median %EWL was 48.8 (23.1–79.6) and the %WL was 27.0 (11.0–44.6). Representing weight changes as %EWL shows that BS was successful in a majority of cases — lower quartile of %EWL was equal to 44.

Arterial hypertension was found in 27 (45%) patients, the number of patients requiring more than one antihypertensive drug was reduced from 23 to 14. A normalization in BP values was achieved in 11 patients resulting in total discontinuation of antihypertensive treatment. The number of patients with diabetes remained the same.

After 6 months following BS, the BM, BSA and BMI (med. 34.0 kg/m²) of patients decreased significantly. The systolic and diastolic BP as well as the average heart rate values were significantly lower (Table 1).

The final effect of BS was related to BM and BMI prior to surgery. Women with the highest preoperative BM had the largest reduction in BM. A significant correlation between BM before BS and the difference score (Δ body mass) was observed

Table 1. Clinical characteristics of patients before and 6 months after bariatric surgery (BS) (n = 60).

Parameter	Before BS	After BS	P
Hypertension	38 (63.3%)	27 (45.0%)	0.003
Body mass [kg]	129.1 (102.4–186.2)	94.75 (65.8–136.4)	< 0.001*
BMI [kg/m ²]	47.0 (39.45–64.25)	34.0 (26.4–50.7)	< 0.001*
BSA [m ²]	2.1 (1.8–2.5)	1.85 (1.5–2.2)	< 0.001*
SBP [mm Hg]	130.0 (110.0–170.0)	120.0 (95.0–160.0)	< 0.001*
DBP [mm Hg]	80.0 (70.0–90.0)	80.0 (60.0–85.0)	< 0.001*
HR [bpm]	79.9 ± 9.35	72.3 ± 7.7	< 0.001**

*Wilcoxon signed-rank test; **Student’s t-test. Data are expressed as percentage, mean ± standard deviation or median and range; BMI — body mass index, BSA — body surface area; SBP — systolic blood pressure; DBP — diastolic blood pressure; HR — heart rate

Table 2. Left ventricle parameters before and 6 months after bariatric surgery (BS) (n = 60).

Parameter	Before BS	After BS	P
LVEDd [mm]	46.3 ± 3.6	45.6 ± 3.6	0.167**
IVS [mm]	11.0 (9.0–14.0)	11.0 (9.0–13.0)	0.002*
PW [mm]	11.0 (9.0–14.0)	11.0 (9.0–13.0)	0.004*
LVM [g]	183.7 ± 32.2	171.5 ± 27.7	< 0.001**
LVMI-BSA [g/m ²]	86.9 ± 14.3	92.1 ± 14.6	0.002**
LVMI-height [g/m ^{2.7}]	46.7 ± 9.0	43.6 ± 8.1	< 0.001**
RWT	0.476 ± 0.064	0.467 ± 0.048	0.231**
LVEF [%]	65 (55.0–68.0)	65 (60.0–68.0)	0.003*

*Wilcoxon signed-rank test; **Student’s t-test. Data are expressed as percentage, mean ± standard deviation or median and range; LVEDd — left ventricular end-diastolic diameter; IVS — interventricular septum thickness; PW — posterior (inferolateral) wall thickness; LVM — left ventricular mass; LVMI — left ventricular mass index adjusted for body surface area; LVMI-height — left ventricular mass index adjusted to height raised to the power of 2.7; RWT — relative wall thickness; LVEF — left ventricular ejection fraction

(r = 0.61, p < 0.001). The same effect was noted for BMI values (r = 0.564, p < 0.001).

Echocardiographic assessment

Before BS, moderate hypertrophy of IVS (≥ 13 mm) was observed in 6 (10.0%) patients, and moderate hypertrophy of the PW in 5 (8.3%) cases. None of the studied patients were diagnosed with severe LVH (≥ 16 mm). Moderate LVH, defined as LVM 187–210 g, was present in 19 (31.7%) patients, and when defined as LVMI-BSA ranging 109–121 g/m², in 1 (1.7%) patient. Significant LVH (LVM ≥ 211 g) was observed in 11 women. None of the studied patients met the LVMI-BSA criteria for severe hypertrophy. After surgery, the LVEDd did not change significantly, however, the IVS and PW thickness were significantly reduced. Moderate hypertrophy of the IVS was observed in 2 (3.3%) women, and moderate hypertrophy of the PW in 1 (1.7%) patient. Hence, the LVM was also reduced. When analyzing the LVM, 12 patients

met the criteria for moderate LVH and 6 patients were diagnosed with severe LVH. The LVMI-BSA was significantly higher after BS, because BM decreased faster than the LVM. The LVMI-height was significantly lower after BS and identified 27 patients with LVH before and 18 patients after the procedure. There was no significant change in RWT when pre- and post-surgery values were compared (Table 2).

All patients studied had normal LV systolic function. LVEF was significantly higher after surgery (Table 2).

Left ventricular diastolic function assessment

Prior to BS, mild LVDD (impaired LV relaxation) was diagnosed only in 3 patients based on MVI, PVI and TDI criteria. These 3 patients had hypertension, were older (48.3 ± 4.7 years) and had higher BMI (50.8 ± 9.1 kg/m²) than the remaining patients.

Table 3. Doppler parameters of left ventricular diastolic function before and 6 months after bariatric surgery (BS) (n = 60).

Parameter	Before BS	After BS	P
MVI E/A	1.1 (0.5–1.7)	1.2 (0.7–2.0)	0.04*
MVI DcT [MS]	192.5 ± 33.4	184.65 ± 29.35	0.194**
PVI S [m/s]	0.6 (0.4–0.9)	0.6 (0.3–1.0)	0.265*
PVI D [m/s]	0.4 (0.3–0.65)	0.45 (0.3–0.9)	0.007*
PVI S/D	1.42 ± 0.27	1.29 ± 0.33	0.003**
PVI Arev [m/s]	0.3 (0.2–0.4)	0.3 (0.2–0.4)	0.289*
E' lateral [cm/s]	12.45 ± 2.9	15.7 ± 3.6	0.094**
E/E' lateral	7.5 ± 2.1	6.6 ± 2.1	0.007**
E' septal [cm/s]	10.3 (6.2–16.6)	11.5 (6.4–17.8)	0.489*
E/E' septal	8.75 (5.8–12.7)	8.35 (5.3–11.5)	0.911*

*Wilcoxon signed-rank test; **Student's t-test. Data are expressed as mean ± standard deviation or median and range; MVI/PVI — mitral/pulmonary vein inflow; E — mitral peak early diastolic velocity; A — mitral peak atrial flow velocity; DcT — deceleration time; S — pulmonary vein systolic velocity; D — pulmonary vein diastolic velocity; Arev — pulmonary vein atrial reversal flow velocity; E' — peak early diastolic velocity of mitral annulus

Table 4. Morphological parameters of the left atrium before and 6 months after bariatric surgery (BS) (n = 60).

Parameter	Before BS	After BS	P
LAd [mm]	36.0 (28.0–40.0)	34.5 (25.0–40.0)	0.003*
LAa [cm ²]	17.3 ± 2.6	16.2 ± 2.4	< 0.001**
LAV [mL]	45.0 (25.0–94.0)	39.0 (21.0–76.0)	< 0.001*
LAVI [mL/m ²]	21.3 ± 5.3	21.4 ± 5.3	0.636**

*Wilcoxon signed-rank test; **Student's t-test. Data are expressed as mean ± standard deviation or median and range; LAd — left atrial diameter; LAa — left atrial area; LAV — left atrial volume; LAVI — left atrial volume index

After surgery, 2 of them still met predefined LVDD criteria. On both examinations, no patients met criteria of significantly increased LVFP.

In the study group, the mean E/A ratio was significantly higher on follow-up examination when compared with the initial one. No significant differences with respect to DcT were observed. However, the value of PVI diastolic velocity (D) was notably higher after surgery, the S/D ratio was significantly lower than that before surgery, and no differences for the S and Arev were noted (Table 3).

TDI evaluation revealed higher E'lat values on follow-up, while the E/E'lat was significantly lower than prior to BS (12.45 ± 2.9 vs. 15.7 ± 3.6 cm/s, p = 0.09 and 7.5 ± 2.1 vs. 6.6 ± 2.1, p = 0.007, respectively). A comparison between the septal E' and E/E' showed no significant differences (Table 3).

Left atrium assessment

Prior to surgery, an enlargement of LAd > 38 mm was observed in 10 (16.7%) women, and LAa ≥ 20 cm² was found in 9 (11.7%) women. Increased values were found of LAV in 9 females (15%; LAV ≥ 63 mL in 4 patients, LAV ≥ 53 mL in 5 patients) and increased values of LAVI in 2 patients (3.3%; LAVI ≥ 34 mL/m² as well as LAVI ≥ 29 mL/m² in 1 female).

After surgery, LAd and LAa exceeded the normal range in 4 (6.7%) women. Four (6.7%) patients had increased LAV values (LAV ≥ 63 mL in 2 patients, LAV ≥ 53 mL in 2 patients). The number of women with increased LAVI was 5 (8.3%), and it was higher than before BS (LAVI ≥ 29 mL/m² in 2 patients, LAVI ≥ 34 mL/m² in 3 patients) due to significant WL and decreased BSA.

The values of LAd, LAa and LAV were significantly lower after BS. This trend was not observed for LAVI (Table 4).

Body mass and echocardiography parameters

An analysis of echocardiographic parameters showed the following significant correlations: BM prior to surgery correlated significantly with the PW thickness ($r = 0.30$, $p = 0.021$), LVM ($r = 0.31$, $p = 0.016$), and LAV ($r = 0.26$, $p = 0.048$). Moreover, a statistical trend for the correlation between the IVS thickness and BM was observed ($r = 0.23$, $p = 0.080$).

The changes in BM (Δ body mass) were significantly correlated with the changes in PW thickness ($r = 0.32$, $p = 0.013$) and with LVM reduction ($r = 0.27$, $p = 0.034$). A marginally significant effect was shown for changes in IVS thickness ($r = 0.25$, $p = 0.058$).

No significant correlations were observed between BP values and echo-parameters obtained during the first examination.

Discussion

Obesity is a recognized factor of increased cardiovascular morbidity and mortality [14]. It leads to unfavorable remodeling of LV including its hypertrophy and LV dysfunction in a high fraction of obese people [15, 16]. The analysis of approximately 1160 severely obese patients performed by Avelar et al. [17] showed that increasing systolic BP and increasing BMI were independently associated with increased LVMI.

This study found moderate LVH, defined as LVM between 187 g and 210 g, in approximately one third of the obese, but none of the subjects studied herein suffered from severe LVH. Only patients with well-controlled hypertension, no evident HF or CAD, were included in the study, which may explain why relatively mild LV remodeling was observed. However, the indexation of LVM to height raised to the power of 2.7 identified in the study group more patients with LVH than the indexation to BSA.

Of note, significant correlations were found between the initial BM and LV wall thickness as well as LVM. Interestingly, we found no relationship between hypertension and the echocardiographic parameters. This indicates that obesity in itself is a driving factor of LVH. Moreover, the postoperative BM decrease correlated with the degree of LVM reduction.

Some authors reported echocardiographic signs of LVDD and increased LVFP in obese patients who were hypertensive and older than the remaining participants of the studied groups [7].

Despite advanced class III obesity according to the WHO, prior to bariatric surgery, we found LVDD of grade I (impaired myocardial relaxation) based on mitral and pulmonary inflow and TDI criteria only in 3 patients who also had hypertension and were older than the rest of the studied patients. After BS, 2 of them still met the predefined criteria for LVDD. None of studied patients met the criteria of increased LV filling pressure (no cases with an average $E/E' > 13$) before and after surgery. As mentioned above, this finding can be potentially explained by the selection criteria used in our study. It is known, that patient age should be taken into account when LVDF parameters are analyzed. The observed group was relatively young, with mean age of 37.1 years. However, significant postoperative improvement in a wide range of Doppler indices of LVDF were observed. A significant increase in E/A ratio in MVI and of diastolic velocity (D) of PVI with a decreased S/D ratio was noted. Moreover, the higher E'lat values observed after surgery reflected an improvement in movement for this part of the mitral annulus. Consequently, the lateral E/E' ratio was significantly lower than that obtained upon first examination. These results are in line with observations suggesting that weight loss following bariatric surgery improves LVDF. Fenk et al. [7] reported results after a 1-year follow-up in obese patients with LVDD, who achieved successful WL and significant improvement in E/A, E' or E/E'.

It is possible that changes in LVDF in the studied women may depend not only on decreased LVM and improved myocardial properties but also decreased content of epicardial fat tissue, which due to mechanical impairment of ventricle filling which may have had an impact on LVDF [18].

Left atrium enlargement reflects a chronic exposure to increased LVFP and is a well-recognized marker of long-lasting LVDD [12]. Prior to surgery, LA enlargement, defined as the presence of at least one abnormal LA echo-parameter (LAd, LAa, LAV), was observed in approximately 16% of patients. All of these parameters were significantly lower postoperatively. Interestingly, the reduction in the LA size was not significant in severely obese patients with LVDD, as reported by Fenk et al. [7] 1 year after BS.

It is of importance in the presented group of patients was that the indexed parameters such as LAVI may sometimes not show any change in volume because both LAV and BSA decrease simultaneously [19]. In the present observation before

surgery, LAVI did not exceed the recommended upper range in all patients with LA enlargement due to higher values of BSA, this was only revealed in postoperative assessment (BSA decreased, whereas LAV was comparable).

As already established, LA enlargement is associated with the risk of developing atrial fibrillation [20].

It is assumed that normalization of LA dimensions reflects a favorable impact of BS not only on LVDF but also on LA morphology and may lower the risk of supraventricular arrhythmias in obese patients [19].

Limitations of this study

This is a single center study comprised of a relatively small group of patients. Moreover, the studied group was rather homogenous. All women included in the study were diagnosed with class III obesity (BMI ≥ 40 kg/m²) and nearly the entire group benefited from a large reduction in BM. Since only relatively young women without significant cardiovascular complications were studied, it is difficult to extrapolate our results to the general population of obese people. In severely obese patients, echocardiographic measurements are especially susceptible to errors due to suboptimal visualization of cardiac structures which can also limit the power of observed correlations.

Conclusions

Weight loss 6 months subsequent to bariatric surgery resulted in improvement of left ventricular diastolic function and left heart morphology in morbidly obese women.

References

- Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation*. 2014 Jun 24. ; 129(25): S102–38, doi: [10.1161/01.cir.0000437739.71477.ee](https://doi.org/10.1161/01.cir.0000437739.71477.ee).
- Kenchaiah S, Evans JC, Levy D, et al. Obesity and the risk of heart failure. *N Engl J Med*. 2002; 347(5): 305–313, doi: [10.1056/NEJMoa020245](https://doi.org/10.1056/NEJMoa020245), indexed in Pubmed: 12151467.
- Bamba V, Rader DJ. Obesity and atherogenic dyslipidemia. *Gastroenterology*. 2007; 132(6): 2181–2190, doi: [10.1053/j.gastro.2007.03.056](https://doi.org/10.1053/j.gastro.2007.03.056), indexed in Pubmed: 17498511.
- Russo C, Jin Z, Homma S, et al. Effect of obesity and overweight on left ventricular diastolic function: a community-based study in an elderly cohort. *J Am Coll Cardiol*. 2011; 57(12): 1368–1374, doi: [10.1016/j.jacc.2010.10.042](https://doi.org/10.1016/j.jacc.2010.10.042), indexed in Pubmed: 21414533.
- Sjöström L, Peltonen M, Jacobson P, et al. Bariatric surgery and long-term cardiovascular events. *JAMA*. 2012; 307(1): 56–65, doi: [10.1001/jama.2011.1914](https://doi.org/10.1001/jama.2011.1914), indexed in Pubmed: 22215166.
- Aggarwal R, Harling L, Efthimiou E, et al. The Effects of Bariatric Surgery on Cardiac Structure and Function: a Systematic Review of Cardiac Imaging Outcomes. *Obes Surg*. 2016; 26(5): 1030–1040, doi: [10.1007/s11695-015-1866-5](https://doi.org/10.1007/s11695-015-1866-5), indexed in Pubmed: 26328532.
- Fenk S, Fischer M, Strack C, et al. Successful weight reduction improves left ventricular diastolic function and physical performance in severe obesity. *Int Heart J*. 2015; 56(2): 196–202, doi: [10.1536/ihj.14-261](https://doi.org/10.1536/ihj.14-261), indexed in Pubmed: 25740581.
- Kasprzak J, Hoffman P, Płońska E, et al. Echokardiografia w praktyce klinicznej — Standardy Sekcji Echokardiografii Polskiego Towarzystwa Kardiologicznego 2007. *Kardiol Pol*. 2007; 65: 1142–1162.
- Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015; 28(1): 1–39.e14, doi: [10.1016/j.echo.2014.10.003](https://doi.org/10.1016/j.echo.2014.10.003), indexed in Pubmed: 25559473.
- Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol*. 1986; 57(6): 450–458, doi: [10.1016/0002-9149\(86\)90771-x](https://doi.org/10.1016/0002-9149(86)90771-x), indexed in Pubmed: 2936235.
- Cuspidi C, Meani S, Negri F, et al. Indexation of left ventricular mass to body surface area and height to allometric power of 2.7: is the difference limited to obese hypertensives? *J Hum Hypertens*. 2009; 23(11): 728–734, doi: [10.1038/jhh.2009.16](https://doi.org/10.1038/jhh.2009.16), indexed in Pubmed: 19322202.
- Nagueh S, Appleton CP, Gillebert TC, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography. *Eur J of Echocardiography*. 2009; 10: 165–193, doi: [10.1093/ejechoard/jep007](https://doi.org/10.1093/ejechoard/jep007).
- Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification. *Eur J Echocardiogr* 2006; Mar. ; 7(2): 79–108, doi: [10.1016/j.euje.2005.12.014](https://doi.org/10.1016/j.euje.2005.12.014).
- Lloyd-Jones D, Adams RJ, Brown TM, et al. Executive summary: heart disease and stroke statistics—2010update: a report from the American Heart Association. *Circulation*. 2010; 121(7): 948–54, doi: [10.1161/CIRCULATIONAHA.109.192666](https://doi.org/10.1161/CIRCULATIONAHA.109.192666).
- Poirier P, Giles TD, Bray GA, et al. Obesity and cardiovascular disease. Pathophysiology, evaluation, and effect of weight loss. *Circulation*. 2006; 113: 898–918, doi: [doi:10.1161/01.ATV.0000216787.85457.f3](https://doi.org/10.1161/01.ATV.0000216787.85457.f3).
- Cuspidi C, Sala C, Tadic M, et al. Left-ventricular hypertrophy and obesity: a systematic review and meta-analysis of echocardiographic studies. *J Hypertens*. 2014; 32(1): 16–25, doi: [10.1097/HJH.0b013e328364fb58](https://doi.org/10.1097/HJH.0b013e328364fb58), indexed in Pubmed: 24309485.
- Avelar E, Cloward TV, Walker JM, et al. Left ventricular hypertrophy in severe obesity: interactions among blood pressure, nocturnal hypoxemia, and body mass. *Hypertension*. 2007; 49(1): 34–39, doi: [10.1161/01.HYP.0000251711.92482.14](https://doi.org/10.1161/01.HYP.0000251711.92482.14), indexed in Pubmed: 17130310.
- Cavalcante JL, Tamarappoo BK, Hachamovitch R, et al. Association of epicardial fat, hypertension, subclinical coronary artery disease, and metabolic syndrome with left ventricular diastolic dysfunction. *Am J Cardiol*. 2012; 110(12): 1793–1798, doi: [10.1016/j.amjcard.2012.07.045](https://doi.org/10.1016/j.amjcard.2012.07.045), indexed in Pubmed: 22980968.
- Garza CA, Pellikka PA, Somers VK, et al. Major weight loss prevents long-term left atrial enlargement in patients with morbid and extreme obesity. *Eur J Echocardiogr*. 2008; 9(5): 587–593, doi: [10.1093/ejechoard/jen117](https://doi.org/10.1093/ejechoard/jen117), indexed in Pubmed: 18490311.
- Wang TJ, Parise H, Levy D, et al. Obesity and the risk of new-onset atrial fibrillation. *JAMA*. 2004; 292(20): 2471–2477, doi: [10.1001/jama.292.20.2471](https://doi.org/10.1001/jama.292.20.2471), indexed in Pubmed: 15562125.