Effects of Bariatric Surgery on Left Ventricular Structure and Function

Eduardo Cavalcanti Lapa Santos, Maria Inês Remígio de Aguiar, Roberto de Oliveira Buril, Alvaro Antônio Bandeira Ferraz, Josemberg Marins Campos, Fernando Ribeiro de Moraes Neto, Patrícia Sampaio Gadelha

Hospital das Clínicas, Universidade Federal de Pernambuco, Recife, PE – Brazil

Abstract

Background: Obesity is associated with changes in left ventricular (LV) structure and function. Bariatric surgery can favorably improve cardiac remodeling. The effects of the procedure in LV diastolic and systolic function have not been clearly defined. The aim of this study was to evaluate the changes in left ventricular structure, systolic and diastolic function in obese patients who have undergone bariatric surgery.

Methods: We evaluated 23 patients (16 women, seven men; age, 32.9 ± 8.9 years) with body mass index > 40 kg/m² who underwent Roux-en-Y gastric bypass (RYGB). Clinical and echocardiographic evaluations were performed preoperatively and 3–7 months after surgery.

Results: After a mean follow-up of 4.7 months, significant reductions were observed in body mass index (from 46.7 ± 5.3 to 36.2 ± 4.7 kg/m²; p< 0.001); thickness of the interventricular septum (from 10.3 ± 1.4 to 8.9 ± 1.2 mm); LV posterior wall (from 9.3 ± 1.3 to 8.4 ± 1.1 mm; p < 0.001) and LV mass (absolute value: from 168.7 ± 35.2 to 149.8 ± 40.7 g, p = 0.008; indexed by height: from 45.1 ± 11.3 to 39.7 ± 10.3, p = 0.006). Normal LV geometry was observed in 60.9% of patients before surgery and in 91.3% at follow-up. Tissue Doppler imaging revealed improved LV diastolic function (mitral E′ lateral 0.16 ± 0.03 preoperatively vs. 0.17 ± 0.03 m/s at follow-up; p = 0.026). Postoperatively, there was no significant difference in LV systolic function.

Conclusions: After a mean follow-up of 4,7 months, bariatric surgery promoted improvement in left ventricular structure and in one of the parameters of diastolic function (E′ velocity). There were no changes in left ventricular systolic parameters. (Arq Bras Cardiol: Imagem cardiovasc. 2016;29(4):118-123)

Keywords: Obesity; Bariatric Surgery; Gastric Bypass; Ventricular Function, Left; Echocardiography; Diastole.

Introduction

Obesity affects cardiac structure and function. Obese patients have greater circulating blood volume, leading to increased left ventricular (LV) stroke volume and cardiac output.[1] These changes can lead to increased left ventricular (LV) mass, LV hypertrophy, diastolic and systolic dysfunction.[2-4] LV hypertrophy is one of the strongest risk factors for cardiovascular morbidity and mortality.[5]

Bariatric surgery for severe obesity is associated with decreased overall mortality.[6] Current guidelines recommend this procedure for obesity refractory to medical treatment in patients with body mass index (BMI) or with BMI ≥ 35kg/m² if the patient has other comorbidities.[7] The procedure has the potential to reverse some of the cardiac alterations caused by obesity, decreasing LV mass, reducing chamber size, and improving LV diastolic filling.[8-10]

Mailing Address: Eduardo Cavalcanti Lapa Santos •
Av. Moraes Rego, SN. Postal Code 50670-420, Cidade Universitária, Recife, PE - Brazil
E-mail: eduardolapa@gmail.com
Manuscript received March 14, 2016; revised April 25, 2016; accepted June 13, 2016.

DOI: 10.5935/2318-8219.20160031

Iimprovement in LV structure and function after bariatric surgery is not consistently observed among several studies.[2] Therefore, the purpose of this study was to evaluate the structural and functional left ventricular abnormalities in obese patients after bariatric surgery.

Materials and Methods

We conducted an observational, longitudinal, analytical, prospective, single-arm cohort study. Subjects underwent Roux-en-Y gastric bypass (RYGB) at Hospital das Clínicas da Universidade Federal de Pernambuco from May 2012 to September 2014. Inclusion criteria were BMI ≥ 40 kg/m², age > 18 years, consent to bariatric surgery, and availability of echocardiography of good technical quality. Exclusion criteria were heart failure, coronary artery disease, valvulopathy, atrial fibrillation, congenital heart disease, diabetes mellitus and chronic obstructive pulmonary disease.

Patients underwent transthoracic echocardiography before bariatric surgery and 3–7 months after the procedure, when weight, height, sex, age, and blood pressure were also recorded. Weight was determined with the patient standing, barefoot, wearing light clothing, on a digital scale. Height was measured using a calibrated wall-mounted stadiometer. BMI was calculated as weight (kg)/height (m)². A calibrated aneroid sphygmomanometer was used to determine arterial pressure using the indirect method of palpation and auscultation.

Echocardiography was performed by two experienced sonographers using a GE Vivid® *i* ultrasound system (GE Healthcare, Wauwatosa, WI, USA), with a 2.5-MHz sector-array transducer. Intra and inter-observer variability were not assessed.

Interventricular septum, posterior wall thickness, and LV end-systolic and end-diastolic diameters (LVESD and LVEDD, respectively) were measured using M-mode in the parasternal long-axis view. When optimal M-mode measurements could not be obtained, linear measurements were acquired using two-dimensional echocardiography.[11] LV endocardial fractional shortening was calculated as (LVEDD−LVESD)/LVEDD × 100%. LV ejection fraction was calculated using the Teichholz formula.[12]

LV mass was calculated using the Devereux formula.[13] LV mass was indexed to the power of 2.7, as proposed by De Simone et al.[14] LV hypertrophy was defined as LV mass/height$^{2.7}$ > 51 g/m$^{2.7}$. Relative wall thickness (RWT) was calculated as 2 × posterior wall thickness/LVEDD; > 0.42 was considered abnormal. LV geometry was categorized as one of the following four patterns: normal geometry (normal LV mass and normal RWT), concentric remodeling (normal LV mass and increased RWT), concentric hypertrophy (increased LV mass and RWT), and eccentric hypertrophy (increased LV mass and normal RWT).

Early diastolic mitral inflow velocity (E wave) and late diastolic mitral inflow velocity (A wave) were measured in apical four-chamber view using pulsed-wave Doppler. Early diastolic tissue velocity (E') was recorded over the lateral mitral annulus. E/A and E/E' ratios were calculated.

Continuous variables were expressed as mean ± standard deviation and categorical variables as frequency or percentage. The differences between categorical variables were analyzed using the Chi-square test. The Student's *t* test was used for continuous variables. The differences between continuous variables before and after bariatric surgery were analyzed using the Student's *t* test for correlated samples. Beta correlation coefficients with 95% confidence intervals were obtained using the linear regression model. All statistical tests were performed at the 5% level of significance. Data analysis was performed using SPSS for Windows, Version 15.0 (SPSS Inc., Chicago, IL, USA).

Informed consent was obtained from all individual participants included in the study. The institutional ethics committee approved the study.

Results

Twenty-three patients were enrolled in the study. Descriptive data are presented in Table 1. Most patients had hypertension (69.6%). At follow-up, significant decreases were observed in BMI ($p < 0.001$) and in systolic ($p < 0.001$) and diastolic ($p = 0.002$) blood pressures.

Table 2 presents a comparison of pre- and postoperative LV structural parameters. Preoperative echocardiograms demonstrated increased interventricular septal thickness in 14 patients (60.9 %) and increased LV mass in seven patients (30.4%), preoperatively. Echocardiography performed about 4.7 months postoperatively, revealed significant decreases in LV posterior wall thickness and RWT ($p < 0.001$ for both).

Table 3 compares pre- and postoperative LV geometry patterns. Most patients had normal LV geometry before RYGB. Concentric LV hypertrophy was the most common abnormal LV geometry, occurring in 21.5% of patients preoperatively and in none at follow-up.

LV systolic function was normal in all patients before surgery and did not change significantly during follow-up. The only significant improvement in LV diastolic function was the increase of E' velocity after surgery (Table 4).

Discussion

The present study confirms previously reported improvements in LV structure after bariatric surgery.[2]

The LV geometry pattern has prognostic implications. De Simone et al.[15] found that patients with LV hypertrophy had a 3.3-fold higher cardiac risk. In the present study, most patients (60.9 %) had normal LV geometry preoperatively. This finding conflicts with previous studies, which showed that most patients had abnormal LV geometry preoperatively.[16,17] The patients in those studies, however, were older and had a higher BMI and arterial pressure than those in the present study; in addition, 91.3 % of our patients had normal LV geometry at follow-up, indicating that reverse remodeling had occurred over the short term.

Table 1 – Demographic data

Clinical data	Preoperative	Postoperative	p value
Age (years)	32.9 (8.9)		
Female	16 (69.6%)		
BMI (kg/m^2)	46.7 (5.3)	36.2 (4.7)	< 0.001*
Weight (kg)	126.3 (25.0)	97.7 (19.0)	< 0.001*
SAP (mmHg)	138.6 (14.9)	118.8 (8.9)	< 0.001*
DAP (mmHg)	85.8 (10.4)	79.7 (6.5)	< 0.002*

** Significant value. Data presented as mean (standard deviation). BMI: body mass index; DAP: diastolic arterial pressure; SAP: systolic arterial pressure.*

Table 2 – Comparison of pre- and postoperative LV structural parameters

Structural parameters	Preoperative	Postoperative	p value
LVEDD (mm)	48.3 (3.3)	49.4 (4.4)	0.165
Septum (mm)	10.3 (1.4)	8.9 (1.2)	< 0.001*
Posterior wall (mm)	9.3 (1.3)	8.4 (1.1)	< 0.001*
RWT	0.39 (0.06)	0.34 (0.04)	< 0.001*
LVM (g)	168.7 (35.2)	149.8 (40.7)	0.008*
LVMi (g/m^2)	45.1 (11.3)	39.7 (10.3)	0.006*

** Significant value. Data presented as mean (standard deviation). LV: left ventricular; LVEDD: LV end-diastolic diameter; LVM: LV mass; LVMi: LV mass index; RWT: relative wall thickness.*

Table 3 – Effects of bariatric sugery on LV geometry

LV geometry	Preoperative	Postoperative
Normal	14 (60.9)	21 (91.3)*
Concentric remodeling	2 (8.7)	0 (0)
Concentric LV hypertrophy	5 (21.7)	0 (0)*
Eccentric LV hypertrophy	2 (8.7)	2 (8.7)*

**p < 0,05. Data expressed as n (%). LV: left ventricular.*

Table 4 – Pre- and postoperative functional parameters of the left ventricle

Functional parameter	Preoperative	Postoperative	p value
LVEF (%)	69.2 (6.8)	67.5 (5.3)	0.317
LV fractional shortening	39.1 (5.5)	37.9 (3.8)	0.404
E-wave velocity (m/s)	0.89 (0.14)	0.91 (0.15)	0.581
A-wave velocity (m/s)	0.61 (0.13)	0.57 (0.14)	0.197
E/A ratio	1.51 (0.34)	1.65 (0.46)	0.065
E' velocity	0.16 (0.03)	0.17 (0.03)	0.026*
E/E' ratio	5.95 (1.27)	5.42 (1.16)	0.083
E-wave deceleration time	188.3 (37.3)	187.1 (42.2)	0.910

** Significant value. Data presented as mean (standard deviation); LV: left ventricular; LVEF: left ventricular ejection fraction.*

No significant change in LVEDD was observed postoperatively, which is consistent with previous studies.[17,18] No patients in the present study had an abnormal LVEDD preoperatively.[19]

Increased interventricular septal thickness was the most common echocardiographic alteration detected preoperatively. Decreased LV wall thickness resulted in a decrease in RWT, LV mass, and LVMI. Before surgery, 30.4% of patients had increased RWT; after the bariatric procedure, all patients had normal RWT. Increased RWT is associated with a 2.56-fold higher cardiovascular risk in hypertensive patients.[20]

A mean reduction of 11.2% was observed in LV mass after the surgery. This was a consequence of the decreased LV wall thickness since the LV end-diastolic diameter did not change after the bariatric surgery.

LV diastolic filling is the first functional parameter to become impaired in obesity.[2] With regard to LV diastolic function, only two patients (8.7 %) had E/A ratio < 1 before surgery. Other studies reported a higher rate of this abnormality.[16,21] Cunha et al.[16] found that 17.4% of their patients had an abnormal E/A ratio and Luaces et al.[21] detected an even higher rate of 27.9%.

Some factors can explain this difference. Firstly, our population was younger (32.9 years) than those in the studies by either Cunha et al.[16] (37.9 years) or Luaces et al.[21] (40.2 years), and increasing age is associated with higher prevalence of diastolic dysfunction.[22] Secondly, diabetic patients were excluded from the present study, and diabetes is known to cause diastolic dysfunction.[22] Tavares et al.[22] showed that, in obese patients before bariatric surgery, the prevalence of diabetes was 3.6-fold higher in the subgroup with diastolic dysfunction. Thirdly, the prevalence of hypertension in our study was also lower than other series (69.6% x 82.5% in Cunha's study).[16] Finally, other studies performed echocardiography only in patients with multiple cardiovascular risk factors or with abnormal electrocardiogram,[23] whilst in the present study all patients enrolled had the test done, irrespective of cardiac risk factors.

There was a trend towards increasing the E/A ratio after surgery but it didn't reach statistical significance ($p = 0.06$). Other studies that showed a significant increase in this parameter had a longer follow-up,[16,18,24] so a longer follow-up in the present study may have revealed increased E/A ratio.

E' velocity is particularly useful in evaluation after bariatric surgery because it is a preload-independent index of LV relaxation.[25,26] This parameter is easy to obtain in obese patients[1] and has prognostic value, with results below the normal range predicting higher cardiovascular mortality.[27] None of the patients showed decreased lateral E' velocities (<10 cm/s) before surgery. Other studies found impaired E' velocities before bariatric surgery.[22,28] Willens et al.[28] found a mean E' velocity of 7.6 cm/s before bariatric surgery, though their patients had higher BMI (54 kg/m^2), higher systolic blood pressure (145 mmHg) and a prevalence of 29.4% of diabetes.

In the present study, E' velocity increased 6.25 % postoperatively. Others have observed significant increases in this parameter after bariatric surgery.[10,17,28]

The E/E' ratio can be used as an index of LV filling pressures.[25] Values below 8 imply normal filling pressures in patients with preserved ejection fraction.[29] All patients in the present study had E/E' < 8 before surgery.

The E/E' ratio tended to decrease but it did not reach statistical significance. The behavior of this parameter after bariatric surgery is heterogeneous among studies. Luaces et al.[30] found no change 12 months after bariatric surgery, but others have observed significant decreases in the E/E' ratio after surgery: Willens et al.,[28] from 12.3 to 10 after a mean follow-up of 7.4 months; and Ippisch et al.,[10] from 7.7 to 6.3 after 10 months of postoperative follow-up. Compared with the present study, follow-up periods were longer, and preoperative E/E' ratios higher, suggesting higher diastolic filling pressures.

Regarding to LV systolic function, none of the patients had reduced LV ejection fraction before surgery since this was an exclusion criterion for the study. This parameter did not change after the bariatric surgery. This is consistent with the findings of other investigators.[30,31] Garza et al.[9] observed similar results, even in the subgroup of patients with reduced LV ejection fraction before surgery.

The results of our study should be interpreted in the context of some limitations. The sample size was small. There was no control group of medically managed obese patients. The study was observational and the decisions regarding patient management were made by the attending physicians. Only patients with grade 3 obesity were included; patients with diabetes were not included. Intra and inter-observer variability of echocardiographic measurements was not assessed.

Conclusions

After a mean follow-up of 4-7 months, bariatric surgery promoted improvement in left ventricular structure and in one of the parameters of diastolic function (E' velocity). There were no changes in left ventricular systolic parameters.

Authors' contribution

Research creation and design: Santos ECL, Aguiar MIR, Moraes Neto FR; Data acquisition: Santos ECL, Aguiar MIR, Buril RO; Data analysis and interpretation: Santos ECL, Gadelha PS; Statistical analysis: Santos ECL; Manuscript drafting: Santos ECL, Moraes Neto FR, Gadelha PS; Critical review of the manuscript as to important intellectual content: Santos ECL, Ferraz AAB, Campos JM, Gadelha PS.

Potential Conflicts of Interests

I declare that this study has no relevant conflicts of interests.

Sources of Funding

This study had no external funding sources.

Academic Association

This manuscript is an integral part of the master's dissertation of Eduardo Cavalcanti Lapa Santos from the Graduate Program in Surgery Studies of Universidade Federal de Pernambuco.

References

1. Kardassis D, O, Schönander M, Sjöström L, Petzold M, Karason K. Impact of body composition, fat distribution and sustained weight loss on cardiac function in obesity. Int J Cardiol. 2012,159(2):128-33.
2. Grapsa J, Tan TC, Paschou S a, Kalogeropoulos AS, Shimony A, Kaier T, et al. The effect of bariatric surgery on echocardiographic indices: a review of the literature. Eur J Clin Invest. 2013;43(11):1224–30.
3. Lakhani M, Fein S. Effects of obesity and subsequent weight reduction on left ventricular function. Cardiol Rev. 2011;19(1):1–4.
4. Zarich SW, Kowalchuk GJ, McGuire MP, Benotti PN, Mascioli EA, Nesto RW. Left ventricular filling abnormalities in asymptomatic morbid obesity. Am J Cardiol. 1991;68(4):377–81.
5. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med. 1990;322(22):1561–6.
6. Sjostrom L, Narbro K, Sjostrom D, Kararson K, Larsson B, Wedel H, et al. Effects of Bariatric Surgery on Mortality in Swedish Obese Subjects. N Engl J Med. 2007;357(8):741–52.
7. Mechanick JI, Kushner RF, Sugerman HJ, Gonzalez-Campoy JM, Collazo-Clavell ML, Spitz AF, et al. American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery medical guidelines for clinical practice for

the perioperative nutritional, metabolic, and nonsurgical support of the bariatric . Obesity. 2009; 17(Suppl 1):S1-70.

8. Ikonomidis I, Mazarakis A, Papadopoulos C, Patsouras N, Kalfarentzos F, Lekakis J, et al. Weight loss after bariatric surgery improves aortic elastic properties and left ventricular function in individuals with morbid obesity: a 3-year follow-up study. J Hypertens. 2007;25(2):439–47.

9. Garza CA, Pellikka PA, Somers VK, Sarr MG, Collazo-Clavell ML, Korenfeld Y, et al. Structural and functional changes in left and right ventricles after major weight loss following bariatric surgery for morbid obesity. Am J Cardiol.2010;105(4):550–6.

10. Ippisch HM, Inge TH, Daniels SR, Wang B, Khoury PR, Witt S, et al. Reversibility of cardiac abnormalities in morbidly obese adolescents. J Am Coll Cardiol. 2008;51(14):1342–8.

11. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's. Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiograph. J Am Soc Echocardiogr. 2005; 18(12):1440–63.

12. Teichholz LE, Herman V. Problems in echocardiographic presence or absence volume determinations : correlations in the of asynergy. Am J Cardiol.1976;37(1):7-11.

13. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, et al. Echocardiographic Assessment of Left Ventricular Hypertrophy: comparison to necropsy finding. Am Heart J. 1986;57(5):450–8.

14. De Simone G, Daniels SR, Devereux RB, Meyer RA, Roman MJ, de Divitiis O, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. J Am Coll Cardiol. 1992;20(5):1251-60.

15. De Simone G, Devereux RB, Daniels SR, Koren MJ, Meyer RA, Laragh JH. Effect of Growth on Variability of Left Ventricular Mass : assessment of allometric signals in adults and children and their capacity to predict cardiovascular risk. J Am Coll Cardiol. 1995;25(5):1056–62.

16. Cunha L de CB, Cunha CL, de Souza AM, Chiminacio Neto N, Pereira RS, Suplicy HL. Evolutive echocardiographic study of the structural and functional heart alterations in obese individuals after bariatric surgery. Arq Bras Cardiol. 2006;(87):562–8.

17. Hsuan CF, Huang CK, Lin JW, Lin LC, Lee TL, Tai CM, et al. The effect of surgical weight reduction on left ventricular structure and function in severe obesity. Obesity. 2010;18(6):1188-93.

18. Kanoupakis E, Michaloudis D, Fraidakis O, Parthenakis F, Vardas P, Melissas J. Left ventricular function and cardiopulmonary performance following surgical treatment of morbid obesity. Obes Surg. 2001;11(5):552-8.

19. Alpert M,Terry BE, Kelly DL. Effect of weight loss on cardiac chamber size, wall thickness and left ventricular function in morbid obesity. Am J Cardiol. 1985;55(6):783-6.

20. Verdecchia P, Schillaci G, Borgioni C, Ciucci a, Battistelli M, Bartoccini C, et al. Adverse prognostic significance of concentric remodeling of the left ventricle in hypertensive patients with normal left ventricular mass. J Am Coll Cardiol. 1995;25(4):871-8.

21. Luaces M, Cachofeiro V, García-Muñoz-Najar A, Medina M, González N, Cancer E, et al.Anatomical and functional alterations of the heart in morbid obesity. Changes after bariatric surgery. Rev Española Cardiol. 2012;65(1):1-3.

22. Tavares IS, Sousa AC, Menezes Filho RS, Oliveira MA, Barreto Filho JÁ, et al.Left ventricular diastolic function in morbidly obese patients in the preoperative for bariatric surgery. Arq Bras Cardiol. 2011;98(4):300–6.

23. Cavarretta E, Casella G, Calì B, Dammaro C, Biondi-Zoccai G, Iossa A, et al. Cardiac remodeling in obese patients after laparoscopic sleeve gastrectomy. World J Surg. 2013;37(3):565-72.

24. Valezi AC, Machado VHS. Morphofunctional evaluation of the heart of obese patients before and after bariatric surgery. Obes Surg. 2011;21(11):1693-7.

25. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quin MA. Doppler tissue imaging : anoninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol. 1997;30(6):1527–33.

26. Sohn DW, Chai IH, Lee DJ, Kim HC, Kim HS, O BH, et al. Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular diastolic function. J Am Coll Cardiol. 1997;30(2):474–80.

27. Wang M, Yip GW, Wang AY, Zhang Y, Ho PY, Tse MK, et al. Peak early diastolic mitral annulus velocity by tissue Doppler imaging adds independent and incremental prognostic value. J Am Coll Cardiol. 2003;41(5):820-6.

28. Willens HJ, Chakko SC, Byers P, Chirinos J, Labrador E, Castrillon JC, et al. Effects of weight loss after gastric bypass on right and left ventricular function assessed by tissue Doppler imaging. Am J Cardiol. 2005;95(12):1521-4.

29. Nagueh SF, Appleton CP, Gillebert TC, Marino PN, Oh JK, Smiseth O, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. Eur J Echocardiogr. 2009;10(2):165-93.

30. Luaces M, Martínez E, Medina M, Miana M, González N, Fernández-Pérez C, et al. The impact of bariatric surgery on renal and cardiac functions in morbidly obese patients. Nephrol Dial Transpl. 2012;27(Suppl 4):iv 53–7.

31. Leichman JG, Wilson EB, Scarborough T, Aguilar D, Miller CC, Yu S, et al. Dramatic reversal of derangements in muscle metabolism and left ventricular function after bariatric surgery. Am J Med. 2008;121(11):966-73.