Anatomical and Functional Alterations of the Heart in Morbid Obesity. Changes After Bariatric Surgery

María Luaces, a, * Victoria Cachofeiro, b Alejandro García-Muñoz-Najar, c Manuel Medina, c Noemi González, d Emilia Cancer, d Azucena Rodríguez-Robles, d Gloria Cánovas, d and Alfonso Antequera-Pérez c

a Servicio de Cardiología, Hospital Universitario de Fuenlabrada, Fuenlabrada, Madrid, Spain
b Departamento de Fisiología, Facultad de Medicina, Universidad Complutense de Madrid, Madrid, Spain
c Servicio de Endocrinología-Medicina Interna, Hospital Universitario de Fuenlabrada, Fuenlabrada, Madrid, Spain

d Servicio de Cardiología, Hospital Universitario de Fuenlabrada, Fuenlabrada, Madrid, Spain

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Abstract

Introduction and objectives: Cardiac adaptation to obesity includes both structural and functional changes in the heart. The therapeutic option of last resort for morbidly obese patients is bariatric surgery. This study aims to assess the anatomical functional changes in the heart for a Spanish cohort of morbidly obese patients, as well as changes after bariatric surgery.

Methods: Patients referred for bariatric surgery were prospectively included. In each case, a transthoracic echocardiography, electrocardiogram, and blood tests were performed before the procedure and repeated 1 year after surgery.

Results: Forty-one patients completed the 1-year follow-up. Of these, 82.9% were female. Mean age was 40.2 ± 6.6 years. Prior to surgery, mean body mass index was 47.41 kg/m², decreasing to 30.43 kg/m² after the procedure. Before surgery, cardiac remodeling was present in 70.7%, most frequently in the form of eccentric hypertrophy (34.1%). At 1-year follow-up, 58.5% showed a normal left ventricular geometric pattern (P = .02). Mitral inflow E/A ratio changed from 1.14 to 1.43 (P < .001). Nevertheless, early mitral velocity measured by Doppler tissue decreased (P = .06).

Conclusions: In morbidly obese patients referred for bariatric surgery, cardiac remodeling is highly prevalent, in most cases in an eccentric manner. Weight loss achieved by bariatric surgery is accompanied by significant improvements in left ventricular structure. Nevertheless, the damage in diastolic function may be permanent despite weight loss.

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Modificaciones anatómofuncionales del corazón en la obesidad mórbida. Cambios tras la cirugía bariátrica

Resumen

Introducción y objetivos: La adaptación cardiaca a la obesidad asocia anomalías estructurales y funcionales del corazón. El último escalamiento terapéutico en la obesidad mórbida lo ofrece la cirugía bariátrica. En este estudio se investigan los cambios anatómofuncionales del corazón en una cohorte española de obesos mórbidos y las modificaciones después de la cirugía bariátrica.

Métodos: Inclusión prospectiva de pacientes referidos para cirugía bariátrica. En cada caso se realizó ecocardiograma transtorácico, electrocardiograma y analítica antes de la cirugía y 1 año después de la intervención.

Resultados: Alcanzaron el seguimiento a 1 año 41 pacientes, con media de edad de 40.2 ± 9.6 años, el 82.9% mujeres. El índice de masa corporal promedio era 47.41, y pasó a 30.43 después de la cirugía. Antes de la cirugía, el 70.7% presentaba remodelado ventricular, fundamentalmente por hiperтроfia excéntrica (el 34.1% de los casos). Al año, el 58.5% tenía un patrón geométrico normal (P = .02). La relación E/A de llenado mitral pasó de 1.14 a 1.43 (P < .001). Sin embargo, la velocidad del anillo mitral medida con Doppler tissue descendió ligeramente (P = .06).

Conclusiones: Los pacientes obesos mórbidos que se someten a cirugía bariátrica presentan con elevada frecuencia datos de remodelado ventricular, que es fundamentalmente de características excéntricas. La pérdida de peso conseguida mediante cirugía bariátrica se acompaña de significativas mejoras estructurales y en muchos casos lleva a la normalización completa del patrón geométrico ventricular. Sin embargo, es posible que la disfunción diastólica llegue a ser permanente.

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INTRODUCTION

Obesity has become a global problem of the first magnitude. Currently, 16.4% of the Spanish population is obese, and 1 in every 10 adults worldwide is obese. Weight loss is one of the pillars upon which any strategy towards improving the cardiovascular profile is based. Excess adiposity and obesity are risk factors for death, and there is an established relationship between obesity and comorbidities such as hypertension, diabetes mellitus, and even cancer. Obesity is a risk factor independent of left ventricular hypertrophy, risk factors for coronary disease, and death from any other cause. Heart disorders in obesity include structural adaptation with left ventricular hypertrophy, ventricular and atrial dilatation, and functional abnormalities, with diastolic dysfunction and, potentially, congestive heart failure. Bariatric surgery is the only solution for patients for whom all other weight loss strategies have failed. It has been demonstrated that this technique achieves a stable and lasting weight loss of up to 50% to 60% of excess weight. Gastric bypass technique offers the better risk-benefit balance. The SOS study showed a reduction in mortality for all causes in the 15-year monitoring of patients who underwent surgical treatment, compared to medical and dietary treatment. Previous studies have also shown that both the mass and thickness of the left ventricle (LV) is reduced after weight loss achieved with bariatric surgery. However, despite the various descriptions available in the literature of heart disease in obesity, there is little information about how the various forms of adaptation respond to bariatric surgery or the expected result, depending on the preoperative situation. Taking these considerations into account, we have carried out this study with a two-fold objective: firstly, to describe the ventricular geometric pattern and cardiac function in patients referred to bariatric surgery; and secondly, to analyze the changes that occur in each type of adaptive pattern after weight loss achieved with bariatric surgery, in order to obtain information that will provide new criteria in the future for selecting the best candidates for this procedure, and thus optimize results.

METHODS

Patient Group

This clinical study consecutively included all patients referred for bariatric surgery. The selection of candidates was performed by a committee that included the endocrinology, general and digestive system surgery, internal medicine, and cardiology departments. Inclusion criteria were age ≥ 18 years and universally accepted indications: long-term obesity (more than 4 years); body mass index (BMI) ≥ 40 despite other weight loss strategies or BMI ≥ 35 in the presence of obesity-related comorbidities (diabetes mellitus, obesity hypventilation syndrome, obstructive sleep apnea syndrome, hypertension). Exclusion criteria were age ≥ 60 years and unacceptable surgical risk due to concomitant comorbidities.

After inclusion by the committee, the patients underwent a cardiovascular examination that included physical examination, 12-lead electrocardiogram (ECG), and transthoracic echocardiogram. The preoperative examination also included anthropometric measurements and blood tests. The protocol was approved by the ethics committee, and all patients signed the informed consent. Following this protocol, 63 patients were included, starting in 2007 (Fig. 1). Four patients were excluded for various reasons (psychiatric decompensation, failure to quit smoking, etc.). The remaining 57 underwent surgery and continued monitoring. This study comprises 41 patients who completed 1 year of monitoring. Patients who completed their monitoring were compared to those who did not. There were no statistically significant differences in clinical and baseline echocardiographic variables.

Anthropometric Measurements

The height and weight of patients was recorded when taking the echocardiogram, with the patients wearing light clothing without shoes. We used wall scales. After 10 min rest in a sitting position, a 12-lead ECG was taken and blood pressure was measured in the nondominant arm. BMI was calculated according to the formula: weight (kg)/height squared (meters).

Patients were classified as follows according to BMI: normal weight, BMI < 25; overweight, BMI 25–<27; mild obesity, BMI 27–<30; grade I or moderate obesity, BMI 30–<35; grade 2 or severe obesity, BMI 35–<40; grade 3 or morbid obesity, BMI 40–<50; super obesity, BMI 50–<60; and super morbid obesity, BMI ≥ 60.

The percentage of excess weight lost was calculated according to the formula:

\[
\text{Percentage of excess weight lost(%) = } \frac{\text{initial weight} - \text{current weight}}{\text{initial weight} - \text{ideal weight}} \times 100
\]
Echocardiogram

Echocardiography studies were performed using a commercially available unit Vivid 1® (GE Healthcare, Waukesha, WI, USA) equipped with a 2.5 MHz probe. Transthoracic echocardiography study was performed according to the recommendations of the European Society of Echocardiography. The mass of the LV in grams was calculated using the Devereaux et al. formula:

\[ \text{Mass of the LV (g)} = 0.81 (1.04 ([\text{LVTDD} + \text{dIVS} + \text{dPW}]^3 - \text{LVTDD}^3)) \times 0.6 \]

Where LVTDD is the LV telediastolic diameter.

The LV mass was indexed to the power of 2.7 to minimise the interference of obesity in the estimate of ventricular mass. The relative parietal thickness (RPT) was calculated using the formula:

\[ \text{RPT} = \frac{(\text{dIVS} + \text{dPW})}{\text{LVTDD}} \]

An indexed value for LV mass ≥51 g/m², for both males and females, and RPT ≥0.45 were considered as defining values for concentric hypertrophy.

According to the RPT and the indexed mass for the LV, 4 LV geometric patterns were defined (Fig. 2):

1. Normal: RPT <0.45 and an indexed LV mass <51 g/m².
2. Concentric remodeling: RPT ≥0.45 and an indexed LV mass <51 g/m².
3. Concentric hypertrophy: RPT ≥0.45 and an indexed LV mass ≥51 g/m².
4. Eccentric hypertrophy: RPT <0.45 and an indexed LV mass ≥51 g/m².

The change in the LV geometric pattern was divided into three levels, depending on the variations in the indexed mass of the LV and the RPT: no change, improvement and worsening.

The myocardial performance index and the volume of the left atrium (LA) was calculated according to validated formulas. Doppler analysis was performed according to standard recommendations. For each value, the average of the measurements for 3 consecutive cardiac cycles was calculated. The assessment of echocardiographic measurements was performed by a single observer under masked conditions. All measurements were performed in a post-processing workstation EchoPAC® (GE Healthcare, Waukesha, WI, USA).

Laboratory Tests

Blood samples were obtained following the clinical protocol approved for bariatric surgery.

Monitoring

Monitoring was performed according to the approved protocol, under clinical and periodic laboratory analyses. The cardiology follow-up visit was performed after 1 year, with new transthoracic echocardiograms, ECG, and measurements of blood pressure during the same visit.

Data Analysis

Continuous variables are expressed as mean ± standard deviation or median (interquartile interval) in case of asymmetry. Categorical variables are expressed in absolute values and percentages. The differences between categorical variables were analyzed using the chi-square test. The Student’s t-test or the ANOVA test was used for continuous variables. Normality of distributions was verified by means of the Kolmogorov-Smirnov test. Differences in distributions of LV geometric patterns were analyzed using McNemar’s test x2 for correlated samples. The differences between continuous variables before and after bariatric surgery were analyzed using the Student’s t-test for correlated samples or the corresponding nonparametric test, as appropriate. Using the “improvement” category of the LV geometric pattern as a reference, we calculated the effects on the clinical variables through the coefficients of a linear regression model that included the preoperative baseline value of the same variable (ANCOVA). The beta correlation coefficients, along with their 95% confidence intervals (95%CI), were obtained using a linear regression model. A value of P<.05 was used as the cut-off value for defining statistical significance. Data analysis was performed using the statistical program SPSS version 15.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Preoperative Situation: Baseline Characteristics

Epidemiological Data

The mean age of the patient group was 40.2 ± 9.6 (23-60) years. Of the 41 patients, 34 (82.9%) were women. Average BMI prior to bariatric surgery was 47.4 ± 5 (range, 39.6-62.8) (Table 1).

Some 87.8% of patients had associated comorbidities. The most frequent of these were hypertension (36.5%), dyslipidemia (36.6%), and smoking (34.1%). Only 4.9% of patients were diabetic. Some 12.2% presented hypoventilation syndrome.

Metabolic Profile and Cardiovascular Risk

In the preoperative study, mean systolic blood pressure was 131 ± 14.6 mmHg, mean diastolic blood pressure was 84.5 ± 16.83 mmHg, and heart rate was 75.7 ± 7.5 bpm. With regard to the metabolic profile, glycemia was 98.3 ± 19.5 mg/dl, low-density lipoprotein (LDL) cholesterol was 123.60 ± 31.59 mg/dl, high-density lipoprotein (HDL) cholesterol was 47.50 ± 13.30 mg/dl and triglyceride levels were 148.21 ± 90.02 mg/dl.
Table 1
Epidemiological Data and Clinical Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preoperative</th>
<th>Postoperative (1 year)</th>
<th>Difference (95%CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>40.2 ± 9.6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>34 ± 8.29</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.61 ± 0.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>123.7 ± 19.7</td>
<td>79.19 ± 15.52</td>
<td>−44.58 (−49.39 to −39.71)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>BMI</td>
<td>47.41 ± 5</td>
<td>30.43 ± 5.47</td>
<td>−16.97 (−18.64 to −15.31)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>131 ± 14.64</td>
<td>111.95 ± 12.64</td>
<td>−19.04 (−23.52 to −14.56)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>84.58 ± 16.83</td>
<td>65.73 ± 8.7</td>
<td>−18.85 (−24.87 to −12.83)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>75.7 ± 7.5</td>
<td>66.32 ± 9.04</td>
<td>−9.5 (−12.54 to −6.32)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Glycemia (mg/dl)</td>
<td>99.36 ± 19.56</td>
<td>84.53 ± 13.64</td>
<td>−14.82 (−19.48 to −10.17)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>196.09 ± 32.86</td>
<td>164.58 ± 32.21</td>
<td>−31.51 (−43.08 to −19.93)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LDLC (mg/dl)</td>
<td>123.6 ± 31.59</td>
<td>100.08 ± 23.28</td>
<td>−23.52 (−35.62 to −14.42)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HDLC (mg/dl)</td>
<td>47.5 ± 13.3</td>
<td>52.75 ± 12.78</td>
<td>5.25 (−9.1 to −1.39)</td>
<td>.009</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>148.21 ± 90.02</td>
<td>82.56 ± 34.15</td>
<td>−65.65 (−88.63 to −44.57)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

95%CI: 95% confidence interval; BMI: body mass index; DBP: diastolic blood pressure; HDLC: high-density lipoprotein cholesterol; HR: heart rate; LDLC: low-density lipoprotein cholesterol; SBP: systolic blood pressure.

Data are expressed as mean ± standard deviation, mean (95%CI) or no. (%).

Echocardiography Findings

Data obtained from the echocardiography study is shown in Table 2.

Structural Characteristics

The LVTDD was 4.9 ± 0.5 cm and LVTSD was 3 ± 0.4 cm. Average thickness of the interventricular septum was 1.05 ± 0.1 cm, and 1 ± 0.1 cm for the posterior wall. The indexed ventricular mass was 51.4 ± 11.2 g/m² and the relative parietal thickness was 0.42 ± 0.09 cm.

Average volume of the LA was 50.4 ± 14.1 ml and the average area of the LA was 17.9 ± 3.2 cm². The indexed body surface resulted in an average of 22.8 ± 6.3 ml/m², while normalization by height resulted in an average value of 31.3 ml/m.

Myocardial Function

Average left ventricle ejection fraction (LVEF) was 65.7 ± 7.2%, and average shortening fraction was 37.3 ± 6%. Volume per beat was 63.8 ± 20.4 ml and cardiac output was 4.78 ± 1.5 L/min, with a cardiac index of 2.1 ± 0.6 L/min/m².

Diastolic Function

The average value for E/A was 1.1 ± 0.3, and 34.1% of patients had an E/A < 1. The mean mitral deceleration time was 151.5 ± 26.7 ms. Using Doppler tissue imaging for mitral anulus velocity, the average e’-wave was 9.9 ± 3.5 and 8.8 ± 2.8 for a’-wave. Isovolumetric relaxation time of LV was 84.1 ± 19 ms.

Left Ventricle Geometry

Before intervention, 29.3% of patients had normal LV geometric patterns (Table 3). The remaining 70.7% had some form of ventricular remodeling, the most frequent being eccentric hypertrophy (which was present in 34.1% of patients) and concentric hypertrophy (in 19.5% of cases).

Relationship Between Left Ventricle Geometric Pattern and Baseline Characteristics

We studied the relationship between the LV geometric pattern and the clinical, epidemiological, and echocardiographic variables that may have been related to it (Table 4). In this regard, we found no statistically significant differences between the systolic and diastolic blood pressure readings in the various patient groups. The average age of patients increased progressively with various remodeling patterns, the average age for the group with normal geometric patterns being 36.7 ± 8.5 years, and 46.5 ± 8.33 years for the concentric hypertrophy group (P = .13). As for the echocardiographic findings, patients with an eccentric hypertrophy pattern were found to have lower LVEF (62.8%, P = .14).

Post-surgical Results. Follow-up at One Year

One year after the surgery, average BMI was 30.43 ± 5.47. The percentage of lost excess weight was, on average, 63.31 ± 20.10%.

Post-surgical Metabolic Profile and Cardiovascular Risk

Mean glycemia 1 year after surgery lowered to 84.5 ± 13.6 mg/dl (P < .001) (Table 1). The other components of the metabolic profile also experienced significant changes: average total cholesterol was reduced to 164.5 ± 32.2 mg/dl (P < .001), LDL cholesterol to 100 ± 23.2 mg/dl (P < .001) and triglycerides to 82.5 ± 34.1 mg/dl (P < .001). In addition, LDL cholesterol increased to 52.7 ± 12.7 mg/dl (P = .009). Systolic blood pressure decreased by 19 mmHg (95%CI, 14.5-23.5) (P < .001) and diastolic blood pressure underwent an average decrease of 18.8 mmHg (95%CI, 12.8-24.8) (P < .001). Furthermore, average heart rate decreased from 75.8 ± 7.6 bpm to 66.3 ± 9 bpm (P < .001).

Post-surgical Echocardiography Findings

Structural Characteristics

We did not detect statistically significant changes in ventricular diameters 1 year after surgery (Table 2). However, we did
consistently observe a decrease in parietal thickness, so that the thickness of the septum went from 1.05 ± 0.16 cm to 0.94 ± 0.18 cm (P = .003) and the posterior wall thickness went from 1 ± 0.16 cm to 0.93 ± 0.18 cm (P = .04). RPT changed from 0.42 ± 0.09 to 0.38 ± 0.06 (P = .01). Indexed ventricular mass lowered from 51.4 ± 11.2 to 46.22 ± 17.1 kg/m² although the difference was not statistically significant, the values obtained 1 year after surgery were significantly greater than the baseline values, and were clearly higher at 1 year when normalized for body surface.

**Myocardial Function**

Systolic function parameters maintained values comparable to those measured before surgery, both in terms of LVEF and shortening fraction.

**Table 2**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preoperative</th>
<th>Postoperative (1 year)</th>
<th>Difference (95%CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVTDD (cm)</td>
<td>4.9 ± 0.56</td>
<td>4.9 ± 0.47</td>
<td>0 (0.21 to 0.2)</td>
<td>.96</td>
</tr>
<tr>
<td>LVTSD (cm)</td>
<td>3.06 ± 0.47</td>
<td>3.16 ± 0.44</td>
<td>0.09 (0.10 to 0.29)</td>
<td>.34</td>
</tr>
<tr>
<td>Septum thickness (cm)</td>
<td>1.05 ± 0.16</td>
<td>0.94 ± 0.18</td>
<td>-0.11 (-0.19 to -0.04)</td>
<td>.003</td>
</tr>
<tr>
<td>Posterior wall thickness (cm)</td>
<td>1 ± 0.16</td>
<td>0.93 ± 0.18</td>
<td>-0.07 (0 to 0.15)</td>
<td>.04</td>
</tr>
<tr>
<td>Relative parietal thickness</td>
<td>0.42 ± 0.09</td>
<td>0.38 ± 0.06</td>
<td>-0.04 (-0.08 to -0.01)</td>
<td>.01</td>
</tr>
<tr>
<td>Indexed LV mass (g/m²)</td>
<td>51.4 ± 1.12</td>
<td>46.22 ± 1.78</td>
<td>-5.17 (-10.53 to -0.53)</td>
<td>.07</td>
</tr>
<tr>
<td>LA area (cm²)</td>
<td>17.94 ± 3.29</td>
<td>19.05 ± 4.76</td>
<td>1.11 (0.63 to 2.86)</td>
<td>.2</td>
</tr>
<tr>
<td>LA volume (ml)</td>
<td>50.47 ± 14.16</td>
<td>52.9 ± 18.4</td>
<td>2.42 (3.67 to 8.62)</td>
<td>.43</td>
</tr>
<tr>
<td>Indexed LA volume (ml/m²)</td>
<td>31.31 ± 8.78</td>
<td>32.83 ± 11.47</td>
<td>1.52 (2.38 to 5.33)</td>
<td>.42</td>
</tr>
<tr>
<td>Indexed LA volume by body surface (ml/m²)</td>
<td>22.84 ± 6.3</td>
<td>28.88 ± 9.56</td>
<td>6.03 (2.98 to 9.09)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

**Systolic function parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preoperative</th>
<th>Postoperative (1 year)</th>
<th>Difference (95%CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF (%)</td>
<td>65.76 ± 7.28</td>
<td>65.07 ± 7.29</td>
<td>-0.53 (-3.3 to -2.24)</td>
<td>.7</td>
</tr>
<tr>
<td>SF (%)</td>
<td>37.39 ± 6.07</td>
<td>35.57 ± 7</td>
<td>-0.18 (-0.45 to -0.08)</td>
<td>.17</td>
</tr>
<tr>
<td>Volume/beat (ml)</td>
<td>63.85 ± 20.45</td>
<td>63.56 ± 13.86</td>
<td>-0.28 (-5.36 to 5.93)</td>
<td>.91</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>4.78 ± 1.51</td>
<td>4.25 ± 0.88</td>
<td>-0.53 (-1.03 to 0.03)</td>
<td>.03</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>2.18 ± 0.62</td>
<td>2.36 ± 0.52</td>
<td>0.18 (0.046 to 0.4)</td>
<td>.11</td>
</tr>
</tbody>
</table>

**Diastolic function parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preoperative</th>
<th>Postoperative (1 year)</th>
<th>Difference (95%CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>E-wave (cm/s)</td>
<td>77.25 ± 17.16</td>
<td>90.23 ± 14.06</td>
<td>12.98 (7.72 to 18.34)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>A-wave (cm/s)</td>
<td>72.89 ± 17.09</td>
<td>69.13 ± 23.41</td>
<td>-3.69 (-2.49 to 9.98)</td>
<td>.23</td>
</tr>
<tr>
<td>e’-wave (cm/s)</td>
<td>9.95 ± 3.5</td>
<td>9 ± 2.64</td>
<td>-1.05 (-2.16 to -0.05)</td>
<td>.06</td>
</tr>
<tr>
<td>a’-wave (cm/s)</td>
<td>8.87 ± 2.81</td>
<td>8.07 ± 2.37</td>
<td>-0.84 (-2.09 to -0.4)</td>
<td>.17</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.14 ± 0.38</td>
<td>1.43 ± 0.41</td>
<td>0.29 (0.16 to 0.41)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>E/e’ ratio</td>
<td>8.71 ± 3.38</td>
<td>11.24 ± 4.72</td>
<td>2.47 (0.93 to 4.01)</td>
<td>.003</td>
</tr>
<tr>
<td>Myocardial performance index</td>
<td>0.46 ± 0.09</td>
<td>0.45 ± 0.08</td>
<td>0 (-0.02 to 0.04)</td>
<td>.66</td>
</tr>
<tr>
<td>Isovolumetric relaxation time for LV (ms)</td>
<td>84.19 ± 19.06</td>
<td>77.18 ± 20.21</td>
<td>-7.00 (-41.1 to 18.11)</td>
<td>.2</td>
</tr>
<tr>
<td>Mitral filling propagation velocity (cm/s)</td>
<td>67.13 ± 21.2</td>
<td>60.21 ± 17.47</td>
<td>-6.37 (-16.22 to 3.47)</td>
<td>.19</td>
</tr>
<tr>
<td>E/MPV ratio</td>
<td>1.3 ± 0.55</td>
<td>1.6 ± 0.47</td>
<td>0.27 (-0.01 to 0.55)</td>
<td>.06</td>
</tr>
</tbody>
</table>

95%CI, 95% confidence interval; LA, left atrium; LV, left ventricle; LVEF, left ventricle ejection fraction; LVTDD, LV telediastolic diameter; LVTSD, LV telesystolic diameter; MVP, mean platelet volume; SF, shortening fraction.

The data express mean ± standard deviation or mean (95%CI).

**Table 3**

<table>
<thead>
<tr>
<th>Before surgery</th>
<th>After surgery (1 year)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Concentric remodeling</td>
</tr>
<tr>
<td>Normal</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Concentric remodeling</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Concentric hypertrophy</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Eccentric hypertrophy</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>24 (58.5)</td>
<td>2 (4.9)</td>
</tr>
</tbody>
</table>

P = .02.

Data are expressed as no. (%).
Regarding hemodynamics, the volume/beat ratio was almost the same as before surgery: 63.5 ± 13.8 ml/min. Cardiac output, however, decreased significantly from 4.7 ± 1.5 L/min/m² to 4.2 ± 0.8 L/min/m² (P = .03). The cardiac index increased, although the difference was not statistically significant.

**Diastolic Function**

Pulsed-wave Doppler E’-wave for mitral filling increased by 12.9 cm/s (95%CI, 7.6–18.3) (P < .001). The E/A ratio improved from 1.1 ± 0.3 to 1.43 ± 0.4 (P < .001) and mitral deceleration time increased on average by 13.5 ms (P = .01). Mitral annulus velocities by Doppler tissue imaging, both early and late, showed slight decreases but were not statistically significant. Consistent with the above, the E/e’ ratio went from 8.77 ± 3.4 to 11.2 ± 4.7 (P = .003).

**Left Ventricle Geometry**

Changes observed in LV geometry are shown in Table 3. After 1 year, 58.5% of patients had a normal pattern (P = .02). Pattern improvement occurred in 21 of the 41 patients; 16 had no changes and 4 experienced a worse pattern (3 cases due to eccentric hypertrophy and 1 case due to concentric hypertrophy). The geometric pattern completely normalized in 15 patients, the LV mass improved in 11 patients, and 13 patients had improved relative parietal thickness.

We analyzed the relationship between the change in geometric pattern and the clinical and echocardiographic variables studied, with the intent of identifying substantial differences between patients in relation to geometric changes experienced (Table 5). The average age of the 4 patients who experienced worsening of the geometric pattern was 44.75 ± 13.6 years, versus 38.5 ± 8.84 years for those who achieved normalization (P = .44). The vast majority of studied parameters showed similar changes in the various patient groups, except for the case of LDL cholesterol, whose reduction was more pronounced in the group with worse geometric outcome (P = .07).

**DISCUSSION**

We present the first series in Spain of patients in whom ventricular structure and mechanics have been analyzed before and after bariatric surgery as the definitive procedure for weight loss in morbid obesity. The data describe Spain’s population profile, which is epidemiologically and anthropometrically different from other European and American series. Patients in our series are young, mostly female, and have grade III obesity, while super obesity (BMI > 50) is more prevalent in the American and Central European populations.43

Our results show a clear benefit from weight loss achieved through bariatric surgery in terms of LV structure and geometry. Special attention has been paid to the change in LV geometric pattern as a structural assessment parameter, not just to mass and parietal thickness. Before surgery, 70.7% of patients had some form of ventricular remodeling, the most frequent being eccentric hypertrophy. This distribution coincides with the general understanding that obesity is accompanied by ventricular hypertrophy.9,16 This is also true in our patients despite their youth and low frequency of hypertension. Remodeling is more often due to an increase in ventricular mass (22 patients had hypertrophy) than in parietal thickness (15 patients). Ventricular geometry in obesity also has other important determining factors, such as preload, afterload and duration of the disease. In this regard, we detected a gradual increase in age accompanying the various stages of ventricular remodeling. The change in ventricular geometry that accompanies age has been recently studied, and is defined by an increase in ventricular thickness, along with a reduction in LV diameters, both diastole and systole.17

We have also detected abnormalities in diastolic function, with 34.1% of patients having an E/A ratio < 1 before surgery. Diastolic...
dysfunction in morbid obesity is due to an increase in blood flow and circulating volume, along with ventricular hypertrophy. The underlying molecular mechanism is not known exactly, although reports have pointed to the role of insulin resistance in the uptake of calcium from the endoplasmic reticulum.

The response of diastolic function to weight loss has been favorable in various studies, although always in terms of parameters that are dependent on hemodynamic conditions: E′ and A′-waves, isovolumetric relaxation time, and mitral deceleration time. On this point, our findings confirm those of other authors.\textsuperscript{9,19} Doppler tissue imaging, however, has been included in only a few studies, with conflicting results.\textsuperscript{20,21} We have not only found no improvement, but have also found a slight reduction in mitral annulus velocity measured by Doppler tissue imaging. This result indicated intrinsic myocyte damage, which would not be reversible despite normalization of other structural aspects of the LV.

We have also not detected changes in the dimensions of the left atrium after weight loss. The assessment of LA size is relevant as an indicator of the increase in LV filling pressures, and their increase in obese patients without complications has been reported.\textsuperscript{4,22}

Moreover, LA remodeling secondary to hemodynamic overload and structural abnormalities may increase the risk of atrial fibrillation in these patients.

We have seen a significant improvement in LV geometric pattern, which has completely normalized in 36.5% of cases and improved in 51.2%. These data confirm those of previous studies.\textsuperscript{23–25} Although the traditional concept that the hypertrophy that accompanies obesity is eccentric,\textsuperscript{5} recent studies, both echocardiographic and cardiac magnetic resonance, have found greater prevalence of concentric patterns.\textsuperscript{26} The observed improvement after bariatric surgery is due both to the normalization of the sphericity pattern and the reduction of left ventricular mass. The interrelationship of changes in hemodynamic situations and the normalization of sympathetic and humoral tone, along with the duration of the obesity, determine the overall effect.\textsuperscript{27}

Finally, the main limitation of this study is the sample size, which may mean it is underpowered in drawing conclusions about the clinical effects found. There was no control group for controlling variability in measurements; however, the fundamental intent of this study was to report the changes that occur in this population.

CONCLUSIONS

Patients who undergo bariatric surgery have a high frequency of ventricular remodeling, which has mainly eccentric characteristics. Weight loss achieved through bariatric surgery is accompanied by significant improvements in several areas: metabolism, hemodynamics and cardiac structure. It is possible, however, that diastolic dysfunction, according to Doppler tissue imaging tests, may be permanent.

FUNDING

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CONFLICTS OF INTEREST

None declared.

REFERENCES