Cardiac remodeling after substantial weight loss: a prospective cardiac magnetic resonance study after bariatric surgery


Abstract

Background: Obesity is a risk factor for left ventricular (LV) hypertrophy and excess cardiovascular disease and mortality. Substantial weight loss is associated with a decrease in cardiovascular mortality. Using volumetric cardiovascular magnetic resonance (CMR) imaging, we studied changes in cardiac anatomy and systolic function in women undergoing substantial weight loss in a university hospital.

Methods: A total of 17 women (body mass index [BMI] 44.1 ± 4.2 kg/m²; age 44 ± 11 yr) scheduled for bariatric surgery underwent volumetric CMR imaging before and 3 and 17 months after surgery.

Results: The body weight declined by 37.2 ± 10.5 kg (32%) with a decrease in BMI to 29.9 ± 4.7 kg/m² (32%, P < .004) during 17 months of observation. The LV mass decreased from 120 ± 23 g to 82 ± 11 g (32%, P < .004), with a linear relationship between the decrease in BMI and decrease in LV mass (P = .008) for the duration of the observation period. After adjustment for systolic and/or diastolic blood pressure, the relationship remained significant (P < .001). The right ventricular (RV) mass declined from 31.7 ± 6.7 g preoperatively to 26.6 ± 4.5 g at 3 months (16%, P < .001) but without additional changes at 17 months. No change was found in the LV or RV end-diastolic volume or ejection fraction.

Conclusion: In morbidly obese women, substantial weight loss was associated with a reduction of LV and RV mass. The decrease in LV mass was linearly related to the reduction in BMI, independent of changes in blood pressure, and might partially explain the reduction in cardiovascular mortality associated with substantial weight loss. The BMI was a predictor of LV mass in this population. (Surg Obes Relat Dis 2009;5:648–652.) © 2009 American Society for Metabolic and Bariatric Surgery. All rights reserved.

Keywords: Bariatric surgery; Cardiac mass; Cardiovascular magnetic resonance; Obesity

The prevalence of obesity has been increasing for several decades, with up to one third of men and women in the United States now estimated to be obese [1,2]. Abdominal obesity, in particular, is an independent risk factor for cardiovascular disease and is associated with excess mortality [3–5].

Obesity is associated with an increased left ventricular (LV) mass and volume and an increased right ventricular (RV) mass [6–8]. Obesity is also associated with increased LV end-diastolic pressure and volume, which can lead to ventricular dilation. Ventricular dilation and its associated increase in wall stress might drive the development of LV hypertrophy among the obese [5]. Concomitant hyperten-
sion, regardless of the presence of the metabolic syndrome, might also contribute to LV hypertrophy among the obese [9]. Obesity is also associated with a decrease in LV systolic and diastolic function [10,11]. Substantial weight loss is associated with a decrease in mortality [12,13].

Previous transthoracic two-dimensional (2D) and M-mode echocardiographic studies have suggested that a regression of LV hypertrophy and improvement in cardiac function occur with weight reduction [14–17]. These data, however, have been derived from a relatively select group of subjects with good echocardiographic windows, because only 70% of transthoracic echocardiographic examinations are interpretable in morbidly obese subjects [14,16,18,19]. Volumetric cardiovascular magnetic resonance (CMR) imaging obtains a better definition of the cardiac structure and function than 2D or M-mode echocardiography and provides more reproducible measurements of cardiac anatomy [20,21]. CMR imaging also allows for accurate anatomic delineation of the heart in the presence of significant amounts of subcutaneous fat [8].

To better understand the cardiac effects of rapid weight loss, we prospectively studied a group of obese women before and after bariatric surgery.

Methods

Study population

A total of 17 women (age 44.5 ± 11.6 yr) recruited from the Beth Israel Deaconess Medical Center Weight Loss Center were enrolled after their acceptance for bariatric surgery. One woman underwent preoperative CMR imaging but relocated after surgery and was unavailable for the follow-up CMR studies. The data from this patient were excluded from the study. Three other patients were unavailable or declined to undergo the 17-month follow-up CMR study. Thus, 16 subjects underwent the 3-month follow-up CMR study and 13 underwent the 17-month follow-up CMR study. Of these women, 10 had a history of hypertension and 6 had a diagnosis of obstructive sleep apnea. The Hospital Committee on Clinical Investigation approved the study, and all the women provided written informed consent.

Bariatric surgery

Bariatric surgery was performed using standard techniques [22] at an American College of Surgeons–accredited bariatric program. Of the 16 women, 4 (25%) underwent laparoscopic adjustable gastric banding and 12 (75%) underwent laparoscopic Roux-en-Y gastric bypass. No clinical evidence of a cardiovascular complication was found in any patient postoperatively.

CMR imaging

All imaging was performed on a commercial 1.5T Philips Achieva whole body scanner (Philips Medical Systems, Best, The Netherlands) with a 60-cm scanner bore. After the initial localizing scans, short axis cine images were obtained, as previously described [23], using a breath-hold, steady-state, free precession cine CMR sequence, with contiguous 10-mm slices and a temporal resolution of 30–35 ms. The patients’ blood pressure, weight, and heart rate were recorded at each scan.

Data analysis

The CMR data were independently analyzed by 2 observers (R.R.J and K.K.P) unaware of the clinical findings using a commercial workstation (EasyVision 5, Philips Medical Systems). The LV and RV endocardial borders were manually traced at end-diastole and end-systole. The LV and RV epicardial borders were traced at end-diastole [23]. The LV and RV end-diastolic volume and end-systolic volume were determined using a summation of the slice thickness multiplied by the slice area. The mass was determined by multiplying the myocardial muscle volume by the density of the myocardium (1.05 g/cm³).

Statistical analysis

Continuous variables are presented as the mean ± standard deviation. The individual patients’ preoperative and postoperative values were compared using a paired Student t test with the Bonferroni adjustment for multiple comparisons among the baseline and 3- and 17-month studies. The relationship between the preoperative and postoperative values was assessed using standard correlation and linear regression analysis. Multivariate regression analysis using a retrospective selection of variables was performed to account for potential confounding variables. Statistical analyses were done using Statistical Analysis Systems for Windows, version 9.1 (SAS Institute, Cary, NC).

Results

The blood pressure and body habitus data are summarized in Table 1. The total body weight had declined by 18.4 ± 3.8
kg (16%, \( P < .001 \)) at 3 months and by 37.2 ± 10.5 kg (32%, \( P < .001 \)) at 17 months. The body mass index (BMI) had declined by 16% and 32% at 3 and 17 months, respectively (both, \( P < .001 \)).

LV changes with weight loss

The changes in LV anatomy are summarized in Table 2. The LV mass had declined by 21.8 ± 12.7 g (18%, \( P < .001 \)) at 3 months postoperatively. The results were similar if we excluded the 3 women who did not undergo CMR imaging at 17 months. After 17 months, the LV mass had declined by 40.0 ± 19.8 g (32% compared with preoperatively, \( P < .001 \)). No change was seen in the LV end-diastolic volume or ejection fraction during the observation period. The baseline BMI and LV mass exhibited a significant linear relationship (\( R = .50, P = .049 \)). A significant linear relationship was also seen between changes in the BMI and changes in the LV mass after surgery (\( P = .008; \)

RV changes with weight loss

The RV anatomic measures are summarized in Table 3. The RV mass correlated with body weight preoperatively and at 3 and 17 months (\( R = .74, R = .83 \), and \( R = .63 \) and \( P < .004, P < .001, \) and \( P = .066, \) respectively). The RV mass had declined by 16% at 3 months (\( P < .001 \)) but did not exhibit additional changes at 17 months. The RV ejection fraction and volumes remained unchanged with weight loss (Table 3).

![Fig. 1. LV mass versus BMI for individual subjects.](image1)

![Fig. 2. LV mass/BMI ratio over time for individual subjects.](image2)

**Fig. 1.** The effect of the changes in BMI on LV mass remained significant on multivariate analysis, which included changes in the systolic and diastolic blood pressure as parameters (\( P = .001 \) after adjustment for either or both). In addition, the LV mass/BMI ratio remained unchanged with weight loss (Fig. 2). In contrast, both the LV mass/body surface area ratio (−11% at 3 months and −18% at 17 months, both \( P < .004 \)) and the LV mass/height ratio (−19% at 3 months and −32% at 17 months, both \( P < .004 \)) declined (Table 2).

**Table 2**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preoperative</th>
<th>Postoperative (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>LV EDV (mL)</td>
<td>152 ± 26</td>
<td>152 ± 29</td>
</tr>
<tr>
<td>LV ESV (mL)</td>
<td>50 ± 18</td>
<td>54 ± 18</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>103 ± 14</td>
<td>97 ± 19</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>68.1 ± 6.4</td>
<td>64.5 ± 6.5</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>120 ± 23</td>
<td>98 ± 15*</td>
</tr>
<tr>
<td>LV mass/height (g/m)</td>
<td>74 ± 12</td>
<td>60 ± 8*</td>
</tr>
<tr>
<td>LV mass/BSA (g/m²)</td>
<td>55 ± 7</td>
<td>49 ± 5*</td>
</tr>
<tr>
<td>LV mass/BMI (g/kg/m²)</td>
<td>2.72 ± 0.41</td>
<td>2.66 ± 0.40</td>
</tr>
</tbody>
</table>

LV = left ventricular; EDV = end-diastolic volume; ESV = endsystolic volume; other abbreviations as in Table 1.

* \( P < .004 \) versus preoperatively.

† \( P < .004 \) versus 3 mo.

![Table 3](image3)

**Table 3**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preoperative</th>
<th>Postoperative (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>RV mass (g)</td>
<td>31.7 ± 6.7</td>
<td>26.6 ± 4.4*</td>
</tr>
<tr>
<td>RV EDV (mL)</td>
<td>157 ± 27</td>
<td>160 ± 30</td>
</tr>
<tr>
<td>RV ESV (mL)</td>
<td>74 ± 23</td>
<td>75 ± 24</td>
</tr>
<tr>
<td>RV ejection fraction (%)</td>
<td>54 ± 8</td>
<td>54 ± 7</td>
</tr>
</tbody>
</table>

RV = right ventricular; other abbreviations as in Table 2.

* \( P < .001 \) versus preoperatively.
Discussion

Both obesity and LV hypertrophy are risk factors for cardiovascular mortality. Using a volumetric CMR approach in the morbidly obese population, we found that substantial weight reduction was associated with a significant regression of LV mass, with no change in LV volume or ejection fraction. The regression in LV mass was linearly related to the magnitude of weight loss after bariatric surgery. When normalized for BMI, the LV mass remained constant, despite a 32% weight loss within 17 months. A regression of the RV mass occurred during the period of rapid weight loss (3 months) after bariatric surgery, with little change thereafter. To our knowledge, these are the first data regarding RV mass changes, identifying a differential response of the 2 ventricles and highlighting the close relationship of the LV mass and BMI in this population.

Several studies have demonstrated that bariatric surgery for morbid obesity is associated with a long-term survival advantage [12,13,24,25]. In a prospective study, Sjöström et al. [12] found a hazard ratio of .76 for overall mortality among subjects who had undergone bariatric surgery compared with matched controls. In a retrospective cohort study, Adams et al. [13] found a 40% decline in mortality in patients who had undergone gastric bypass surgery compared with an age-, gender-, and BMI-matched control group. The LV mass is an independent risk predictor for cardiac mortality [26,27]. Our finding of LV mass regression with weight loss could partially explain these findings.

Other investigators have used surface echocardiography to examine the LV mass changes in this population. Cunha et al. [28] demonstrated LV mass regression at 6 months after bariatric surgery using M-mode echocardiography in a group of 23 patients. Leichman et al. [29] identified a smaller decrease in the LV mass at 3 months. Using volumetric CMR methods, we found regression of the LV mass as soon as 3 months after bariatric surgery, with the degree of LV mass regression linearly related to the decrease in total body weight, with maintenance of the LV mass/BMI ratio. The change in LV mass was independent of changes in blood pressure. Additionally, using volumetric CMR imaging, with its improved reproducibility compared with echocardiography, we demonstrated these differences with a relatively modest sample size.

Previous studies have shown that weight loss, through intensive dieting or weight loss surgery, has led to changes in LV dimensions, including a reduction of the calculated LV mass and wall thickness [14–17,30]. Using 2D echocardiography, Karason et al. [16] examined the effects of weight loss at 1 year after bariatric surgery in 41 obese subjects. They found a significant reduction in the LV mass and wall thickness compared with 31 “conventionally treated” obese subjects and 43 non– obese-matched controls. However, 13% of their obese subjects were excluded from the analysis of LV wall thickness and mass and 28% of their subjects were excluded from the estimations of LV volumes because of suboptimal echocardiographic data, highlighting the limitations of echocardiography in this population [16]. We were able to acquire data from all subjects at both baseline and all follow-up points.

Himeno et al. [20] used M-mode echocardiography to characterize a decrease in LV mass among 22 obese subjects after a 12-week exercise and dietary intervention program. Similar to our findings, the change in LV mass was independent of blood pressure. Again, 12% of their study population were excluded because of uninterpretable echocardiographic data.

Hinderliter et al. [14] used 2D echocardiography to study 82 subjects, 63 of whom had been randomized to a 6-month exercise or weight management program, and 19 controls. The intervention group had a decreased relative LV wall thickness, with a trend toward a decrease in LV mass relative to the control group. As with the other echocardiographic studies, 42 patients (29%) of the overall study population of 144 had echocardiograms “in which left ventricular dimensions or wall thicknesses could not be quantified with confidence” [14].

Unlike previous 2D and M-mode echocardiographic studies in the morbidly obese population, we did not find a decrease in LV end-diastolic volume with weight loss. The reasons for this conflict are uncertain, but they might reflect the volumetric approach of CMR imaging compared with the geometric modeling of 2D and M-mode echocardiography.

None of the previously cited echocardiographic studies were able to address RV mass or volume. Our data demonstrated a decrease in RV mass in the early period of weight loss. The reason for the initial parallel decline in RV mass and weight but subsequent maintenance of RV mass despite ongoing total body weight loss is unexplained.

Our report had several limitations. The study population consisted of entirely middle-age women. Our findings might not be applicable to all age groups or to men. The mechanism by which cardiac remodeling occurs is not fully explained, but it appears to be independent of a reduction in blood pressure or other metabolic factors (e.g., hyperlipidemia). We did not assess the metabolic or inflammatory parameters that might affect LV mass. The potential confounder of 2 different surgical approaches could not be addressed by the present study. Also, we could not determine whether the decline in LV mass reflects a decrease in fatty infiltration of the myocardium or a decrease in LV myocyte hypertrophy. Whether the RV changes we observed were a direct result of weight loss or were possibly due to improvements in obstructive sleep apnea or a decline in pulmonary artery pressure in this population is uncertain.
Conclusion

In morbidly obese women, rapid weight loss was associated with significant regression of the LV and RV mass, with maintenance of the LV mass/BMI ratio and no change in LV or RV volume or ejection fraction. The decline in LV mass was linearly related to the weight reduction, independent of changes in blood pressure, and might partially explain the reduction in cardiovascular mortality associated with substantial weight loss.

Disclosures

The authors claim no commercial associations that might be a conflict of interest in relation to this article.

References

[21] Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. Ann Intern Med 1991;114:345–52.