Major weight loss prevents long-term left atrial enlargement in patients with morbid and extreme obesity

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Aims To assess long-term changes in left atrial (LA) volume in patients with morbid obesity [body mass index (BMI) ≥ 35 kg/m² with co-morbidities] and extreme obesity (BMI ≥ 40 kg/m²), after surgically-induced weight loss (WL) after gastric bypass surgery.

Methods and results We reviewed 57 patients who underwent gastric bypass surgery and had echocardiograms both before and after the operation. A control group was frequency-matched for BMI, sex, age, and for duration of follow-up. After a mean follow-up of 3.6 years, LA volume did not change significantly in patients who underwent bariatric surgery, but increased in the control group by 15 ± 28 ml (P, 0.0001), and 0.1 ± 0.2 ml (P, 0.0001) for height-indexed LA volume, with a difference between cases and controls that remained significant after adjusting for potential confounders (P, 0.01). In the study population as a whole, there was a positive correlation between change in body weight and change in LA volume (r = 0.22, P, 0.006) independent of clinical conditions associated with LA enlargement.

Conclusion Change in body weight is associated with change in LA size independent of obesity-associated co-morbidities. Successful WL induced by bariatric surgery prevents the progressive increase in LA volume.

KEYWORDS Obesity; Bariatric surgery; Left atrial volume; Diastolic function

Introduction

Increased cardiovascular risk in obese patients is explained in part by haemodynamic and metabolic derangements due to co-morbidities directly related to obesity. Obesity is itself a direct cause of structural cardiac abnormalities, such as increased left ventricular (LV) mass,1,2 that can predispose to cardiomyopathy,3 congestive heart failure (CHF),4 and atrial fibrillation.5 Excess body weight in obese patients leads to increments in total blood volume and cardiac output, and a decrease in total peripheral resistance.6 Consequently, increased LV filling pressure and volume have been implicated in chamber dilation leading to greater wall stress and myocardial mass.7

Cross-sectional and short-term longitudinal studies suggest that obesity is associated with diastolic dysfunction. There is evidence that dynamic LV filling patterns measured with transthoracic echocardiography improve after short-term weight loss (WL).8 Two previous studies have suggested that short-term WL is associated with decreases in left atrial (LA) size, suggesting that WL is associated with improvement in diastolic function.9,10 However, these studies had a relatively short follow-up, did not adjust for change in arterial blood pressure, or lacked a control group.

We conducted a retrospective study to test the hypothesis that major WL is associated with improvements in LA volume, independent of any concomitant improvement of systemic blood pressure. We assessed long-term changes in LA volume in patients with morbid obesity [body mass index (BMI) ≥ 35 kg/m² with obesity-related co-morbidities] and extreme obesity (BMI ≥ 40 kg/m²), after surgically-induced WL after gastric bypass surgery. Obese patients who did not undergo the procedure served as a control group.
Methods

Study population

We included all the patients who underwent Roux-en-Y gastric bypass surgery for morbid or extreme obesity from 1995 to 2005 at Mayo Clinic, Rochester, MN, and had two or more transthoracic echocardiographic examinations, one within 2 years before the operation and one >6 months after the procedure. In patients who had multiple echocardiograms before the surgery, we selected the one closest in time to the date of operation; in patients who had multiple echocardiograms after the operation, we selected the study furthest from the surgery date.

We obtained a control group by frequency match for sex, age (±2 year), BMI (±2 kg/m²) at the time of the first echocardiogram, and follow-up duration (±6 months). The controls were randomly selected from patients with morbid or extreme obesity who underwent at least two echocardiographic evaluations at Mayo Clinic Rochester between 1995 and 2006, but who did not undergo bariatric surgery. We excluded patients with past medical history of cardiovascular disease, atrial fibrillation, obstructive sleep apnoea, idiopathic cardiomyopathy, and severe valve disease, as well as those with technically limited echocardiograms. This study was approved by the Institutional Review Board, and we included only the clinical records of patients who had authorized their use for research.

Echocardiographic measurements

From the original videotaped or digitally recorded signals, we performed digital off-line analysis of LA volume in all patients. The measurements were performed by a single observer (C.A.G.) blinded to the clinical history, amount of WL, study group, and original echocardiographic report. LA volume was measured using the biplane area-length method. We obtained the apical four and two-chamber views for determination of LA area and length from the middle of the plane of the mitral annulus to the posterior wall. If the LA in the two-chamber view was foreshortened, the apical long-axis view was used. The maximal LA chamber area and length were measured at end of ventricular systole. We calculated LA volume from the formula \[ \text{Volume} = \frac{1}{2} \times (A1 + A2) \times L \times H \], where A1 is the four-chamber LA area, A2 is the two-chamber or apical long axis LA area, and L is the smallest of the two lengths obtained. We determined LA volume indexed by height. Indexing LA volume by body surface area was considered inappropriate for this study, because body surface area incorporates weight and body size in the calculation. Therefore, hypothetical WL associated with decreased LA size might not show any change in LA volume indexed by body surface area, because both numerator and denominator would change in the same direction. We obtained transmitral flow velocities from all studies that included pulsed-wave Doppler. We measured dynamic diastolic flow from the apical four-chamber view with the sample volume placed between the tips of the mitral valve leaflets. The peak early (E) and late (A) flow velocities and deceleration time were measured and averaged over three cardiac cycles during normal respiration, and the ratio of early diastolic to late diastolic flow (E/A ratio) was calculated.

Clinical and demographic data

Clinical and demographic data were obtained from the medical records of the clinical encounter nearest in time to the date of the baseline echocardiogram. We abstracted information regarding history of hypertension, diabetes mellitus, dyslipidemia, pulmonary disease, atrial fibrillation, obstructive sleep apnoea, idiopathic cardiomyopathy, and coronary artery disease. Definition of coronary artery disease required a diagnostic angiogram with evidence of coronary stenosis, or documented history of myocardial infarction, angina, coronary artery bypass grafting, or percutaneous coronary revascularization.

Weight, height, heart rate, and blood pressure measurements were obtained from the echocardiogram reports or the clinical visit closest in time to the echocardiogram.

Measurements of weight change

Because no consensus exists about the best measure of weight change, we decided to use three different approaches: (i) WL was defined as the absolute weight change between baseline and last follow-up; this parameter is independent of other patient characteristics like height; (ii) Percentage of WL (%WL) was calculated by dividing the WL by weight at the time of the first echocardiogram and reflects the percent of weight lost in relation to the patient’s total weight; and (iii) Change in BMI (ΔBMI) was defined as the absolute change in BMI; this parameter accounts for the WL relative to height in meters squared (m²).

Statistical analysis

We divided the statistical analyses into two sets; the first set compared measurements in patients who underwent gastric bypass surgery with measurements in the control group. For the second set of analyses, we pooled data from the entire studied population (patients with and without surgery) to analyse a wide spectrum of weight changes over time against changes in LA volume. All values for quantitative measures are expressed as mean ± standard deviation.

Patients with gastric bypass surgery vs. controls

At baseline, differences between groups with and without bariatric surgery were assessed with an unpaired t-test for continuous variables and χ² for categorical variables. Differences between baseline and follow-up within groups were assessed with a paired t-test. Comparisons of changes between cases and controls were performed with an unpaired t-test. We did not compare the two groups with a paired t-test because we used a frequency match strategy rather than individual match. Multivariate analysis was performed to assess bariatric surgery as a predictor of change in LA volume. We also created different models by adjusting for potential confounders of this association. We constructed a base model that included age at the time of the first study, sex, history of systemic hypertension, diabetes mellitus, and coronary artery disease; we then added obstructive sleep apnoea, change in blood pressure, and atrial fibrillation separately.

Pooled data

We calculated Pearson’s correlation coefficients to test univariate relationships between change in body weight (including the three measurements of weight change), and change in LA volume. Among the entire group of participants, there was a wide range of weight change that included patients with major, mild, or modest WL, weight gain, and patients in whom weight did not change at all. We performed multiple linear regressions using change in LA volume as the dependent variable, and change in body weight expressed in different dimensions as an independent variable, as well as other covariates that may be implicated in change in LA volume. Furthermore, we stratified the pooled data according to the presence or absence of atrial fibrillation, obstructive sleep apnoea, and hypertension, and we tested the correlation between change in body weight and change in LA volume. We compared correlation coefficients between patients with and without each of these diagnoses.

A subgroup analysis for diastolic dysfunction severity included patients with baseline and follow-up measurements of E/A ratio, deceleration time, and height-indexed LA volume. We arbitrarily classified diastolic function according to the following parameters: (i) Normal: If E/A ratio was ≥1 with height-indexed LA volume <0.52 ml/cm and deceleration time ≥160 ms. (ii) Impaired relaxation: if EA ratio <1. (iii) Restrictive pattern: if E/A ratio was ≥1.
with either height-indexed LA volume \( \geq 0.52 \) ml/cm or deceleration time \(<160\) ms. The cut-off value for height-indexed LA volume was established using the highest quartile of this measurement at baseline in the study population.

In order to assess reliability of the measurements, two independent observers (E.O. and C.A.G.) measured a randomized sample of 10% of all echocardiograms. The inter-observer and intra-observer concordance were evaluated by Pearson correlation coefficients.

Results

A total of 64 patients who underwent bariatric surgery met the inclusion criteria. Seven patients were excluded because of a history of heart transplantation \((n=2)\), severe cardiac valvular disease \((n=4)\), and cardiac amyloidosis \((n=1)\). Therefore, we included 57 cases, and 57 frequency-matched controls. Within the cases, 22 patients were men \((39\%)\); age at the time of the first echocardiogram was \(52 \pm 9\) years and BMI was \(49 \pm 9\) kg/m\(^2\). The mean age, sex, duration of follow-up, and BMI were similar in surgical and control subjects. Baseline clinical characteristics of patients and controls are noted in Table 1. The group who underwent gastric bypass surgery included more subjects with hypertension than the control group \((P = 0.01)\), whereas the group without surgery had more current smokers \((P = 0.009)\).

Patients with gastric bypass surgery vs. controls

At follow-up, patients who underwent bariatric surgery showed a significant reduction in body weight, body surface area, BMI, and heart rate, Table 2. Only the control group had significant increases in LA volume and height-indexed LA volume at follow-up, with mean change of \(15 \pm 28\) ml for LA volume \((P < 0.0001)\), and \(0.09 \pm 0.16\) ml for height-indexed LA volume \((P < 0.0001)\). Changes in LA volume \((P = 0.02)\), height-indexed LA volume \((P = 0.01)\), and E/A ratio \((P = 0.04)\) were different between cases and controls. When we adjusted for potential confounders in the multivariate analysis, the difference between cases and controls was still significant for LA volume \((P = 0.01)\) and for height-indexed LA volume \((P = 0.01)\).

A subgroup analysis of the 29 patients with atrial fibrillation showed that LA volume at baseline was greater in patients who underwent bariatric surgery than in patients without the procedure. Only the control group showed a significant increase in LA volume at follow-up \((P = 0.02)\), Table 2.

Pooled data

Analysis of the pooled data showed a positive and significant correlation between change in LA volume and change in body weight measured by WL \((r = 0.1994)\), \%WL \((r = 0.2307)\), and \(\Delta\)BMI \((r = 0.2209)\), \(P < 0.05\) for all, Figure 1; statistical significance remained after multivariate analyses \((P < 0.05)\), Table 3. These correlations were also maintained when we used height-indexed LA volume and when we assessed interaction of the measurements of change in body weight \((WL, \%WL, \Delta BMI)\) with either hypertension or obstructive sleep apnoea \((P < 0.05)\).

Table 4 shows stratified analysis on measures of change in body weight \((WL, \%WL, \Delta BMI)\) and changes in LA volume, according to the presence or absence of either atrial fibrillation, obstructive sleep apnoea, or hypertension, Table 4.

These correlations were significant only in the groups diagnosed with obstructive sleep apnoea and hypertension \((P < 0.05)\), but the difference between correlation coefficients of patients with and without a diagnosis was not significant \((P > 0.9)\).

Analysis for diastolic dysfunction using mitral flow Doppler information included 27 cases and 27 controls. Six patients who underwent gastric bypass surgery worsened one level, and 2 improved one level; in the group without gastric bypass surgery 4 worsened one level, and 1 patient improved one level. Analysis showed no statistical difference in rates of worsening and improvement between cases and controls.

Inter-observer and intra-observer agreement for LA volume measurements were strong, with Pearson correlation coefficients of 0.84 and 0.91, respectively.

Discussion

In the present study, long-term change in body weight correlated directly with change in LA volume. Although baseline LA volumes were similar in patients who did and did not
undergo gastric bypass surgery, patients who lost weight after surgery had a lesser change in LA volume compared with patients who did not undergo the procedure and had only a mild (at best) WL. In addition, the association between changes in weight and changes in LA volume was independent of obesity-associated co-morbidities.

To the best of our knowledge, this is the first longitudinal study showing a direct correlation between change in body weight and change in LA size independent of changes in blood pressure. This observation confirms and extends previous evidence suggesting that obesity may be implicated in LA structural changes, independent of obesity-related co-morbidities like hypertension, diabetes mellitus, and obstructive sleep apnoea.

Cross-sectional studies have also suggested that obesity is associated with increased LA size independent of hypertension. Gottdiener et al. studied a population of hypertensive men and showed that obesity was the strongest predictor of LA size and that a weak correlation existed between systolic blood pressure and LA size after adjusting for obesity. Later, Karason et al. assessed the change in LA volume in patients who lost weight after gastroplasty. Although this study reported a decrease in LA volume at 12 months, it was not clear if the association was independent of any improvements in blood pressure or other obesity-related co-morbidities. In our study, weight change was associated with change in LA volume in the overall group after a mean follow-up of 3.6 years. When patients were divided into those who underwent gastric bypass surgery vs. those who did not, both groups had an average increase in LA volume, although the group without surgery had a greater increase in LA volume.

Because increased LA size is a marker of chronic increased LV filling pressures, our findings support the hypothesis that patients with obesity are more likely to have diastolic dysfunction and that these changes may be partially modified by WL. A study in patients without coronary artery disease suggested an association between obesity and diastolic dysfunction, reporting a significant negative correlation between E/A ratio and BMI, and an increased end-diastolic LV volume as assessed by cardiac catheterization. Longitudinal data are limited to a few studies evaluating the association between change in body weight and diastolic dysfunction. Alpert et al. reported that substantial WL improved acute transmitral Doppler E/A ratio and E-wave deceleration time, which they attributed to a decrease in LV mass and favourable changes in loading conditions. Tissue Doppler findings in patients after marked WL have shown an increase in LV lateral and septal mitral annular early diastolic tissue velocities indicative of an improvement in diastolic function. These changes were related directly to the amount of WL. In contrast to previous studies, we did not find a significant difference in Doppler measures of diastolic function when we compared patients with and without gastric bypass surgery. Our results, however, were limited because only a subset of our patients had all the Doppler measurements.

Table 2  Anthropometric, clinical, haemodynamic, and echocardiographic data at baseline and follow-up

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>Controls</th>
<th>Baseline</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>57</td>
<td>143 ± 33</td>
<td>100 ± 26</td>
<td>57</td>
<td>141 ± 33</td>
<td>138 ± 34</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>57</td>
<td>49 ± 9</td>
<td>35 ± 8</td>
<td>57</td>
<td>48 ± 8</td>
<td>47 ± 9</td>
</tr>
<tr>
<td>Body surface area (m²)</td>
<td>57</td>
<td>2.51 ± 0.36</td>
<td>2.24 ± 0.34</td>
<td>57</td>
<td>2.47 ± 0.34</td>
<td>2.40 ± 0.31</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>57</td>
<td>78 ± 16</td>
<td>67 ± 17</td>
<td>51</td>
<td>78 ± 18</td>
<td>76 ± 16</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>57</td>
<td>133 ± 19</td>
<td>128 ± 16</td>
<td>49</td>
<td>133 ± 19</td>
<td>131 ± 18</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>50</td>
<td>77 ± 15</td>
<td>73 ± 14</td>
<td>48</td>
<td>76 ± 14</td>
<td>77 ± 12</td>
</tr>
<tr>
<td>2D Echocardiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left atrial volume (mL)</td>
<td>57</td>
<td>78 ± 23</td>
<td>81 ± 30</td>
<td>57</td>
<td>76 ± 28</td>
<td>91 ± 35</td>
</tr>
<tr>
<td>Indexed left atrial volume (mL/cm)</td>
<td>57</td>
<td>0.46 ± 0.13</td>
<td>0.48 ± 0.16</td>
<td>57</td>
<td>0.44 ± 0.14</td>
<td>0.53 ± 0.19</td>
</tr>
<tr>
<td>Atrial fibrillation at baseline</td>
<td>10</td>
<td>104 ± 28</td>
<td>106 ± 38</td>
<td>19</td>
<td>87 ± 26</td>
<td>115 ± 35</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>35</td>
<td>0.79 ± 0.24</td>
<td>0.79 ± 0.23</td>
<td>38</td>
<td>0.90 ± 0.26</td>
<td>0.93 ± 0.29</td>
</tr>
<tr>
<td>Peak A velocity (m/sec)</td>
<td>35</td>
<td>1.2 ± 0.4</td>
<td>1.3 ± 0.5</td>
<td>38</td>
<td>1.1 ± 0.4</td>
<td>1.0 ± 0.4</td>
</tr>
<tr>
<td>Deceleration time (ms)</td>
<td>32</td>
<td>204 ± 49</td>
<td>199 ± 47</td>
<td>33</td>
<td>217 ± 50</td>
<td>220 ± 54</td>
</tr>
</tbody>
</table>

Values at baseline and follow-up are given as the mean ± SD. ***P < 0.001; †P < 0.0001 comparing baseline to follow-up values. ‡P < 0.05; §P < 0.001; **P < 0.0001 compared cases and controls. Bold for statistically significant values.

Possible explanations of our findings

Diastolic dysfunction in patients with obesity is more prevalent in persons with obesity-associated conditions such as hypertension. Several studies, however, have suggested that obesity itself is directly related to structural cardiac abnormalities. For example, increased circulating blood volume in obese subjects induces eccentric hypertrophy that impairs LV compliance. In the presence of a non-compliant LV, LA pressure increases to maintain adequate LV filling, leading to an increase in atrial wall tension and consequent LA enlargement. In patients with hypertension, chronic exposure to increased systemic vascular resistance results in LV hypertrophy that impairs LV relaxation and
Affects LV diastolic filling. However, recent data suggest that the association between obesity and diastolic dysfunction goes beyond systemic hypertension. A recent study showed that obesity correlated with LV diastolic dysfunction in patients with and without a history of hypertension. This correlation was independent of systolic and diastolic blood pressure values and heart rate. Obstructive sleep apnoea, a common condition in patients with obesity, could also explain the presence of diastolic dysfunction in patients with and without a history of hypertension. Sidana et al. showed that obese patients with moderate or severe obstructive sleep apnoea have a higher prevalence of LV diastolic dysfunction than patients with mild or no obstructive sleep apnoea. Otto et al. found that moderately obese patients without obstructive sleep apnoea did not have impaired LV diastolic function. In our study, neither obstructive sleep apnoea nor hypertension explained the association between changes in body weight and changes in LA size, although this correlation appeared to be stronger in patients with a baseline diagnosis of obstructive sleep apnoea and hypertension.

Figure 1 Correlations between change in body weight change in left atrial volume in the entire study group. A positive correlation means that weight gain was associated with an increase in left atrial volume. ΔBMI, change in body mass index; ΔLA vol, change in LA volume. r, correlation coefficients; %WL, percentage of weight loss. *P < 0.05.

Table 3 Univariate and multivariate assessment of the effect of change in weight on the change in LA volume

<table>
<thead>
<tr>
<th>Model</th>
<th>Unadjusted</th>
<th>Adjusted for age, gender, diabetes mellitus, hypertension, coronary artery disease and change in systolic and diastolic blood pressure</th>
<th>Adjusted for age, gender, diabetes mellitus, hypertension, coronary artery disease, and obstructive sleep apnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>b ± SEM</td>
<td>P-value</td>
<td>b ± SEM</td>
<td>P-value</td>
</tr>
<tr>
<td>WL</td>
<td>0.18 ± 0.08</td>
<td>0.034</td>
<td>0.60 ± 0.08</td>
</tr>
<tr>
<td>%WL</td>
<td>0.18 ± 0.08</td>
<td>0.034</td>
<td>0.60 ± 0.08</td>
</tr>
<tr>
<td>ΔBMI</td>
<td>0.18 ± 0.08</td>
<td>0.034</td>
<td>0.60 ± 0.08</td>
</tr>
</tbody>
</table>

Model 1: Unadjusted; Model 2: Adjusted for age, gender, diabetes mellitus, hypertension, coronary artery disease and change in systolic and diastolic blood pressure; Model 3: Adjusted for age, gender, diabetes mellitus, hypertension, coronary artery disease, and obstructive sleep apnoea. All correlations were statistically significant (P < 0.05). β, parameter estimate for each measurement of weight loss; SEM, standard error of mean; WL, weight loss; %WL, percentage of weight loss.
Strengths and limitations

Strengths of our study include a long follow-up, inclusion of patients with a wide range of weight change, and paired measurements of LA volume. We included a control group of patients without gastric bypass surgery, allowing us to compare the effect of WL in patients with and without surgery and also to examine a wide spectrum of weight change at follow-up. In addition, we had comprehensive clinical information related to the presence of and changes in obesity-related co-morbidities.

Our study has several limitations. First, we included patients referred for at least two echocardiograms, half of whom had a history of gastric bypass surgery; our study hence had a relatively small sample size. These factors increase the chances of selection bias. Second, surgical patients may be different from those who do not have gastric bypass surgery. To minimize these differences, a paired analysis was performed such that each patient served as his or her own control, and patients were further compared with a control group matched for sex, age, and BMI. A third limitation was the presence of potential confounders that may affect changes in LA volume over time. To limit confounding, we performed adjusted analyses for the most relevant variables. Last, we had to rely on retrospective historical data, and information was missing in some patients. However, when we analysed the characteristics of excluded patients, they appeared to be similar to those included in our final sample.

Implications of our findings

Our findings help to understand the complex interaction between obesity, CHF, and atrial fibrillation that has been partially explained by the parallel increase in the incidence and prevalence of these three conditions in the last decades. Indeed, CHF and atrial fibrillation have been called ‘the two new epidemics of cardiovascular disease’.22 Our data provides a mechanistic explanation for how obesity may cause CHF and atrial fibrillation. First, obesity is strongly associated with CHF, most likely as a consequence of chronic decrease in LV compliance manifested by LA enlargement. Second, obesity, recently identified as a risk factor for atrial fibrillation,5 may cause this arrhythmia by contributing to the enlargement and remodeling of the LA. Therefore, our data support the concept that the increased prevalence of CHF and atrial fibrillation may be related to the current epidemic of obesity.

Conflict of interest: none declared.

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References


Table 4 Correlation coefficients in stratified analysis of patients with and without the diagnosis of atrial fibrillation, obstructive sleep apnoea, or hypertension

<table>
<thead>
<tr>
<th></th>
<th>WL % WL</th>
<th>Δ BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in LA volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Present (n = 29)</td>
<td>0.3138</td>
</tr>
<tr>
<td>Obstructive sleep apnoea</td>
<td>Absent (n = 84)</td>
<td>0.1675</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Present (n = 85)</td>
<td>0.2337*</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Absent (n = 28)</td>
<td>0.1983</td>
</tr>
<tr>
<td>Change in indexed LA volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Present</td>
<td>0.3346</td>
</tr>
<tr>
<td>Obstructive sleep apnoea</td>
<td>Absent</td>
<td>0.17</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Present (n = 90)</td>
<td>0.2367*</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Absent (n = 23)</td>
<td>0.2199</td>
</tr>
</tbody>
</table>

Positive and negative signs represent the presence or absence of the disease at baseline. ΔBMI, body mass index; WL, weight loss; % WL, percentage of weight loss. *P < 0.05.


