Original Article

The impact of bariatric surgery on renal and cardiac functions in morbidly obese patients

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Abstract

Background. Cardiac adaptation to obesity includes both structural and functional alterations in the heart. The kidneys also suffer the consequence of excessive increase of body weight. This study aims to assess the functional, cardiac and renal changes in a cohort of morbidly obese patients, as well as changes after bariatric surgery—the last therapeutic option for these patients.

Methods. Patients referred for bariatric surgery were prospectively included. In each case, transthoracic echocardiography and a blood test were performed before the procedure and repeated 1 year after surgery. The estimation of the glomerular filtration rate (GFR) was addressed by the Cockcroft–Gault lean body weight formula.

Results. Sixty-one patients completed the 1-year follow-up. Of these, 81.9% were female. The mean age was 41.1 ± 9.8 years and the mean body mass index was 47.4 ± 5 kg/m², decreasing to 30.5 ± 5.07 kg/m² after the procedure. Before surgery, the estimated GFR was 92.7 ± 25.4 mL/min, with hyperfiltration being present in 14.8% of patients, whereas an impaired GFR was detected in 8.3%. Patients showed preserved systolic function and cardiac remodelling. Diastolic function was abnormal in 27.9% of patients. At the 1-year follow-up, favourable changes in the left ventricular geometry and related haemodynamic status were observed. There was no significant change in the estimated GFR in the overall group, although hyperfiltration was ameliorated in 9.8% and a poor GFR was improved in 3.3%. The improvement was not associated with changes in either blood pressure or the BMI. However, in this group of patients the amelioration of the GFR was associated with an increased stroke volume and improvement in diastolic function.

Conclusions. In morbidly obese patients, GFR is usually normal and only a small percentage of them show hyperfiltration or a reduced GFR. Bariatric surgery has a favourable impact on renal function in only a reduced group of patients who also experience an improvement in cardiac performance.

Keywords: bariatric surgery; cardiac remodelling; diastolic dysfunction; hyperfiltration; obesity

Introduction

Changes in lifestyle that have occurred throughout the last decades have been used to explain the current sharp rise in the prevalence of obesity, a major health problem associated with increased morbidity and mortality, especially those of a cardiovascular origin [1]. Obesity also promotes the development of other co-morbidities such as diabetes, hypertension and dyslipidaemia, which are important risk factors for chronic kidney disease progression and death [2–4].

The heart adapts to obesity through remodelling, which is a process that can involve left ventricular hypertrophy and dilatation, as well as atrial changes [5–7]. These changes can affect cardiac function and finally initiate the development of heart failure. At the renal level, obesity is accompanied by hyperfiltration, renal hypertrophy and other changes that can promote the development of proteinuria in the long term [3, 4, 8, 9].

Body weight loss is a fundamental strategy for improving the risk of obese patients, and especially so for those with morbid obesity for which weight reduction is difficult to attain through conventional means. Bariatric surgery is a useful way of losing weight for these patients since the reduction can reach between 50 and 60% of body weight [10]. Some studies have demonstrated that bariatric surgery improves cardiac remodelling [5, 11] and is associated with a reduced number of cardiovascular deaths and lower incidence of cardiovascular events in
obese adults after 15 years of follow-up [12]. Regarding the renal consequences, it is not well established whether these beneficial effects are maintained in the long term even though a reduction in proteinuria has been reported [13, 14] (or whether these changes are associated with or accompanied by the improvement of other alterations observed in obese patients). Therefore, the aim of this study was to evaluate the impact of bariatric surgery on renal function and other targets of organ damage in morbidly obese patients at 1 year after bariatric surgery.

Materials and methods

This clinical study included all patients referred for bariatric surgery. A panel including surgeons, endocrinologists and cardiologists carried out the selection of candidates. Inclusion criteria were age >18 years and universally accepted indications: long-term obesity (>4 years); body mass index (BMI) >40 kg/m², or ≥35 kg/m² with obesity-related comorbidities, i.e. diabetes mellitus, obesity hyperventilation syndrome, obstructive sleep apnoea, hypertension. Candidates were excluded if they had unacceptably high-surgical risk due to co-morbidities.

Upon acceptance by the panel, patients underwent a cardiovascular examination including physical examination, 12-lead electrocardiogram (ECG) and a transthoracic echocardiogram. The preoperative examination also included anthropometric measurements. Serum and plasma samples were obtained for a metabolic profile-glucose, lipid and creatinine levels. The estimation of the glomerular filtration rate (GFR) was addressed by the Cockcroft–Gault lean body weight (CG-LBW) equation since it has been suggested to be the most reliable in the context of obesity [15, 16]. The local Ethics Committee approved the protocol and all patients signed the informed consent.

Anthropometric measurements

The height and weight of patients were recorded when taking the echocardiogram, with the patients wearing light clothing without shoes. Wall scales were employed for weight measurements. After 10 min of rest in a sitting position, a 12-lead ECG was taken and blood pressure was measured in the non-dominant arm. The BMI was calculated according to the formula: weight (kg)/height squared (m).

Patients were classified as follows according to the BMI (www.asbs.org/Newsite07/media/asmbFs.fs): normal weight, BMI <25; overweight: BMI 25–30; Grade 1 obesity: BMI 30–35; Grade 2 obesity: BMI 35–40; Grade 3 or morbid obesity: BMI 40–50; super-obesity: BMI 50–60 and super-morbid obesity: BMI >60.

Echocardiogram

Echocardiographic methods have been described elsewhere [5]. Briefly, the left ventricular mass was indexed to the power of 2.7 to minimize the interference of obesity in the estimate of ventricular mass [17]. The relative wall thickness (RWT) was calculated using the formula RWT = (IVS + PW)/EDLV, where IVS, interventricular septum; PW, posterior wall; EDLV, end-diastolic left ventricular diameter.

Follow-up

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; plasma samples were obtained for a metabolic profile-glucose, lipid and creatinine levels during the same visit.

Surgical technique

Y-en-Roux gastric bypass was the first choice in the surgical planning. For those patients with high-surgical risk or super-obesity (>50 kg/m²), a tubular gastrectomy was planned.

Data analysis

Continuous variables are expressed as the mean ± standard deviation. Categorical variables are expressed in absolute values and percentages. The differences between categorical variables were analysed using the Chi-square test. Student’s t-test or the ANOVA test was used for continuous variables. The normality of distributions was verified by means of the Kolmogorov–Smirnov test. The differences between continuous variables before and after bariatric surgery were analysed using Student’s t-test for correlated samples or the corresponding non-parametric test, as appropriate. Using the improvement category of the LV geometric pattern as a reference, we calculated the effects on clinical variables through coefficients of the linear and logistic regression models that included the preoperative baseline value of the same variable. The beta correlation coefficients, along with their 95% confidence intervals (95% CI), were obtained using a linear regression model. A value of P<0.05 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).

Results

Baseline characteristics

Sixty-one patients were included in the study. The mean age was 41.1±9.8 years. Most patients were female (81.9%). Prior to surgery, the mean BMI was 47.4±5 kg/m². The glucose and lipid profiles are presented in Table 1. Co-morbidities were present in 81.4% of patients. Among them, the most common was hypertension, present in 44.1% of patients. Of note, dyslipidaemia was present in 35.6% of patients. Drug treatment for hypertension included ACEIs (or AT1 antagonists), whereas all dyslipidaemic patients were on statins. Only 10.2% were diabetic (Table 1). None of the patients had previous cardiovascular events.
Impact of bariatric surgery

One year after surgery, the mean BMI decreased to 30.54 ± 5.07 kg/m², consistent with Grade 1 obesity. This was accompanied by significant decreases in systolic and diastolic blood pressure and an improvement in their metabolic profile, i.e. glucose, cholesterol and triglyceride levels. This was a consistent finding for the entire cohort of patients. Consequently, an important reduction in the incidence of hypertension (69%), dyslipidaemia (73%) and diabetes (68%) was observed. None of the patients had cardiovascular events during the follow-up.

The cardiac geometry improved after surgery. There was a significant reduction in both the RWT and the left ventricular mass index, Table 2. However, diastolic function addressed by tissue Doppler analysis of velocities did not improve despite favourable changes in the LV geometric pattern and the related haemodynamic status. Actually, further impairment in diastolic function was observed.

There was no significant change in the estimated GFR in the overall group (Table 1). Changes in the estimated GFR were detected in a small group of patients. Among them, improvement—whether decrease over baseline hyperfiltration (9.8%) or increase over poor (3.3%) estimated GFR—was more frequent than impairment. The improvement was not associated with changes in blood pressure or the BMI, but it was related to an increased stroke volume (P = 0.03). Diastolic function, however, was a better predictor of improvement in renal function (AUC, 0.63) although it did not reach statistical significance (P = 0.14), probably due to the small number of patients with improved renal function. On the other hand, deterioration of the GFR was not associated with any other clinical or haemodynamic variable.

Discussion

Obesity exerts a profound effect on renal function and structure. Hyperfiltration is a common feature observed in obese patients that is partially explained by the increase in circulating volume due to the excess of body weight [8, 9]. The present study, however, shows that obese patients show a wide range of estimated GFRs moving from hyperfiltration (estimated GFR >120 mL/min) to

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**Table 1. Epidemiological and clinical characteristics of the patients**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pre-op</th>
<th>Post-op (1 year)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>41.9 (9.8)</td>
<td>37.45 (10.7)</td>
<td>0.001</td>
</tr>
<tr>
<td>Women</td>
<td>50 (81.9)</td>
<td>42 (83.6)</td>
<td>0.02</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.61 (0.08)</td>
<td>1.62 (0.08)</td>
<td>0.73</td>
</tr>
<tr>
<td>B.W. (kg)</td>
<td>124.1 (21.1)</td>
<td>92.1 (16.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>47.41 (5.0)</td>
<td>30.6 (5.07)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>131.2 (16.2)</td>
<td>112.9 (12.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>80.1 (12.2)</td>
<td>67.6 (9.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>75.1 (9.0)</td>
<td>66.04 (8.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>98.8 (18.6)</td>
<td>83.6 (11.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>188.7 (40.4)</td>
<td>169.9 (35.1)</td>
<td>0.001</td>
</tr>
<tr>
<td>LDL-cholesterol (mg/dL)</td>
<td>125.5 (32.6)</td>
<td>105.7 (29.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL-cholesterol (mg/dL)</td>
<td>46.5 (12.9)</td>
<td>53.7 (12.7)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>144.6 (91.6)</td>
<td>82.56 (34.15)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>0.71 (0.15)</td>
<td>0.69 (0.11)</td>
<td>0.63</td>
</tr>
<tr>
<td>Estimated GFR (mL/min)</td>
<td>92.7 (25.1)</td>
<td>95.7 (23.4)</td>
<td>0.28</td>
</tr>
<tr>
<td>Co-morbidities %</td>
<td>16 (44.1%)</td>
<td>9 (14.7%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>HTN</td>
<td>6 (10.2%)</td>
<td>2 (3.2%)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>DM</td>
<td>21 (35.6%)</td>
<td>6 (9.8%)</td>
<td>&lt;0.04</td>
</tr>
</tbody>
</table>

Data are expressed as mean (standard deviation). BW, body weight; BMI, body mass index; SBP, systolic blood pressure. DBP, diastolic blood pressure; HR, heart rate; GFR, glomerular filtration rate; HTN, hypertension; DM, diabetes mellitus.

**Table 2. Echocardiographic data before and after bariatric surgery**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pre-op</th>
<th>Post-op (1 year)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVTD (cm)</td>
<td>4.93 (0.52)</td>
<td>4.92 (0.47)</td>
<td>0.98</td>
</tr>
<tr>
<td>LVTS (cm³)</td>
<td>3.08 (0.48)</td>
<td>3.15 (0.43)</td>
<td>0.28</td>
</tr>
<tr>
<td>RWT</td>
<td>0.42 (0.09)</td>
<td>0.37 (0.06)</td>
<td>0.006</td>
</tr>
<tr>
<td>LVM (g/m²·7)</td>
<td>50.7 (11.07)</td>
<td>45.9 (16.04)</td>
<td>0.04</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>63.2 (19.1)</td>
<td>64.7 (13.03)</td>
<td>0.5</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>4.7 (1.4)</td>
<td>4.2 (0.9)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Diastolic function parameters

- E wave (cm/s) | 78.3 (16.9)| 88.8 (14.6) | <0.001 |
- A wave (cm/²) | 73.5 (18.2)| 66.4 (22.3) | 0.07    |
- e' wave (cm/²) | 9.5 (3.6)| 8.9 (2.5)   | 0.001   |
- E/A ratio | 1.14 (0.38)| 1.42 (0.42) | <0.001  |
- E/c’ ratio | 9.5 (3.8)| 10.9 (4.2)  | 0.07    |

Data are expressed as mean (standard deviation). LVTD, left ventricular telediastolic diameter. LVTS, left ventricular telesystolic diameter; CO, cardiac output; RWT, relative wall thickness; LVM, left ventricular mass index; E/A ratio, E/A waves ratio.

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Y-en-Roux gastric bypass was performed in 84% of patients (51 cases) and the remaining 16% received a tubular gastrectomy.

**Cardiac structure and function**

The data obtained in the echocardiographic study are presented in Table 2. Baseline left ventricular ejection fraction was 66.1 ± 7.95%. The stroke volume was 63.2 ± 19.1 mL and the cardiac output was 4.7 ± 1.4 L/min. The end-diastolic left ventricular diameter was 4.9 ± 0.5 cm while the end-systolic LVD was 3.08 ± 0.48 cm. Patients showed left ventricular remodelling as suggested by the RWT (0.42 ± 0.09) and the left ventricular mass index 50.7 ± 11.07 g/m².7, both in the upper normative limits. Diastolic function was abnormal in 27.9% of patients, with an altered E/A ratio.

**Renal function**

At baseline, the mean estimated GFR was 92.7 ± 25.4 mL/min. Hyperfiltration was present in 13.3% of patients, whereas an impaired GFR was detected in 8.3%. On a univariate analysis, only age was associated with a poor estimated GFR (mean age, 55.4 ± 2.8 versus 39.84 ± 9.2 years, P < 0.001). Other than this, the baseline-estimated GFR was not associated with any other clinical feature or co-morbidity including BMI, blood pressure, cardiac output, diabetes mellitus, left ventricular hypertrophy, left ventricular structure or diastolic function (data not shown).

**Postoperative findings: 1-year follow-up**

One year after surgery, the mean BMI decreased to 30.54 ± 5.07 kg/m², consistent with Grade 1 obesity. This...
impaired renal function (estimated GFR <60 mL/min). Moreover, our data show that bariatric surgery has a minimal impact on the estimated GFR in morbidly obese patients considered overall after a year of follow-up even with a sharp reduction in the BMI and cardiac output.

Ample discussion has focused on the most appropriate formula for estimating the GFR in obese patients since an overestimation has been reported with most of them. In the present study, we used the CG-LBW formula, which has been shown to be the most reliable in these types of patients since it is adjusted by lean body weight [15, 16]. The data demonstrated that baseline-estimated GFR, which shows a wide range, was not correlated with BMI, indicating that body weight does not have a major impact on renal function, at least in our cohort of patients. No correlation was found between other anthropometric, clinical or metabolic parameters such as glucose, blood pressure levels or the presence of other co-morbidities or estimated GFR’s. This was observed even in the reduced group of patients (13.3%) with hyperfiltration. This affirmation is further supported by the fact that improvement in these parameters after bariatric surgery was not accompanied by major changes in the estimated GFR. These results contrast with previous studies in which a higher incidence of hyperfiltration was found in morbidly obese patients [13, 18, 19]. This hyperfiltration was accompanied by variable increments in the filtration fraction and albumin excretion [20, 21]. However, the reduced number of patients (8.3%) with the estimated GFR <60 mL/min was older than those with estimated GFR >60 mL/min, suggesting that a longer evolution of obesity could be necessary in order to affect renal function. Hypertension was also more frequent among patients with a poor estimated GFR compared with others (75 versus 44%). Diabetes mellitus was uncommon among our patients, suggesting a limited effect on the deterioration of renal function in this group of patients. In fact, it has been suggested that obesity not only causes renal impairment, but also accelerates its deterioration [22, 23].

It is noteworthy that, although there are no significant changes in creatinine or the estimated GFR in the overall cohort, a reduced number of patients showed improvement in their renal function at the expense of the amelioration of their hyperfiltration. Although the difference did not reach statistical significance, probably due to a reduced sample size, this improvement was seen in patients with an increased stroke volume after surgery, suggesting a better cardiac performance. This idea can be further supported by the fact that these patients also had improved diastolic function. On the other hand, impairment in renal function mainly consisted of further increases in hyperfiltration, despite the amelioration of other metabolic and haemodynamic parameters that can modulate afferent arteriole tone and, in consequence, directly affect intraglomerular pressure [24]. Taken together, our findings suggest that bariatric surgery has little effect on renal function. However, some studies have reported a reduction in renal damage, supported by a reduction in proteinuria [13, 14, 25] being observed after surgery, although some complications have also been reported [21, 26].

Data regarding renal function in the context of cardiac performance in morbidly obese patients undergoing bariatric surgery are scarce. As expected, morbid obesity was accompanied in our patients by changes in cardiac function and structure, mainly characterized by the left ventricular hypertrophy and diastolic dysfunction, which confirms the results of previous studies [5, 27–29]. Several factors influence the development of cardiac remodelling. Load conditions, circulating volume and blood pressure hormones such as insulin and leptin, as well as neurohormonal activation with increased sympathetic tone are interrelated and ultimately lead to cardiac hypertrophy and geometric remodelling in an effort to adapt to the overall demands [6, 7, 28, 30]. Bariatric surgery leads to the cessation of stimuli for cardiac remodelling in several ways, beyond the weight loss. The progressive normalization in the load conditions and neurohormonal tone translate into regression of the cardiac hypertrophy to normal cardiac mass and geometric remodelling [5, 29, 31].

One striking finding was the association between improvements in diastolic function and renal function after surgery, despite the reduced number of patients in our study and considering the overall impairment of diastolic function observed in our patients. This lack of improvement contrasts with previous observations [29, 31, 32], but it is necessary to point out that, in our study, diastolic function has been addressed using Tissue Doppler analysis and not just pulsed-wave Doppler. Tissue Doppler velocities are less dependent on the haemodynamic status and load conditions, which clearly improve in our patients and may be a determinant of the observed improvements in pulsed-wave Doppler parameters [33]. This supports the hypothesis that obesity has deleterious effects on the elastic properties of the heart that may be irreversible even after the amelioration of other structural features of the heart. Persistent diastolic dysfunction may be a harbinger of ongoing myocardial dysfunction that may eventually lead to heart failure with preserved ejection fraction. Recent data have shown a significant reduction in long-term cardiovascular mortality after bariatric surgery. A longer follow-up may be necessary in order to elucidate the potential role of this subclinical heart damage over the years.

The relationship between diastolic and renal improvement is bilateral. Although our study was not primarily designed to address this issue, our finding reflects the importance of the association and suggests the need for further studies to explore the underlying mechanisms, as this is a high-risk group of patients.

Limitations
One potential limitation of the present study is a small sample size. Another concerns the fact that GFR evaluation could have been compromised by the use of creatinine, which is itself dependent on muscle mass and protein intake. While it is true that cystatin C could have been employed, or likewise radioisotopic filtration markers (i.e. 125I-iothalamate), which have been shown to accurately measure GFR especially in subjects with renal insufficiency, such techniques tend to pose their own limitations [34–36]. Therefore, further studies are needed.
to confirm that the present observations are not causal, but rather due to the interaction between renal and cardiac interactions in the context of the obesity. In addition, the lack of information regarding the protein excretion of these patients, which can give an idea of the degree of renal damage, could help to evaluate the consequence of weight loss in this regard. Finally, it is noteworthy to mention that in our cohort there was a reduced number of diabetic patients compared with other studies. This may change in the future since bariatric surgery is an emerging option for curing diabetes mellitus—the so-called ‘metabolic surgery’.

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Conflict of interest statement. None declared.

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