Subclinical Left Ventricular Dysfunction in Severe Obesity and Reverse Cardiac Remodeling after Bariatric Surgery

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Abstract

**Aim:** Obesity is associated with an increased cardiovascular risk. This study aimed to assess the role of echocardiography in the early detection of subclinical cardiac abnormalities in a cohort of obese patients with a preserved ejection fraction (EF) undergoing bariatric surgery.

**Methods and Results:** Forty consecutive severely obese patients (body mass index ≥35 kg/m²) referring to our center for bariatric surgery were enrolled in this prospective cohort study. Despite a baseline EF of 61% ± 3%, almost half patients (43%) had a systolic dysfunction (SD) defined as global longitudinal strain (GLS) < −18%, and most of them (60%) had left ventricular hypertrophy (LVH) or concentric remodeling (CR). At 10-months after surgery, body weight decreased from 120 ± 15 kg to 83 ± 12 kg, body mass index from 44 ± 5 kg/m² to 31 ± 5 kg/m² (both \( P < 0.001 \)). Septal and left ventricular posterior wall thickness decreased respectively from 10 ± 1 mm to 9 ± 1 mm (\( P = 0.004 \)) and from 10 ± 1 mm to 9 ± 1 mm (\( P = 0.007 \)). All systolic parameters improved: EF from 61% ± 3% to 64% ± 3% (\( P = 0.002 \)) and GLS from −17% ± 2% to −20% ± 1% (\( P < 0.001 \)). Epicardial fat thickness reduction (from 4.7 ± 1 mm to 3.5 ± 0.7 mm, \( P < 0.001 \)) correlated with the reduction of left atrial area (\( P = 0.001 \), \( R = 0.35 \)) and volume (\( P = 0.02 \), \( R = 0.25 \)). Following bariatric surgery, we observed a reduced prevalence of LVH/CR (before 60%, after 22%, \( P = 0.001 \)) and a complete resolution of preclinical SD (before 43%, after 0%, \( P < 0.001 \)). Moreover, a postoperative reduction of at least 30 kg correlated with regression of septal hypertrophy (\( P < 0.001 \)).

**Conclusions:** Obese patients candidate to bariatric surgery have an high prevalence of preclinical SD and LVH/CR, early detectable with echocardiography. Bariatric surgery is associated with reverse cardiac remodeling; it might also have a preventive effect on atrial fibrillation occurrence by reducing its substrate.

**Keywords:** Atrial fibrillation, diastolic function, left ventricular remodeling, Obesity, strain, systolic function

**Introduction**

Obesity is associated with an almost doubled risk of coronary artery disease, heart failure and sudden death regardless of age, cholesterol, systolic blood pressure, smoke, glucose intolerance, and left ventricular hypertrophy (LVH), as previously known from literature.\(^1\)\(^3\) The excess of adipose tissue affects the cardiovascular system through hemodynamic, inflammatory and metabolic modifications leading to a slow and subtle accumulation of epicardial fat, LVH, progressive diastolic and systolic dysfunction (SD).\(^4\) Furthermore, an increased volume of epicardial fat is associated with a greater risk of atrial arrhythmias such as paroxysmal and persistent atrial fibrillation (AF).\(^5\)

Significant weight loss obtained with bariatric surgery may lead to reverse cardiac remodeling, associated with beneficial effects on myocardial structure and systo-diastolic function.\(^6\)

In these high-risk patients preventive treatments are more effective when started before the onset of overt diastolic and SD. Novel diagnostic tools such as speckle-tracking echocardiography may detect subtle changes in myocardial systolic function even in asymptomatic patients with preserved ejection fraction (EF).\(^7\)

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This study was aimed to assess the role of echocardiography in the early detection of subclinical cardiac abnormalities in a cohort of obese patients undergoing bariatric surgery.

Methods

Study design

This prospective cohort study assessed forty consecutive patients undergoing bariatric surgery from January to October 2017 within our center. Oral and written informed consent was obtained from all patients before enrolment. Patients were included in the study if they were severely obese (body mass index [BMI] ≥35 kg/m²) and eligible for bariatric surgery. Exclusion criteria were past medical history of coronary artery disease, previously known hypertension or on treatment with any antihypertensive drug, a reduced EF (EF <52% in males, EF <54% in females), or echocardiographic images of insufficient quality for global longitudinal strain (GLS) analysis.

Endpoints of the study

The primary endpoint was the prevalence of morpho-functional cardiac abnormalities. The variation of morpho-functional parameters after bariatric surgery was the secondary endpoint.

Study protocol

All patients underwent a complete clinical and echocardiography assessment before surgery and 10 months later, including laboratory tests with assessment of serum N-terminal pro-brain natriuretic peptide (NT-proBNP).

Echocardiographic evaluation

Echocardiography was performed using Philips IE 33 with phased-array at frequency of 1.9–3.8 MHz. Standard apical, parasternal, subxiphoid, M-mode, color Doppler, pulse Doppler and tissue Doppler views were acquired. Dedicated software (QLab; Philips Medical Systems, Andover, MA, USA) was used for GLS analysis; volumes and EF were measured with the biplane Simpson’s method. All the echocardiograms were performed and examined according to the standards of the European Association of Cardiovascular Imaging guidelines.8

All images were acquired and off-line anonymously analyzed by a single cardiologist with broad experience in cardiovascular imaging (C.R.). Twenty examinations were then randomly and blinded re-assessed by C.R and a second expert echocardiographer (S.F.).

Both morphology and function of left ventricle were analyzed. End-diastolic diameter, end-diastolic volume (EDV) and end-systolic volume, septal and posterior wall thickness, mass and relative wall thickness along with atrial and ventricular volumes were measured. Left ventricular (LV) EF, lateral S2, GLS using speckle tracking technique along with right ventricle (RV) contractility (Tricuspid Annular Plane Systolic Excursion) were examined. E wave, A wave, E wave deceleration time, septal and lateral e’ velocity, were analyzed and E/A, septal E/e’, lateral E/e’ and average E/e’ ratios were calculated. Grading of diastolic function was performed according to ASE guidelines.9 Epicardial fat thickness was also measured by subcostal view. According to afore mentioned guidelines, increased LV EDV was defined as >150 ml in males and >106 ml in females. Increased septal thickness was defined as >10 mm in males, >9 mm in females. LVH was defined in the presence of an LV mass/body surface area ratio >115 in males, >95 in females. Relative wall thickness was used to discriminate patients with normal LV geometry, eccentric hypertrophy, concentric hypertrophy, concentric remodeling (CR). Increased left atrium (LA) area was defined as >20 cm². Abnormal GLS was defined as >−18%.

Reproducibility

Reproducibility of echo-Doppler and tissue Doppler measurements in our laboratory was previously reported.1011 For this study, variability was assessed in a randomly selected subset of 20 patients. Intraobserver coefficients of variation were 4.04% (P = 0.94), 4.56% (P = 0.90) and 9.07% (P = 0.88) for LV septal and posterior wall thickness and epicardial fat thickness, respectively and 2.15% (P = 0.95) for GLS. Corresponding intra class correlation coefficients were all included between 0.88 and 0.95. Interobserver coefficients of variation were 4.83% (P = 0.90), 5.01% (P = 0.88) and 10.64% (P = 0.84) for septal and posterior wall thickness and epicardial fat thickness, respectively and 3.12% for GLS, with corresponding intra class correlation coefficients of 0.82, 0.79, 0.70 and 0.81 respectively.

Statistical analysis

Continuous variables, presented as means and standard deviations, were compared by nonparametric tests: Mann–Whitney’s test was used for independent data and Wilcoxon’s signed-rank test for paired data (pre-post evaluations). Categorical variables, presented as counts and percentages, were compared using the Chi-square test with Yates’ correction or Fisher’s exact test. All analyses were performed using the SPSS for Windows version 18.0 (SPSS, Inc., Chicago, Illinois, USA) and a two-sided significance level of ≤0.05 was considered statistically significant. Univariate logistic analysis was used to determine the association between risk factors and cardiac remodeling. The relative risk was computed with its 95% confidence interval.

Results

This study enrolled forty patients (male: female ratio of 11:29), with a mean age of 42 ± 11 years and a BMI of 44 ± 5 kg/m² at the time of surgery. Thirty-five patients (88%) underwent sleeve gastrectomy, while five patients (13%) had gastric bypass. No one had hypertension, there were 15 (38%) current smokers, 7 (18%) with dyslipidemia, 4 (10%) with diabetes and 7 (18%) with a family medical history of heart disease. All demographics are reported in Table 1.

Baseline systolic and diastolic blood pressures during preoperative outpatient visit were, respectively, 144 ± 17 mmHg and 84 ± 14 mmHg.
Prevalence of morpho-functional cardiac abnormalities

Baseline echocardiography [Tables 2 and 3] revealed LVH or CR in 24 patients (60%), an abnormally increased septal thickness in 24 patients (60%), an abnormally increased LV EDV in 23 patients (58%), an abnormally increased LA area in 30 patients (75%).

Four patients (10%) had a Grade-I diastolic dysfunction, while other patients had a normal diastolic function. The average E/A ratio was 1.28 ± 0.34, while lateral E’ was 13.49 ± 3.65 cm/s and lateral E/e’ ratio was 6.31 ± 1.17. According to inclusion criteria, no patients had a reduced EF, although speckle tracking imaging analysis showed an abnormal GLS (>−18%) in 17 patients (43%). The mean epicardial fat thickness was 4.7 ± 1 mm.

Follow-up evaluation

After bariatric surgery it was observed a body weight decrease of 34 ± 12 kg (mean 29% ± 9 reduction, from 120 ± 15 kg to 83 ± 12 kg, P < 0.001). Twenty-four patients (61%) lost at least 30 kg. BMI decreased from 44 ± 5 kg/m² to 31 ± 5 kg/m² (P < 0.001), as described in Figure 1.

At 10-months echocardiographic assessment, LV, RV, LA and right atrium dimensions appeared all to be reduced; in particular, septal and LV posterior wall thickness decreased respectively from 10 ± 1 mm to 9 ± 1 mm (P = 0.004) and from 10 ± 1 mm to 9 ± 1 (P = 0.007). Compared with baseline assessment, no patient showed any degree of diastolic dysfunction at 10-month evaluation. Lateral E’ improved (from 13.49 ± 3.65 cm/s to 14.59 ± 3.25 cm/s, P = 0.029) as long as septal E’ (from 9.05 ± 1.95 cm/s to 10.19 ± 2.01 cm/s, P = 0.022), lateral E/e’ ratio (from 6.31 ± 1.17 to 5.82 ± 1.06, P = 0.012) and septal E/e’ ratio (from 9.24 ± 1.68 to 8.32 ± 1.80, P = 0.023).

Systolic parameters were also improved at 10-month evaluation: EF varied from 61% ± 3% to 64% ± 3% (P = 0.002) and GLS varied from −17% ± 2% to −20% ± 1% (P < 0.001). Lastly, epicardial fat thickness decreased from 4.7 ± 1 mm to 3.5 ± 0.7 (P < 0.001) and NT-proBNP level increased from 29 ± 29 pg/ml to 52 ± 32 pg/ml (P = 0.002). All echocardiographic measurements are described in Table 3.

Reverse cardiac remodeling

As shown in Figure 2 and Table 2, at 10-months after bariatric surgery, the prevalence of LVH or CR reduced from 60% to 22% (P = 0.001), abnormally increased septal thickness reduced from 60% to 25% (P = 0.003), abnormally increased LV EDV reduced from 58% to 22% (P = 0.003), LVH reduced from 25% to 5% (0.028), and abnormally increased LA area reduced from 75% to 35% (P = 0.001).

Besides, follow-up echocardiographies showed a complete resolution of subclinical SD with GLS>−18%: Previously observed in 43% patients, afterwards in none, P < 0.01.

Furthermore, losing at least 30 kg through bariatric surgery was associated with regression of septal hypertrophy (P < 0.01).

Weight loss correlated with septal thickness reduction (P = 0.02 R = 0.125) and with RV diameter decrease (P = 0.03

| Table 1: Demographics at baseline |
|----------------------------------|
| Variable                        | n (%) | mean (SD)  |
|----------------------------------|-------|------------|
| Female gender                    | 29 (73)|            |
| Age, years                       | 42±11            |
| Type of bariatric surgery        |       |            |
| Sleeve gastrectomy               | 35 (88%)          |
| Gastric bypass                   | 5 (13%)           |
| Height, cm                       | 166±7            |
| Weight, kg                       | 120±15           |
| BMI, kg/sqm                      | 44±5            |
| Systolic blood pressure, mmHg    | 144±17           |
| Diastolic blood pressure, mmHg   | 84±14            |
| Cardiovascular Risk Factors      |       |            |
| Hypertension                     | 0               |
| Current smoke                    | 15 (38%)          |
| Dyslipidemia                     | 7 (18%)          |
| Diabetes mellitus                | 4 (10%)          |
| Family medical history of heart disease | 7 (18%)          |
| Haemoglobin, g/dL                | 13.8±1.6          |
| Glucose, mg/dL                   | 94±18            |
| Creatinine, mg/dL                | 0.72±0.15         |
| eGFR (ml/min/sqm)                | 207±47           |
| NT-proBNP (pg/ml)                | 29±29            |

BMI, body mass index; eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal prohormone of brain natriuretic peptide

R = 0.19. Furthermore, the reduction of epicardial fat thickness correlated with LA area decrease (P = 0.01, R = 0.35) and with LA volume reduction (P = 0.02, R = 0.25). All correlations are shown in Figure 3.

DISCUSSION

According to the well-known pathophysiological cascade in obese subjects, as explained by Alpert MA, hemodynamic alterations produced by obesity induce LV dilatation, leading to LVH because of elevated LV wall stress. Therefore, the presence of LVH predisposes to LV diastolic dysfunction; eventually, if LV wall stress remain chronically elevated because of inadequate LVH, LV SD may ensue. According to these previous findings, in our study we observed a high prevalence of LV dilatation and LV remodeling (LVR) or LVH (respectively 58% and 60%), a smaller prevalence of diastolic dysfunction (10%) and an even lower prevalence of SD (no patient had a compromised...
EF), despite a high proportion (43%) of patients with an abnormal GLS.

In fact, the documentation of a preserved EF by echocardiography does not exclude the possible presence of subtle alterations in LV myocardial composition and/or geometry. Speckle tracking echocardiography, assessing myocardial strain, provides more detailed information on global and regional active LV deformation, allowing to detect the presence of a subclinical SD.\[13\] In our study we found a reduced GLS (>−18%) in 43% of patients, with a mean value of −17% ± 2%. This is in line with the study of Koshino \textit{et al.}, where 28 obese patients undergoing bariatric surgery, with a mean BMI of 51, presented −11% ± 4% as GLS value at baseline assessment.\[14\] These data support the hypothesis that even in obesity cardiomyopathy the apparently isolated diastolic dysfunction (with preserved EF), may be associated with subclinical SD, as already

![Figure 2: Reverse cardiac remodeling](image)

![Figure 3: Correlations](image)

**Table 2: Morpho-functional cardiac abnormalities**

| Condition                                    | Before $n$ (%)/mean (SD) | After $n$ (%)/mean (SD) | $P$   |
|----------------------------------------------|--------------------------|-------------------------|-------|
| Increased LV end diastolic volume            | 23 (58%)                 | 9 (22%)                 | 0.003 |
| Increased septal thickness                   | 24 (60%)                 | 10 (25%)                | 0.003 |
| Normal LV geometry                           | 16 (40%)                 | 31 (78%)                | 0.001 |
| LV hypertrophy or concentric remodeling      | 24 (60%)                 | 9 (22%)                 | 0.001 |
| LV hypertrophy                               | 10 (25%)                 | 2 (5%)                  | 0.028 |
| Concentric hypertrophy                       | 7 (17%)                  | 2 (5%)                  | 0.157 |
| Eccentric hypertrophy                        | 3 (8%)                   | 0                       | 0.124 |
| LV concentric remodeling                     | 14 (35%)                 | 7 (18%)                 | 0.127 |
| Increased LA area                            | 30 (75%)                 | 14 (35%)                | 0.001 |
| Diastolic dysfunction                        | 4 (10%)                  | 0                       | 0.124 |
| GLS >−18%                                    | 17 (43%)                 | 0                       | <0.001|
| Epicardial fat thickness, mm                 | 4.7±1                    | 3.5±0.7                 | <0.001|

LV, left ventricular; LA, left atrial; GLS, global longitudinal strain.
demonstrated for other cardiomyopathies by Pacileo et al.\(^{15}\). Therefore, the assessment of obese patients with speckle tracking echocardiography may help to identify patients with higher cardiovascular risk by detecting subclinical SD earlier than clinical overt manifestations, allowing them to receive more intensive controls and earlier interventions. According to the American Society for Metabolic and Bariatric Surgery, bariatric surgery is recommended for obese people with BMI ≥40 kg/m\(^2\), with BMI ≥35 kg/m\(^2\) and obesity-related comorbidities and for patients unable to achieve weight loss in other ways; thus, subclinical SD may be considered an obesity-related comorbidity and allow obese patient to receive earlier bariatric surgery.

As described by Cuspidi et al., bariatric surgery exerts important cardioprotective effects in morbidly obese patients.

### Table 3: 10-months echocardiography

|                      | Before       | After        | Delta (%) | P     |
|----------------------|--------------|--------------|-----------|-------|
| **Weight, kg**       | 120±15       | 83±12        | -34 (-29%)| <0.001|
| **BMI, kg/sqm**      | 44±5         | 31±5         | -12 (-29%)| <0.001|
| **Epicardial fat, mm** | 4.7±1        | 3.5±0.7      | -1.2 (-24%)| <0.001|
| **Left ventricle**   |              |              |           |       |
| End-diastolic diameter, mm | 48±3        | 47±3         | -1 (-2%)  | 0.133 |
| End-diastolic volume, ml | 117±24       | 101±20       | -16 (-12%)| 0.001 |
| End-systolic volume, ml | 44±10        | 37±9         | -8 (-16%) | <0.001|
| Septal thickness, mm | 10±1         | 9±1          | -1 (-9%)  | 0.004 |
| Posterior wall thickness, mm | 10±1        | 9±1          | -1 (-9%)  | 0.007 |
| Mass, g              | 197±41       | 163±30       | -34 (-17%)| 0.031 |
| **Right ventricle**  |              |              |           |       |
| Diastolic diameter, mm | 37±3         | 36±3         | -1 (-3%)  | 0.029 |
| TAPSE, mm            | 23±4         | 24±5         | +1 (5%)   | 0.512 |
| PAPs, mmHg           | 28±4         | 27±4         | +1 (+2%)  | 0.516 |
| **Left atrium**      |              |              |           |       |
| Area, cm\(^2\)      | 18±3         | 16±2         | +2 (+10%) | 0.009 |
| Volume, ml           | 51±12        | 43±10        | -9 (-16%) | 0.002 |
| **Aorta**            |              |              |           |       |
| Aortic root, mm      | 31±3         | 32±3         | +0 (0%)   | 0.272 |
| Ascending aorta, mm  | 30±3         | 31±3         | +1 (+3%)  | 0.338 |
| **Systolic function**|              |              |           |       |
| EF, %                | 61±3         | 64±3         | +2 (+4%)  | 0.002 |
| Lateral S\(_2\), cm/s | 9.8±1.2      | 10.4±1.5     | +1 (+7%)  | 0.020 |
| GLS, %               | -17±2        | -20±1        | +3 (+19%) | <0.001|
| **Diastolic function**|              |              |           |       |
| E, cm/s              | 82±14        | 83±15        | +0 (0%)   | 0.738 |
| A, cm/s              | 67±14        | 66±16        | -1 (-2%)  | 0.764 |
| E/A                  | 1.28±0.34    | 1.35±0.43    | +0.06 (+5%)| 0.412 |
| DT, ms               | 173±31       | 183±24       | +10 (+6%) | 0.156 |
| Lateral e\(_',\) cm/s | 13.49±3.65   | 14.59±3.25   | +1.11 (+10%)| 0.029 |
| Lateral E/e\(_'\)    | 6.31±1.17    | 5.82±1.06    | -0.49 (+7%)| 0.012 |
| Septal e\(_',\) cm/s | 9.05±1.95    | 10.19±2.01   | +1.13 (+15%)| 0.022 |
| Septal E/e\(_'\)     | 9.24±1.68    | 8.32±1.80    | -0.96 (+9%)| 0.023 |
| **Valves**           |              |              |           |       |
| Aortic insufficiency | 0 (0%)       | 5 (13%)      |           |       |
| Mitral insufficiency | 16 (39%)     | 14 (35%)     |           |       |
| Tricuspid insufficiency | 10 (26%)   | 10 (26%)     |           |       |
| Pulmonary insufficiency | 0 (0%)    | 0 (0%)       |           |       |
| NT-proBNP, pg/ml     | 29±29        | 52±32        | +23 (+296%)| 0.002 |

BMI, body mass index; AP, antero-posterior; SI, superior-inferior; TAPSE, tricuspid annular plane systolic excursion; PAPs, systolic pulmonary artery pressure; EF, ejection fraction; GLS, global longitudinal strain; DT, deceleration time, NT-proBNP, N-terminal prohormone of brain natriuretic peptide.
through LVH regression, improvement in LV geometry and diastolic function, reduction of left atrial size. According to this explanation and in line with data from similar studies such as Shin et al., Mostfa and Kurnicka et al., in our study many structural and functional parameters improved significantly after bariatric surgery [Tables 2 and 3] and 10-months echocardiography showed a significant reduction of LV dilatation, septal hypertrophy, a normalization of LVR/LVH along with a resolution of subclinical SD. Diastolic dysfunction was no longer evident after bariatric surgery. Most diastolic parameters significantly improved at 10-month assessment, especially lateral and septal E/e’ ratios which are surrogate indexes of LV filling pressures.

Furthermore, an interesting outcome of this study is that losing at least 30 kg leads to regression of septal hypertrophy ($P < 0.001$).

The association between LA dilatation and AF has been known in literature for years and many studies proved it; actually, in the population of the Framingham Heart Study it appeared that there was a 39% increase in AF risk every 5 mm increment of LA diameter.

Less is known about the association between epicardial fat and AF. Epicardial fat is a unique fat compartment located between the myocardial surface and the visceral layer of the pericardium; among its physiological functions there are myocardial protection against hypothermia, mechanical protection for coronary circulation and energy source in the homeostasis of the myocardium; but epicardial fat is also considered a source of inflammatory mediators that might directly influence myocardium and coronary arteries. Recently, it also appears that this tissue, when abundant, penetrates deeply into the heart and invades spaces such as the interatrial septum.

Its thickness is highly associated with paroxysmal and persistent AF, independently of traditional risk factors. In this study, 10-months echocardiography following bariatric surgery showed not only a significant decrease in epicardial fat thickness, but also a decrease in LA size (diameter, area, volume). Furthermore, epicardial fat reduction was linearly correlated with LA area decrease and LA volume reduction.

These data support the hypothesis that in obese patients both epicardial fat deposition and LA dilatation are the results of a negative cardiac remodeling. Abundant epicardial fat and significant LA dilatation can be ideal substrates for atrial arrhythmias such as AF. In this context, weight loss obtained with bariatric surgery was associated with a 10-month reverse remodeling able to reduce both substrates and then potentially able to act as a protective factor on AF.

Obese patients also have considerably lower plasma natriuretic peptide levels than individuals with a normal BMI, resulting in an even more complicated diagnosis and management of heart failure. The present study shows a significant increase of serum NT-proBNP after bariatric surgery in line with many articles, such as Chen-Tournoux et al. one where 132 obese subjects present five times increased level of serum NT-proBNP 6 months after weight loss surgery, which is not attributable to clinical situations that usually upregulate its secretion because of well demonstrated cardiac improvements after bariatric surgery.

Limitations
This study has some limitations: It did not evaluate major adverse cardiac events; it was a monocentric study; follow-up was limited to 10 months. Furthermore, AF was not evaluated as an end-point. Blood pressure values were not recorded throughout the follow-up. Further studies could be helpful to assess the incidence of clinical events.

Conclusions
This study shows that speckle tracking echocardiography may detect subclinical SD in obese patients with a normal EF, allowing early detection of those at higher risk of overt clinical events.

Bariatric surgery is associated with significant 10-month reverse cardiac remodeling and might exert a possible protective effect on AF occurrence by reducing its substrate.

Informed consent
Informed consent was obtained from all individual participants included in the study.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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