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Original Article

Impact of obesity and surgical weight reduction on cardiac remodeling

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ABSTRACT

Background: the implication of obesity on cardiometabolic risk factors and incident diabetes has been previously demonstrated, but the impact of weight changes on cardiac structure independent of obesity-related comorbidities has not been extensively studied
Aim: to study impact of obesity and surgical weight reduction on cardiac structure.
Patients and methods: fifty two patients withbody mass index (BMI) $\geq$40 kg/m^2, free of previous or overt cardiac risk factors and diseases were included, all patients underwent bariatric surgery; Conventional echocardiography (2D, M-Mode, Doppler), tissue Doppler velocity (TDI), strain and speckle tracking echocardiography for left and right ventricles were performed before and 6 m after surgery.
Results: mean age was 38.2 $\pm$ 5.6, BMI 42.3 $\pm$ 3.4 kg/m^2, 65% were female and 35% were male. 6 months postopeatively; there was significant increase of left ventricular end systolic volume (LVESV) and left ventricular end diastolic volume (LVEDV) from 66.57 $\pm$ 22 to 37.2 $\pm$ 12 $p < 0.001$, and 169.4 $\pm$ 43.2 to 120.36 $\pm$ 19.6 ml with $p < 0.001$ respectively and increased ejection fraction (EF%) from 59 $\pm$ 8 to 67 $\pm$ 7 ml $p < 0.001$, significant reduction in left ventricular mass index (LVMI) from 143 $\pm$ 11 to 95.5 $\pm$ 7gm/m^2 $p < 0.001$. Significant increase in right ventricular systolic area (RVSA) from 16.3 $\pm$ 4.1 to 10.1 $\pm$ 2.7 cm^2 $p < 0.001$ but was insignificant in right ventricular diastolic area (RVDA) from 30.2 $\pm$ 1.5 to 26.7 $\pm$ 2 cm^2 $p = 0.05$, fraction area change (FAC) from 49.5 $\pm$ 2.1 to 52 $\pm$ 1.2% $p = 0.7$, Tricuspid annular plane systolic exertion (TAPSE) from 20.3 $\pm$ 2.8to22.6 $\pm$ 3.5 mm, $p = 0.56$and pulmonary arterty systolic pressure (PASP) from 32.2 $\pm$ 5.2 to 29.2 $\pm$ 2.1 mmHg, $p = 0.81$.Early tissue Doppler diastolic velocity (Em) of the LV increased from 7.1 $\pm$ 2.1 to12 $\pm$ 3.5 $p < 0.001$ and that of RV from 6.2 $\pm$ 2.8 to 9.2 $\pm$ 1.4, $p = 0.05$ and tissue Doppler strain of the LV and RV invrased from -16.1 ± 2.5 to -22.8 ± 3.1, $p < 0.001$, -11.2 ± 2.6 to -17.3 ± 3.4, $p < 0.001$ respectively. Left ventricular longtiduinal systolic strain (LVLPSS) increased from -17.2 ± 2.1 to -22.7 ± 3.9 $p < 0.001$ and right ventricular longtiduinal systolic strain (RVLPSS) increased from -12.8 ± 1.5 to -18.1 ± 2.7 $p < 0.001$.
Conclusion: Obesity adversely affects cardiac function independent of obesity-related comorbidities . Weight reduction significantly increase the systolic and diastolic function of both ventricles.

1. Introduction

Obesity (BMI >30 kg/m^2) is an independent risk factor for incident heart failure (HF) .Obesity adversely affects the circulatory system with resultant endothelial dysfunction resulting in systemic hypertension, coronary artery disease, and vascular calcification. In addition, obesity causes changes in the heart including an increase in left ventricular (LV) mass, LV hypertrophy, LV and left atrial (LA) dilatation, and diastolic as well as systolic dysfunction in some cases.[1] Also in obesity, the Frank–Starling curve is shifted to the left because of increase in LV filling pressure and volume resulting in LV dilatation and, eventually, systolic dysfunction.[2]

Traditional treatments to achieve weight loss, such as diet, lifestyle, and behavioral therapy have proven relatively ineffective in treating morbidly obese patients with BMI >40 kg/m2 especially when used in isolation.[3]

Surgery for the treatment of morbid obesity can be offered according to guidelines established by the National Institutes of Health (United States) and the National Institute for Clinical Excellence (United Kingdom).[4]

Surgical procedures can be classified into 3 categories: restrictive, malabsorptive, or combination procedures. Restrictive operations decrease the size of the stomach (either by a synthetic gastric band, stapling, or size reduction by “sleeve gastrectomy”), leading to satiety with smaller volumes of food that eventually leads to food intolerance and weight loss. Malabsorptive

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operations consist of bypassing segments of bowel, which thereby cause malabsorption of nutrients (such as the biliopancreatic diversion with or without duodenal switch and ileal interposition). The combination group of operations involves both aspects of restriction and malabsorption such as the Roux-en-Y gastric bypass.[5]

Although the implication of weight change on cardiometabolic risk factors and incident diabetes in obesity has been previously demonstrated, but the impact of weight changes on cardiac structure independent of obesity-related comorbidities has not been extensively studied.[6]

1.1. Aim

The aim was to study the impact of obesity and surgical weight reduction on cardiac remodeling

1.2. Patients and methods

Single centre, prospective, observational study included 85 adult patients >18y of both genders with morbid obesity (BMI $>40\ kg/m^2$) undergoing bariatric surgery (laparoscopic gastric band or ligation) in the period from december 2015 to January 2017 at Benha university hospital with follow up for 6 months after surgery

The local ethics committee has approved the study and the subjects gave written informed consent.

1.3. Exclusion criteria

Patients with pervious or overt cardiac risk factors and diseases that affect cardiac structure or function as smoking, diabetes, hypertension, dyslipidemia, family history of coronary heart disease, rheumatic heart disease, impaired systolic function, asthmatic bronchitis, chronic renal or liver impairment, failure to assess 17 segments 2 D strain or operative complication were excluded from the study. So only 52 patients fulfilled the inclusion criteria.

2. Methods

History: to exclude diabetis, hypersnion, previous admission to hospital with cardia problems, long acting penicillin or bronchial asthma.

Clinical examinaion: weight and height to measure BMI, blood pressure and heart rate assessment, complete chest and local cardiac examition.

Laboratory: CBC, fasting and 2 h postpredal blood sugar, lipid profile, liver function and kidney fnction.

ECG: 12 lead resting ECG to exclude ischmic changes or rythem disturbance

2.1. Echocardiography

All echocardiographic evaluations were performed by a VIVID 7 echocardiography equipment (General electric, Horten, Norway) using a 2.5-mHz transducer

- **By 2D echo**: LVEDV, LVESV and EF% were measured according to the modified Simpson's method and RVSA, RVDA to calculate RV fractional area change then LA volume measured and index to body surface area.
- **By M-Mode**: LV internal systolic and diastolic dimensions and wall thickness were measured. Left ventricular mass (LVM) was calculated using the formula that has been proposed by Devereux,[7] and normalized for body surface area to obtain the LV mass index (LVMI). Left ventricular hypertrophy (LVH) was defined as LVMI of $131\ g/m^2$ for men and $100\ g/m^2$ for women.

LV mass (Penn) = 1.04 ([LVIDD + PWTD + IVSTD]3- [LVIDD]3) −13,6 g

BSA = (W 0.425x H 0.725) x 0.007184 [7]

TAPSI of the lateral leaflet of the tricuspid valve in the apical 4 chamber view or in the short axis view at the level of aortic valve.

- **CW Doppler**: Pulmonary artery systolic pressures (PASP) was estimated by the maximum velocity of the tricuspid regurgitant jet using the modified Bernoulli equation and then adding to this value an estimated right atrial pressure.
- **Doppler Tissue Imaging**: pulsed wave Doppler tissue imaging (DTI) was performed in the apical views to acquire mitral and tricusped annular velocities. Measurements included the systolic (S), early diastolic (E′), and late diastolic (A′) velocities.[8]
- **Strain Doppler Method**: Doppler-derived strain was obtained by placing a 10-mm sample bar at the basal part of the 6 LV walls in the 3 different apical views. The image sector was narrowed to allow for the highest frame rate (>200 frames/s), and the imaging angle was kept as low as possible (usually below 30°) to allow for a better parallel alignment to the wall of interest**.** The data are stored in digital format and analyzed offline by dedicated software that allows calculating local peak systolic strain.[8]
- **Left and right ventricular global longitudinal strain**: standard two-dimensional grey scale loops of the LV were acquired in conventional apical four-chamber, two chamber and long axis views. Data were stored digitally and transferred for off-line analysis, special care was taken to ensure frame rates of between 50 and 90 frames per second in all patients. The regions of interest were defined manually by marking the endocardial border, the automatic tracking of endocardial contour was verified carefully and the region of interest was corrected manually to ensure optimal tracking of the entire myocardial wall. Segmental strain analysis was performed by dividing each left ventricular image into six segments; peak systolic longitudinal strain was calculated by averaging the peak systolic value of the eighteen segments, derived from the three apical views.[9]
- All echo reports were read in a blinded manner by three cardiologist; Inter- and intra-rater correlation was calculated using the Kappa test (poor agreement, 0.40; moderate agreement, 0.40-0.59; good agreement, 0.60–0.74; excellent agreement, 0.75–1.00).

2.2. Statistical analysis

Results are presented as mean ± SD for continuous variables. Results are presented as absolute numbers or percentages for categorical variables. Differences in continuous variables between groups were tested using independent *t*-test, Mann-Whitney *U* test or paired *t*-test, as appropriate. Differences in categorical variables between groups were tested using the x^2 test with Yates Correction. All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS 16.0, Inc., IL, USA). A P-value of 0.05 was considered statistically significant.

2.3. Results

The study included 85 adult patients with morbid obesity undergoing restrictive bariatric surgery (laparoscopic gastric

band or ligation), 33 patients were excluded so the study included 52 patients with mean age of 38.2 ± 5.6 years and a body mass index (BMI) of 42.3 ± 3.4 kg/m^2, 65% were female and 35% were male.

Conventional echocardiography (2D, MM, Doppler), tissue Doppler velocity and strain and speckle tracking echocardiography for left and right ventricles were performed to all patients before and 6 m after surgery.

After 6 months of follow up 49 patients (94%) achieved significant reduction of body weight from 130.7 ± 27.3 kg to 90.5 ± 24.4 kg ($p < 0.01$). The mean percentage weight loss was 20 ± 7% of baseline weight and BMI from 42.3 to 28.5 kg/m^2 ($p < 0.001$).

2.3.1. Conventional echo parameters

There were dilated LV volumes with normal ejection fraction; LV parameters showed statistically significant improvement after surgery; LVESV improved from 66.57 ± 22 to 37.2 ± 12 $p < 0.001$, LVEDV improved from169.4 ± 43.2 to 120.36 ± 19.6 ml with $p < 0.001$ and EF% 59 ± 8 to 67 ± 7 ml with $p < 0.001$. Also, there was significant reduction in LVMI from143 ± 11 to 95.5 ± 7gm/m^2 $p < 0.001$ but LA volume index didn't show significant change p = 0.75. Table 1

As for the RV there was insignificant change in the RVSA from 12.3 ± 4.1 to 10.1 ± 2.7 cm^2 $p < 0.001$, RVDA from 29.2 ± 1.5 to 27.7 ± 2 cm^2 p = 0.05, FAC from 49.5 ± 2.1 to 52 ± 1.2% p = 0.7, TAPSI from 20.3 ± 2.8 to 22.6 ± 3.5 mm p = 0.56 and PAS from 32.2 ± 5.2 to 29.2 ± 2.1 mmHg with p = 0.81. Table 1

2.3.2. TDI velocity and strain

There were significant improvement in the both ventricular systolic and diastolic functions that were detected by TDI velocity and strain after surgery as early tissue Doppler diastolic velocity (Em) of the LV improved from 7.1 ± 2.1 to12 ± 3.5 $p < 0.001$ and that of RV from 6.2 ± 2.8 to 9.2 ± 1.4 p = 0.05 and tissue Doppler strain of the LV and RV improved from −16.1 ± 2.5 to −22.8 ± 3.1 $p < 0.001$, −11.2 ± 2.6 to −17.3 ± 3.4 $p < 0.001$ respectively. Table 1

2.3.3. Longitudinal peak systolic strain

There was statistically significant improvement in LPSS of both ventricles postoperatively as LVLPSS improved from −17.2 ± 2.1 to −22.7 ± 3.9 $p < 0.001$ Fig. 1 and RVLPSS improved from −12.8 ± 1.5 to −18.1 ± 2.7 $p < 0.001$. Table 1

2.4. Discussion

Obesity may contribute to the development and progression of cardiac dysfunction through several mechanisms. Ventricular remodeling is common, with LV eccentric hypertrophy developing in response to the expanded intravascular volume present in obesity. Afterload is elevated, not only because of increased preload but also because of elevated vascular resistance caused by excess adipose tissue and higher conduit artery stiffness.20 Similar to other studies.[10]

Impaired LV systolic function in persons with obesity occurs only in the presence of coexisting heart disease or other risk factors, where adverse loading conditions and duration of obesity may contribute to LV systolic dysfunction.[11]

Most echocardiographic studies assessing left ventricular (LV) systolic function in obese patients used ejection phase indices like LV ejection fraction and LV fractional shortening, reported normal or hyperdynamic LV systolic function and no or little differences in lean and obese subjects.[11]

Most of studies demonstrated the value of weight reduction on improvement of left ventricular function that was correlated to modification of the associated risk factors as diabetes, hypertension or dyslipidemia but limited studies evaluated obese patients without associated risk factors or diseases and its effect on cardiac function.

The present study included fifty two patients free of either previous or overt cardiac risk factors and diseases that affect cardiac structure and remodeling. All patients underwent bariatric surgery (laparoscopic gastric band or ligation); conventional echocardiography (2D, M-Mode, Doppler), tissue Doppler velocity, strain and speckle tracking echocardiography for left and right ventricles were performed for all patients before and 6 m after surgery.

Conventional echo parameters showed that inspite of increased LV volumes befor surgery there was normal systolic function, but after surgery there was significant decrease of LVESV and LVEDV from 66.57 ± 22 to 37.2 ± 12 ($p < 0.001$), and from 169.4 ± 43.2 to 120.36 ± 19.6 ml ($p < 0.001$) respectively, with increased EF% from 59 ± 8 to 67 ± 7 ml ($p < 0.001$) and also there was significant decrease in LVMI from143 ± 11 to 95.5 ± 7gm/m^2 ($p < 0.001$).

These results were in agreement with Graziani et al.[12] who showed a significant decrease in weight and BMI paralleled by a significant reduction of left ventricular (LV) mass and LV end-

Table 1
comparison of the echo parameters before and after surgery.

		Before surgery	After surgery	P value
LA volume index(mm/m^2)		32.3 ± 3.4 g/m^2	28.2 ± 3.2	0.75
LV systolic function	LVESV(mL)	66.57 ± 22	37.2 ± 12	<0.001
	LVEDV(mL)	169.4 ± 43.2	120.36 ± 19.6	<0.001
	EF%	59 ± 8	67 ± 7	<0.001
LMVI gm/m^2		143 ± 11	95.5 ± 7	<0.001
RV systolic function	RVSA(cm2)	12.3 ± 4.1	10.1 ± 2.7	0.05
	RVDA(cm2)	29.2 ± 1.5	27.7 ± 2	0.05
	FAC%	49.5 ± 2.1	52 ± 1.2	0.7
TAPSI (mm)		20.3 ± 2.8	22.6 ± 3.5	0.56
PASP (mmHg)		32.2 ± 5.2	29.2 ± 2.1	0.81
TDI velocity	Em of LV free wall	7.1 ± 2.1	12 ± 3.5	<0.001
(cm/sec)	Em of RV free wall	6.2 ± 2.8	9.2 ± 1.4	0.05
	E/e'	9.1 ± 4.2	7.8 ± 2.0	0.05
TDI strain (%)	Strain of LV free wall	−16.1 ± 2.5	−22.8± 3.1	<0.001
	Strain of RV free wall	−11.2 ± 2.6	−17.3 ± 3.4	<0.001
LPSS (%)	LVLPSS	−17.2 ± 2.1	−22.7 ± 3.9	<0.001
	RVLPSS	−12.9 ± 1.5	−17.5 ± 2.7	<0.001

BMI, body mass index; LV, LVEDV, LV end-diastolic volume; LVESV, LV end-systolic volume; LVEF, LV ejection fraction; LVMI: LV mass index; RVEDA, RV end-diastolic area; RVESA, RV end-systolic area; FAC,fractional area change; TAPSE, Tricuspid annular plane systolic exertion;PASP, pulmonary artery systolic pressure,. TDI; tissue Doppler image; LPSS, longitudinal peak systolic strain; LVLPSS,LV longitudinal peak systolic strain; RVLPSS, RV longitudinal peak systolic strain.

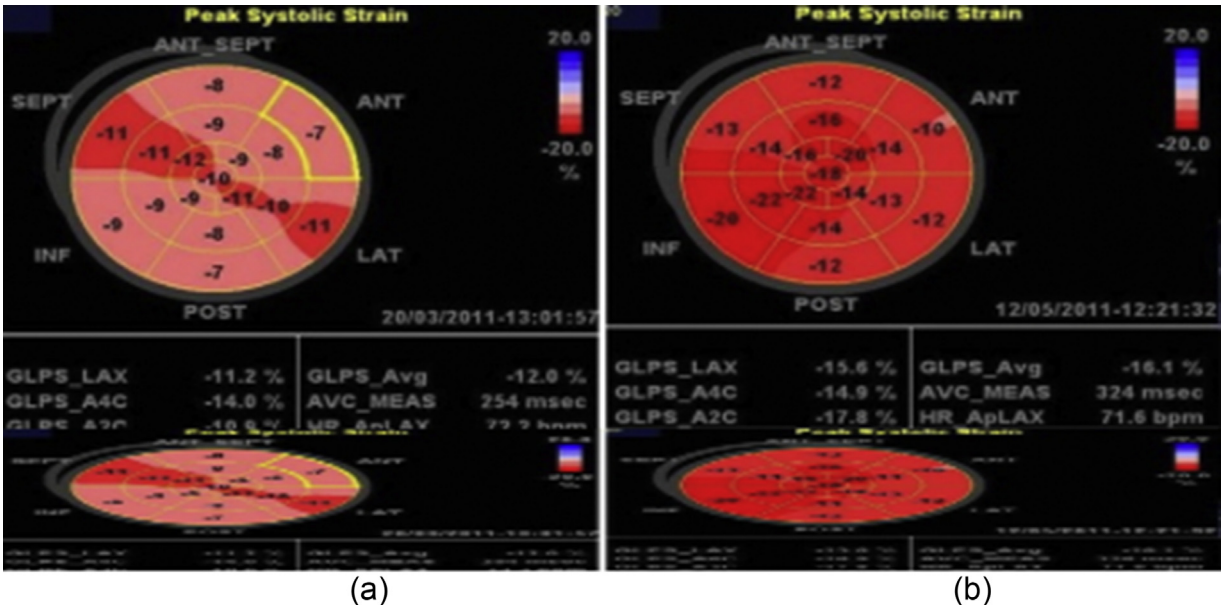

Fig. 1. Female patient 39y old, obese with BMI 42 kg/m^2. LV global longitudinal strain-12% before surgery (a), increased to −16.1% 6 months after surgery (b).

diastolic and end-systolic volumes and a significant increase of LV ejection fraction.

Sevda et al.[13] found that LV ejection phase indices, LV ejection fraction and fractional shortening, were normal at baseline bu they didn't demonstrate significant changes at follow-up, which can be explained by shorter follow up period that was only for only 3months.

The present study showed that tissue Doppler velocity, strain and speckle tracking of both LV and RV that showed statistically significant increase 6 m after surgery.

Improved echocardiography techniques suggests that all obese patients harbor a degree of systolic dysfunction, albeit subclinically in milder forms, such that only subtle markers of systolic dysfunction might be demonstrable such as basal septal strain.[14]

Barbosa et al.[10] showed that morbidly obese patients have lower indices of LV and RV systolic and diastolic functions when compared with healthy controls, and they concluded that strain imaging may provide a more accurate assessment of the ventricular function in obese patients.

Despite the well-documented relationship between obesity and heart failure, there has been no reliable predictor to evaluate ventricular function in patients with obesity. LVEF is the most popular method to assess LV systolic function, but Koshino et al.[15] performed the first study showing an association between obesity and myocardial performance as measured by strain and strain rate using 2D-speckle tracking in people without LV systolic dysfunction.

Koshino et al.[15] studied changes in myocardial mechanics in patients with obesity following major weight loss after bariatric surgery and found that LV and RV myocardial contractility, as measured by longitudinal strain was significantly reduced in patients with obesity and after bariatric surgery strain increased that was accompanied by major weight loss.

2.5. Clinical implication

Conventional echo parameters were not sufficient for evaluating the left and right sides of the heart and can't detect subclinical affection that can be easily detected by the tissue Doppler, strain and speckle tracking. So preoperative evaluation of the morbid obese patients is better to be by advanced echo techniques.

2.6. Conclusion

Obesity adversely affects cardiac structure independent of obesity-related comorbidities. Weight reduction significantly improves the systolic and diastolic function of both ventricles.

2.7. Limitations

First, a small number of patients were included in the study as it was difficult to apply the inclusion criteria. Second to compare the value of surgical weight reduction to diet regimen as only limited patients achieved significant weight reduction on diet regimen.

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