Effects of weight loss on ventricular systolic and diastolic functions and left ventricular mass assessed by tissue doppler imaging in obese geriatric women: preliminary report*

Murat Varli[1], Sibel Turhan[2], Sevgi Aras[1], Teslime Atli[1] and Gurbuz Erdogan[3]

[1]Department of Geriatric Medicine and [2]Department of Cardiology, Ankara University School of Medicine, Cebeci, Ankara, [3]Department of Endocrinology and Metabolic Diseases, Ufuk University School of Medicine, Balgat, Ankara, Turkey

ABSTRACT. ***Background and aims:*** *Obesity is one of the most common diseases in the world. Particularly in elderly subjects, the effects of weight loss on cardiac functions have not been previously investigated by means of pulsed wave tissue doppler imaging (PWTDI). Using PWTDI, we examined the effects of weight loss on cardiac functions and left ventricular (LV) mass in obese geriatric women.* ***Methods:*** *Thirteen obese women aged 66-83 years (mean age 71.2±4.9 yrs) with a body mass index 35.6-49 kg/m^2 (mean body mass index 39.9±4.3 kg/m^2) were evaluated by echocardiography and PWTDI. Only subjects with uncomplicated obesity were included. All measurements, including anthropometric variables, systolic and diastolic indices, and LV mass, were made before and after a 6-month Orlistat plus hypocaloric diet. Myocardial systolic wave (Sm) velocity, isovolumic acceleration (IVA), myocardial precontraction time (PCTm) and the PCTm to contraction time (CTm) ratio were calculated as systolic indices. Early diastolic wave (Em), late diastolic wave (Am), Em to Am ratio, myocardial relaxation time (RTm), deceleration time (DT) and isovolumic relaxation time (IVRT) were determined as diastolic measurements.* ***Results:*** *Subjects lost an average of 8.4±1.2 kg. LV mass decreased significantly after weight loss (p<0.001). In addition, IVRT decreased significantly (p=0.038). Only RTm decreased significantly (p=0.016), whereas other PWTDI parameters of LV remained the same. In the right ventricle, Sm velocity, IVA, Em, and Am velocities were similar. However, the PCTm to Am ratio decreased significantly (p=0.006), and the Em to Am ratio increased (p=0.04) and RTm decreased significantly (p=0.016) after weight loss.* ***Conclusions:*** *In obese geriatric women, weight loss improves ventricular diastolic functions and decreases LV mass. It also contributes to partial improvement in right ventricular systolic function.*
(Aging Clin Exp Res 2010; 22: 206-211)

INTRODUCTION

Obesity is one of the most common risk factors for cardiovascular disease, and is associated with increased cardiovascular mortality and morbidity (1, 2). In addition, in recent years, the prevalence of obesity has been markedly increasing worldwide, not only among young adults but also in both elderly men and women. The 2005 National Health Interview Survey found that 25.1% of men and 28.8% of women among older adults, respectively, were obese (3, 4).

Several reports (e.g., 5, 6) have shown that obesity increases total blood volume and cardiac output, because of the increased metabolic activity of excessive fat and abnormal myocardial relaxation and filling compared with non-obese people. In addition, increased left ventricular (LV) wall stress, LV mass, and abnormal loading conditions lead to impaired ventricular diastolic and systolic functions in obese subjects.

The changes in LV structure and LV mass with increasing obesity are partially explained by the hemodynamic changes that accompany obesity. Chronic volume overload and a rise in cardiac output lead to LV dilatation and compensatory (eccentric) LV hypertrophy. Metabolic and hormonal factors can also affect the cardiac morphology of obese people (7). Willens et al. (8) observed that severe obesity in adults is associated with LV systolic

*The study was presented orally at the 7th National Congress of Geriatrics in Izmir, Turkey, but not published in full or in part.

Key words: Elderly, left ventricular mass, obesity, orlistat, ventricular function.

Correspondence: Murat Varli, MD, Dodurga Türkkonut evleri Cömertler sitesi No: 4/7 Çayyolu2, Ankara 06170, Turkey.
E-mail: mvarli2003@yahoo.com

Received June 2, 2009; accepted in revised form August 4, 2009.

and diastolic dysfunction, and impaired right ventricular (RV) diastolic function.

Particularly in the geriatric population, the role of weight loss on LV mass and cardiac functions is likely to be considerable, but its influence on cardiac structure and ventricular functions in elderly subjects is still poorly understood.

Some studies (e.g., 8, 9) indicate that new echocardiographic methods, such as pulsed wave tissue doppler imaging (PWTDI) are more sensitive in detecting the presence of early sub-clinical changes in ventricular function than conventional echocardiographic methods.

The aim of this study was to evaluate the effects of weight loss with Orlistat plus a low-calorie diet on ventricular systolic and diastolic functions. LV mass was assessed by standard echocardiographic examination and tissue doppler imaging in elderly women with uncomplicated severe obesity for a period of six months.

METHODS

Study protocol

A 6-month, self-controlled prospective study was conducted at a geriatric outpatient clinic. This study complies with the Declaration of Helsinki. The study protocol was approved by the local ethics committee, and informed consent was obtained from all subjects.

Eighteen elderly people with a body mass index (BMI) >35 kg/m² between the ages of 66 and 83 were enrolled, but only 13 subjects completed the study, since five of them were lost to follow-up. Exclusion criteria were hypertension, heart failure, ischemic or valvular heart disease, atrial fibrillation, respiratory disease, diabetes mellitus, stroke, hepatic and/or renal dysfunction, systemic illness and malignancy. None of the subjects was taking medications that could affect weight gain or loss. The findings of medical history, physical examination, electrocardiogram, chest radiogram, and routine blood measurements such as total blood count, renal and hepatic functions, and serum electrolytes were normal in all subjects.

Physical examination, routine blood measurements, and anthropometric measurements such as weight, height, BMI, and waist and hip circumferences were performed at baseline and six months later. Weight and height were measured with subjects wearing light clothes and no shoes. Waist circumference was measured horizontally half-way between the iliac crest and the lower ribs, and hip circumference was measured at the greatest diameter over the buttocks. Both measurements were taken by the same person. Each measurement was performed in duplicate, and the average value was used to calculate waist and hip circumferences. BMI was calculated as the ratio of weight to height squared for each subject. The waist-to-hip ratio (WHR) was calculated as waist circumference (cm) divided by hip circumference (cm).

After baseline assessment and for the whole study period, all subjects were prescribed 120 mg t.i.d. Orlistat, a gastrointestinal lipase inhibitor, with breakfast, lunch and dinner, and a reduced-calorie diet regimen (1300-1500 kcal/day), containing 30% of calories from fat. Treatment compliance and adverse reactions with medication were evaluated in the first month and at every visit to the clinic. All subjects received dietary and appropriate physical activity counseling during the study period.

Echocardiographic examination

Standard echocardiography and pulsed wave tissue doppler imaging were performed with subjects in partial left decubitus on a Vingmed System 7 (Vivid 7 GE, Horten, Norway) before therapy and at the end of 6 months.

Standard echocardiography

Left ventricular internal dimensions, wall thickness and left atrial systolic dimensions were obtained from two dimensional guided M-mode echocardiographic tracings in the parasternal long axis view, according to the recommendations of the American Society of Echocardiography (10).

Fractional shortening (FS) was calculated as the percent change in LV internal dimension between systole and diastole. Mitral inflow velocities were obtained by pulsed wave doppler recording in the apical 4-chamber view, placing the sample volume at the tips of mitral valve leaflets. The peak early (E; meters per sec) and late (A; meters per sec) mitral inflow velocities and isovolumic relaxation time (IVRT; millisec; the time interval between the end of systolic output and the onset of E wave flow) and deceleration time (DT; millisec) were measured as indexes of LV global diastolic function. Pulsed doppler measurements of the LV systolic outflow tract was performed by placing the sample volume close to the aortic valve. The LV pre-ejection period (PEP; millisec; from electrocardiogram QRS to the beginning of systolic ejection), LV ejection time (LVET; millisec; from the beginning to the end of LV ejection) and the PEP/ LVET ratio were determined as systolic time intervals.

Tissue doppler imaging

Tissue doppler imaging was performed from the apical 4-chamber view; a 3-mm sample volume was placed at the level of the basal LV lateral mitral annulus and right ventricular (RV) lateral tricuspid annulus. Three waves were obtained in each cycle: one systolic wave (Sm), one early diastolic wave (Em) and one late diastolic wave (Am). Isovolumic acceleration (IVA) (precedes Sm and begins before the R wave on the electrocardiogram) was calculated by dividing myocardial peak velocity during isovolumic contraction by the time interval from the onset of this wave to the time at peak velocity. The peak velocity of Sm (cm per sec), IVA (me-

Table 1 - *Characteristics of study subjects before and after weight loss.*

Clinical characteristics	Before	After	Difference		p
	Mean±SD		mean	(%)	
Weight (kg)	88.9±10	80.5±8.8	-8.4	(9.4)	<0.001
BMI (kg/m²)	39.9±4.3	36.1±4.6	-3.8	(9.4)	<0.001
Waist circumference (cm)	113.6±8	107.8±8	-5.8	(5.1)	0.001
Hip circumference (cm)	120±8.7	109.4±8.9	-10.6	(8.8)	<0.001
Waist-to-hip ratio	0.94	0.98	+0.04	(4.2)	0.034

BMI: body mass index.

ters per sec squared), myocardial precontraction time (PCTm; millisec; from the onset of electrocardiogram QRS to the beginning of Sm), contraction time (CTm; millisec; from the beginning to the end of Sm) and their ratio (PCTm/CTm) were calculated as myocardial systolic indexes. Em (cm per sec), and Am (cm per sec) peak velocities, their ratio (Em/Am) and myocardial relaxation time (RTm; millisec; time interval between the end of Sm and the onset of Em) were calculated as myocardial diastolic indexes. All measurements were calculated from three consecutive cycles, and the average of three measurements was recorded.

Reproducibility

For inter-observer variability, a second observer calculated 10 measurements, and for intra-observer variability, the first observer measured 10 measurements on another day. Intra- and inter-observer variabilities were assessed as the difference between two readings in percent of the mean.

Left ventricular mass

The LV mass was calculated using the formula of Devereux and Reichek [1.04 × (diastolic LV diameter + septal thickness + LV wall thickness)^3 – (diastolic LV diameter)^3 – 13.6], with the Penn convention (11).

The LV mass index was calculated by dividing it by height$^{2.7}$ as previously reported (12), expressed in units of gram per meter$^{2.7}$ (g/m$^{2.7}$).

Statistical analysis

All continuous data were expressed as means±SD. The data were analysed by SPSS (version 13.0; SPSS Inc., Chicago, IL, USA). The data before and after weight loss were compared by the paired-sample *t*-test. $p<0.05$ was considered significant.

RESULTS

The study group consisted of 13 severely obese elderly women. This mean age and BMI at baseline were 71.2±4.9 yrs (range, 66-83 yrs) and 39.9±4.3 kg/m^2 (range, 35.6-49 kg/m^2), respectively. The characteristics of subjects before and after weight loss are listed in Table 1.

At the end of the 6-month follow-up, the combination of Orlistat and diet therapy in the elderly women with obesity resulted in a significant decrease in weight, from 88.9±10 to 80.5±8.8 kg ($p<0.001$), BMI from 39.9±4.3 to 36.1±4.6 kg/m² ($p<0.001$), waist circumference from 113.6±8 to 107.8±8 cm ($p=0.001$) and hip circumference from 120±8.7 to 109.4±8.9 cm ($p<0.001$). All subjects lost at least ≥5% of their initial body weight, with a mean decrease of 8.4±1.2 kg.

The effects of weight loss on two dimensional and doppler echocardiographic parameters of the left ventricle are listed in Table 2. No significant changes were not-

Table 2 - *Standard echocardiographic parameters of left ventricle before and after weight loss.*

	Before	After	p
	Mean±SD		
LVEDD (cm)	4.9±0.4	4.8±0.3	ns
LVESD (cm)	3.0±0.3	3.1±0.3	ns
IVST (cm)	1.05±0.09	1.0±0.07	0.016
LVPWT (cm)	1.02±0.06	0.99±0.07	ns
Left atrium (cm)	4.09±0.04	4.04±0.04	ns
FS (%)	37.3±5.6	35.7±3.9	ns
PEP (ms)	90.5±13.3	86.1±14.7	ns
LVET (ms)	291.7±21.2	297.2±23.9	ns
PEP/LVET ratio	0.31±0.05	0.29±0.06	ns
LVM (g)	186.7±24.7	172±29.9	<0.001
LVM index (g/m$^{2.7}$)	71.8±9.6	65.8±10.4	<0.001
E (m/s)	0.58±0.15	0.62±0.15	ns
A (m/s)	0.84±0.19	0.82±0.10	ns
E to A ratio	0.70±0.14	0.76±0.16	ns
DT (ms)	288.3±30.6	277.5±36.4	ns
IVRT (ms)	106.2±12.4	97±9.8	0.038

ns: non-significant. LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; IVST: interventricular septum thickness; LVPWT: left ventricular posterior wall thickness; FS: fractional shortening; PEP: pre-ejection period; LVET: left ventricular ejection time; LVM: left ventricular mass; E: mitral early peak velocity; A: mitral late peak velocity; DT: deceleration time; IVRT: isovolumic relaxation time.

Table 3 - *Tissue doppler parameters of left ventricle before and after weight loss.*

	Before	After	p
	Mean±SD		
Sm (cm/s)	8.4±1.9	8.5±1.6	ns
IVA (m/s^2)	4.3±1.5	4.9±1.4	ns
Em (cm/s)	7.8±2.5	7.4±1.8	ns
Am (cm/s)	12.8±2.9	12.0±2.9	ns
Em to Am ratio	0.64±0.25	0.64±0.19	ns
PCTm (ms)	96.6±14.7	98.1±14.0	ns
CTm (ms)	274.4±27.3	288.3±25.8	ns
PCTm to CTm ratio	0.35±0.08	0.34±0.06	ns
RTm (ms)	92.2±16.6	76.4±15.2	0.016

ns: non-significant. Sm: systolic wave; IVA: isovolumic acceleration; Em: early diastolic wave; Am: late diastolic wave; PCTm: myocardial precontraction time; CTm: contraction time; RTm: myocardial relaxation time.

ed in the LV end-systolic diameter (LVESD), LV end-diastolic diameter (LVEDD), left atrial dimension, LV posterior wall thickness (LVPWT) or FS. However, interventricular septum thickness (IVST) decreased significantly after weight loss (p=0.016). The LV mass and adjusted LV mass index decreased significantly after weight loss (p<0.001). Mitral inflow parameters and PEP, LVET and the PEP/LVET ratio were not different compared with baseline values. The only parameter that markedly changed after weight loss was IVRT, which fell significantly (p=0.038).

The effects of weight loss on the tissue doppler imaging parameters of LV function are listed in Table 3. In the left ventricle, none of the parameters changed, except the RTm interval, before and after weight loss. RTm decreased from 92.2±16.6 to 76.4±15.2 (p=0.016). Table 4 lists the effects of weight loss on tissue doppler imaging parameters of RV function. In the right ventricle, Sm velocity, IVA, and Em and Am velocities were similar before and after weight loss. Among systolic measurements, although PCTm decreased (p=0.077) and CTm increased (p=0.065), but not significantly, the PCTm to CTm ratio decreased significantly (p=0.006). Among diastolic measurements, Em to Am ratio increased (p=0.04) and RTm decreased significantly (p=0.016) after weight loss in severely obese subjects. Intra and inter-observer variabilities in tissue doppler imaging measurements were 3.2% and 4.1% respectively.

Table 4 - *Tissue doppler parameters of right ventricle before and after weight loss.*

	Before	After	p
	Mean±SD		
Sm (cm/s)	13.8±2.7	14.5±3.6	ns
IVA (m/s^2)	4.8±1.0	4.7±1.6	ns
Em (cm/s)	10.7±1.9	12.4±3.6	ns
Am (cm/s)	16.0±2.7	15.6±3.7	ns
Em to Am ratio	0.67±0.11	0.79±0.14	0.04
PCTm (ms)	90±11	80±15	ns
CTm (ms)	269.4±26.8	292.2±32.1	ns
PCTm to CTm ratio	0.33±0.05	0.27±0.06	0.006
RTm (ms)	88.8±20.4	71.6±20.9	0.016

ns: non-significant. Sm: systolic wave; IVA: isovolumic acceleration; Em: early diastolic wave; Am: late diastolic wave; PCTm: myocardial precontraction time; CTm: contraction time; RTm: myocardial relaxation time.

DISCUSSION

Weight loss after Orlistat and a reduced-calorie diet therapy in elderly women with severe obesity is associated with improvement in both RV and LV diastolic functions and sub-clinical improvement in RV systolic function, as well as a decrease in LV mass and adjusted LV mass index compared with baseline values at the end of the 6-month follow-up. Previous studies (8, 13, 14) have shown that obesity affects both systolic and diastolic functions and induces LV mass increase. It is a condition that may cause left and right ventricular dysfunction, even in subjects without co-morbid cardiovascular risk factors.

The Framingham Heart Study described obesity as an independent predictor of left ventricular hypertrophy and the strongest determinant of LV mass independently. Obesity and increased LV mass are also related to higher mortality and morbidity (15, 16). Although several studies (4-13) have investigated the influences of obesity on ventricular function and LV mass, studies evaluating the effects of weight loss on myocardial functions have been limited.

Maniscalco et al. (17) recently evaluated the effects of surgically-induced weight loss on right ventricular function through the right myocardial performance index (R-MPI) in uncomplicated severely obese adults. They observed that decreased R-MPI suggests an improvement in right ventricular dysfunction. Willens et al. (18) showed an increase in tricuspid annular systolic and early diastolic velocities and mitral annular early diastolic velocity after substantial weight reduction through gastric bypass surgery in severely obese subjects; their results indicated an improvement in RV systolic and diastolic function and LV diastolic function. However, in that study, most of the subjects presented co-morbidities such as hypertension, diabetes mellitus and obstructive sleep apnea syndrome.

In our study, we observed an increase in the Em to Am ratio but a decrease in the RTm and PCTm to CTm ratios after weight loss among the parameters of RV systolic and diastolic functions evaluated by PWTDI. The Em to Am ratio and RTm changes were consistent with improved RV diastolic function, whereas a decrease in the PCTm to RTm ratio was consistent with improved RV systolic

functions. In the left ventricle, there were no significant differences in the parameters of LV systolic and diastolic functions determined by PWTDI. However, only the RTm significantly decreased. This finding suggests improved LV diastolic function after weight loss. These results are similar to those of the studies mentioned above. However, unlike the study by Willens et al. (18), ours included uncomplicated, conventionally treated elderly women with severe obesity, and the weight loss achieved was lower (mean: 8.4 kg).

It is well documented that obesity is associated with an increase in total blood volume, cardiac output, LV mass and LV hypertrophy, which may lead to ventricular dysfunction. Increased left ventricular filling pressures and LV wall stress in obese subjects lead to ventricular hypertrophy and higher LV mass. Left ventricular loading conditions as well as LV mass affect LV functions. Right ventricular structural and functional changes may be affected by similar hemodynamic and morphologic alterations (5, 19, 20). Some authors (7, 18, 21) have observed that weight loss induced medically or surgically leads to reduction of LV mass and improves ventricular functions. It is possible that decreased LV mass after weight loss contributes to improved ventricular functions, leading to a decrease in LV filling pressure and wall stress.

In the present study, LV mass and the adjusted LV mass index ($g/cm^{2.7}$) were significantly lower compared with values before weight loss in the severely obese elderly women. Our data match those of previous studies (e.g., 7, 22).

As LV mass is a strong predictor of sudden cardiac death and is also associated with increased mortality, reduction of LV mass after losing weight may reduce mortality in obese people. However, in our study, we did not find any changes in the left atrial, left ventricular or right ventricular dimensions, except for a decrease in IVST at the end of the study. Karason et al. (7) also showed that increased chamber size in obese subjects did not regress, even though LV mass and wall thickness were lower after weight loss. They suggested that chamber dilatation consistent with obesity may be less reversible than LV mass and thickening. Decreased LV mass after weight loss may be associated with reduced diastole filling pressure and improved myocardial relaxation, although no changes occur in atrial and ventricular cavity sizes.

According to the results of our study, weight reduction first leads to decreased LV mass and then improves LV and RV diastolic functions in obese elderly women. However, it did not improve their systolic function, but led to a decrease in the PCTm/CTm ratio of RV. This change appears to be a mild improvement in RV systolic function, which cannot be detected by standard echocardiographic methods.

The results of our study reflect the short-term effects of weight loss. Significant improvements in ventricular functions and LV mass were accompanied by modest weight loss. Although subjects with obesity still remained obese after their weight loss, positive effects on cardiac functions appeared, but favorable alterations in systolic functions may require more time. Therefore, further prospective studies with larger numbers of subjects are needed to clarify the long-term effects of weight loss on ventricular systolic functions. The small number of subjects, absence of hemodynamic measurements, and relatively short follow-up were the limitations of our study. In addition, velocities assessed by PWTDI are limited by rotational and restraining forces in the contracting and relaxing heart. Strain rate imaging, which measures segmental tissue deformation and studies performed with larger numbers of subjects and longer follow-up periods may be useful in resolving these limitations.

Previous studies involved obese individuals younger than 65 years of age. To our knowledge, this is the first prospective study evaluating the influences of weight reduction on ventricular functions and LV mass by means of PWTDI in obese elderly women. Although obese individuals have sub-clinical ventricular dysfunction determined by echocardiography, long-standing obesity leads to heart failure, called obesity cardiomyopathy, which usually develops in people with severe, sustained obesity. Therefore, prevention and treatment of obesity are very important (5).

CONCLUSIONS

Despite its limitations, our study indicates that significant weight loss contributes to improved LV and RV diastolic functions, regression of LV mass, and mildly improved (sub-clinical) RV systolic function in severely obese elderly women. Such women, particularly those with severe obesity, should be encouraged to lose weight. However, the potential risks and benefits of weight loss should be evaluated individually.

REFERENCES

1. Pascual M, Pascual DA, Soria F et al. Effects of isolated obesity on systolic and diastolic left ventricular function. Heart 2003; 89: 1152-6.
2. Adams KF, Schatzkin A, Harris TB et al. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. N Engl J Med 2006; 355: 763-78.
3. Kruger J, Ham SA, Prohaska TR. Behavioral risk factors associated with overweight and obesity among older adults: the 2005 National Health Interview Survey. Prev Chronic Dis 2009; 6: 1-17.
4. Peterson LR, Waggoner AD, Schechtman KB et al. Alterations in left ventricular structure and function in young healthy obese women. J Am Coll Cardiol 2004; 43: 1399-404.
5. Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. Am J Med Sci 2001; 321: 225-36.
6. Powell BD, Redfield MM, Bybee KA, Freeman WK, Rihal CS. Association of obesity with left ventricular remodeling and diastolic dysfunction in patients without coronary artery disease. Am J Cardiol 2006; 98: 116-20.

7. Karason K, Wallentin I, Larsson B, Sjöström L. Effects of obesity and weight loss on left ventricular mass and relative wall thickness: survey and intervention study. BMJ 1997; 315: 912-6.
8. Willens HJ, Chakko SC, Lowery MH et al. Tissue Doppler imaging of the right and left ventricle in severe obesity (Body Mass Index > 35 kg/m²). Am J Cardiol 2004; 94: 1087-90.
9. Wong CY, O'Moore-Sullivan T, Leano R, Byrne N, Beller E, Marwick TH. Alterations of left ventricular myocardial characteristics associated with obesity. Circulation 2004; 10: 3081-7.
10. Schiller NB, Shah PM, Crawford M et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 1989; 2: 358-67.
11. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. Circulation 1977; 55: 613-8.
12. De Simone G, Devereux RB, Daniels SR, Koren MJ, Meyer RA, Laragh JH. Effect of growth on variability of left ventricular mass: assessment of allometric signals in adults and children and their capacity to predict cardiovascular risk. J Am Coll Cardiol 1995; 25: 1056-62.
13. Crisostomo LL, Araujo LMB, Camara E et al. Comparison of left ventricular mass and function in obese versus nonobese women < 40 years of age. Am J Cardiol 1999; 84: 1127-9.
14. Otto ME, Belohlavek M, Khandheria B, Gilman G, Svatikova A, Somers V. Comparison of right and left ventricular function in obese and nonobese men. Am J Cardiol 2004; 93: 1569-72.
15. Levy D, Anderson KM, Savage DD, Kannel WB, Christiansen JC, Castelli WP. Echocardiographically detected left ventricular hypertrophy: prevalence and risk factors. The Framingham Heart Study. Ann Intern Med 1988; 108: 7-13.
16. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990; 322: 1561-6.
17. Maniscalco M, Arciello A, Zedda A et al. Right ventricular performance in severe obesity. Effect of weight loss. Eur J Clin Invest 2007; 37: 270-5.
18. Willens HJ, Chakko SC, Byers P et al. Effects of weight loss after gastric bypass on right and left ventricular function assessed by tissue Doppler imaging. Am J Cardiol 2005; 95: 1521-4.
19. Crisostomo LL, Araujo LMB, Camara E et al. Left ventricular mass and function in young obese women. Int J Obes 2001; 25: 233-8.
20. Dorbala S, Crugnale S, Yang D, Di Carli MF. Effect of body mass index on left ventricular cavity size and ejection fraction. Am J Cardiol 2006; 97: 725-9.
21. De Simone G, Romano C, Caprio CD et al. Effects of sibutramine-induced weight loss on cardiovascular system in obese subjects. Nutr Metab Cardiovasc Dis 2005; 15: 24-30.
22. Alpert MA, Terry BE, Mulekar M et al. Cardiac morphology and left ventricular function in normotensive morbidly obese patients with and without congestive heart failure, and effect of weight loss. Am J Cardiol 1997; 80: 736-40.