ORIGINAL ARTCLE

Risk and Early Changes of Left Ventricular Structure and Function in Young Obesity

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ABSTRACT

Aim: The increased adipose tissue in obesity had an extensive capillary vessels, secretes mediators that could influence left ventricular (LV) functions directly. Previous study mostly showed normal ejection fraction in obesity. Peak systolic velocity is a non-load-dependent echocardiography technique that strongly related with contractility compared to strain methods.

Methods: This was a cross sectional analytic study, taken at Dr. Kariadi Hospital, Semarang, Indonesia. Subjects had Body Mass Index (BMI) ≥18,5 kg/m^2, 15-40 years old age, without any co-morbid. Subjects categorized as an obese (BMI ≥27 kg/m^2) and non-obese (BMI <27).

Results: The study enrolled 110 subjects, 63 (57%) of them were obese. In LV structure, obese subjects had a higher RWT and LVMI (0,34 vs 0,45; p=0,00 and 73,38 vs 99,02 g/m^2; p=0,00). In LV systolic function, there was no difference in LVEF, but obese subjects had a significant lower Sm (68,87 vs 70,81%; p=0,120 and 4,72 vs 3,62 cm/s; p=0,00). In LV diastolic function, obese subjects had a lower Em (8,84 vs 7,05 cm/s; p=0,00), a higher E/e' ratio and LAVI (7,59 vs 10,03 ms; p=0,00 and 15,39 vs 19,00 ml/m^2; p=0,00). Obese subjects had 1,36 times higher to develop abnormal structure; 1,22 times higher to develop early systolic dysfunction..

Conclusion: In youngisolatedobesity, althoughwith normal ejectionfraction, we found higher risk and early changes of left ventricular structure, andearlydysfunctionboth onsystolicanddiastolicfunction.

Keywords: Obesity, LV structure, tissue velocity echocardiography

INTRODUCTION

The number of individuals with obesity has been increasing until today. Changes of dietary habit, physical inactivity and urbanization are suspected as the causes of the increasing number of individuals with obesity in developing countries up to 3–5 times higher compared to that of developed countries[1]. Obesity is a condition in which the ratio of body fat tissues to total body weight is higher than it of normal condition, or a condition where a person has accumulated so much body fats which makes the body weight is well above its normal value[2]. The increasing body fats or adipocytes can affect both preload and after load due to hyper-dynamic circulation, chronic excess body fluids and the increasing of peripheral resistance[3]. Apparently, adipose tissues are not only as energy reserves, but also as endocrine organ which can secrete hormones and mediators which affect the heart directly. So that, it can be said that individual with obesity has a broad spectrum of cardiovascular disorder, starting from hyper-dynamic circulation to sub-clinical heart structural change[4,5]. Some of studies which tried to observe the relationship between the increasing Body Mass Index (BMI) with ventricular functions showed inconsistent findings. The modality of non-invasive imaging of echocardiography with conventional techniques is often generating less optimal results for evaluating heart structure in detail, and detecting heart functional changes of obese people. The most recent techniques of echocardiography such as Tissue DopplerImaging (TDI), strain, strainrate (SR) and 2D-speckle tracking can image systolic and diastolic dysfunctions or disorders which may begin earlier on healthy individuals with obesity[6]. This new method also minimize the pitfalls of systolic functions calculation due to increasing preload which usually happened on obesity people. This study aimed to find any initial structural change or change of ventricular function in healthy individuals with obesity.

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MATERIAL AND METHODS

Subjects of this study were young adults, age 15-40 years old, with BMI ≥ 18,5 Kg/m^2 undergoing general check-up in Dr Kariadi Hospital, without any histories of coronary heart disease, valvular heart disease, cardiomyopathy, congenital heart disease, hypertension, diabetes mellitus, arrhythmias, pericardial disease, and anemia. The obese and non-obese criteria were characterized based on their BMI, using the Indonesia's criteria where subject with BMI≥27 kg/m^{2}was defined as obese. Each subject performed an echocardiography examination, including 2-dimensional echocardiography, tissue doppler imaging/TDI and tissue velocity imaging/TVI. The left ventricular structure measured with left ventricular mass index (LVMI), relative wall thickness (RWT). The left ventricular diastolic function were calculated from the E/A ratio, E/e' ratio, deceleration time, and average of peak velocity of early rapid filling with TVI (Em). The left ventricular systolic function was measured with left ventricular ejection fraction (LVEF), and average of peak velocity of systolic using TVI (Sm). The average of velocity by TVI were obtained from the basal velocity, the mid velocity and the apical velocity of LV. The echocardiography device used was Vivid S5, brand of GE Health Care, Norway using probe of 3S-RS adult with frequencies of 1.5–3.5MHz. The echocardiography results are observed and measured by two observers. The reliability of the two observers were measured using inter-item correlation test. The relationship between obesity, left ventricular systolic and diastolic functions and left ventricular structures were analyzed

using multivariate logistic regression test with consideration to variables of confounders of ages, gender and BP.

Ethics: All samples of this study are willing to join the study and signed the Informed Consent form. This study had obtained ethical clearance of Ethics Committee of Faculty of Medicine of Diponegoro University/ DR Kariadi Hospital.

RESULTS

There were 112 of young adults enrolled in this study, but 2 of them were excluded due to mitral regurgitation finding. The 110 subjects who met the inclusion and exclusion criteria were analyzed. The reliability between the two observers was 0,55-0,88 meaning a good agreement between the two observers. Table 1 shows the data of sample's characteristics including the normality test results.

Descriptive analysis of left ventricles structural changes prevalence is as follows: apparently 63 of the sample were obese people, 21 of the obese sample (33.33%) had normal structures while 42 others (66.66%) had abnormal structures. Characteristics of left ventricular structural remodeling on patients with obesity are shown in Figure 1, were mostly of the obese subjects (30,20%) develop a concentric remodeling of LV. In LV structure, obese subjects had a higher RWT and LVMI (0,34 vs 0,45; p=0,00 and 73,38 vs 99,02 g/m^2; p=0,00). The Young-Adults with obesity population has a tendency of 1.36 times higher to experience the change of left Results of multivariate analysis between obesity and left ventricular structures show the significant relationship between Body Mass Index (BMI) and the occurrence of changes of left ventricular structures and its systolic and diastolic functions (Table 2). In LV systolic function, there was no difference in LVEF, but obese subjects had a significant lower Sm (68,87 vs 70,81%; p=0,12 and 4,72 vs 3,62 cm/s; p=0,00) especially in basal inferoseptal and anterolateral segments. There was no correlation between BMI and LVEF (Figure 2), but when the average of systolic velocity of LV (Sm) was measured, there was a significantly negative relationship between BMI and Sm (Fig. 3). The young obese subjects had 1.26 times higher to experience LV systolic functional disorder. What is also interesting is that on all obese subjects shows normal LVEF.

In LV diastolic function, obese subjects had a lower E/A ratio and E' (1,75 vs 1,56; p=0,01 and 8,84 vs 7,05 cm/s; p=0,00), a higher E/e' ratio and LAVI (7,59 vs 10,03 ms; p=0,00 and 15,39 vs 19,00 ml/m^2; p=0,00), a longer decT and IVRT (180,68 vs 198,13; p=0,00 and 63,81 vs 76,80 ms; p=0,00). Discriminant multivariate analysis showed that among those parameters: RWT, E/e' ratio, IVRT and E' was the echocardiographic parameters that significantly changed in young isolated obesity. The obese subjects had 1.33 times higher to experience LV diastolic functional disorder than others without obesity.

Table 1: Subject characteristics and normality data

Variable (scale)	Mean	SD	Median	p	Normality test
BMI (Kg/m^2)	28,30	5,45	27,68	0,20	KS
Age (year)	30,83	5,74	31,00	0,20	KS
SBP (mmHg)	114,32	9,80	120	0,00*	KS
DBP (mmHg)	74,82	7,20	80	0,00*	KS
RWT	0,40	0,09	0,38	0,12	SW
LVMI (g/m$^{2)}$	88,06	26,09	87,56	0,20	KS
LVEF (%)	69,98	6,45	70,00	0,20	KS
E/A ratio	1,67	0,38	1,58	0,00*	KS
E/e'ratio	8,99	2,02	8,51	0,01*	KS
decT (second)	190,67	32,01	189,00	0,31	SW
IVRT (second)	71,14	12,85	70,00	0,01*	SW
LAVI	17,46	5,25	16,69	0,09	KS
Sm (cm/sec)	2,78	0,96	2,67	0,03*	KS
Em (cm/sec)	6,15	1,76	5,95	0,012*	KS

*p>0,05 show normal distribution

BMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure, RWT=relative wall thickness; LVMI= left ventricular mass index; LVEF=left ventricular ejection fraction; E=early rapid filling; A=atrial kick; e'=early rapid filling by tissue Doppler; decT=deceleration time; IVRT=isovolumetric relaxation time; LAVI=left atrial volume index; Sm=average of systolic velocity by tissue velocity imaging; Em=average of early rapid filling velocity by tissue velocity imaging

Table 2. Multivariate analysis of Obesity with LV structure and functions

Dependent variable	Non-dependent variable	Exp(B)	Sig
LV structure	**BMI**	**1,36**	**0,00***
	Sex	0,97	0,97
	Age	1,06	0,27
	SBP	1,02	0,79
	DBP	0,10	0,97
LV systolic function	**BMI**	**1,26**	**0,00***
	Sex	0,20	0,01*
	Age	0,99	0,87
	SBP	0,95	0,35
	DBP	1,20	0,02*
LV diastolic function	**BMI**	**1,33**	**0,00***
	Sex	0,59	0,41
	Age	1,09	0,07
	SBP	0,99	0,91
	DBP	1,02	0,77

Fig. 1: LV structure of Obese subjects

RWT	≤95 (♀) ≤115 (♂)	>95 (♀) >115 (♂)
>0,42	Remodeling Konsentrik 19 (30,2%)	Hipertrofi Konsentrik 17 (25,4%)
≤0,42	Normal 20 (31,7%)	Hipertrofi Eksentrik 7 (12,7%)

Left ventricle mass Index (gm/m²)

Fig. 2. Correlation between BMI and LVEF (r=0,06;p=0,49)

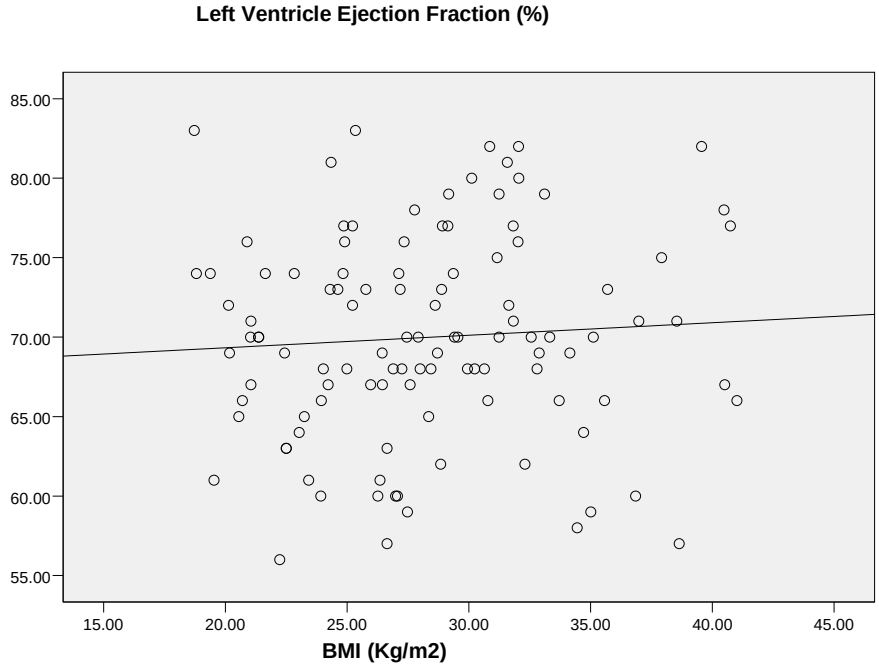
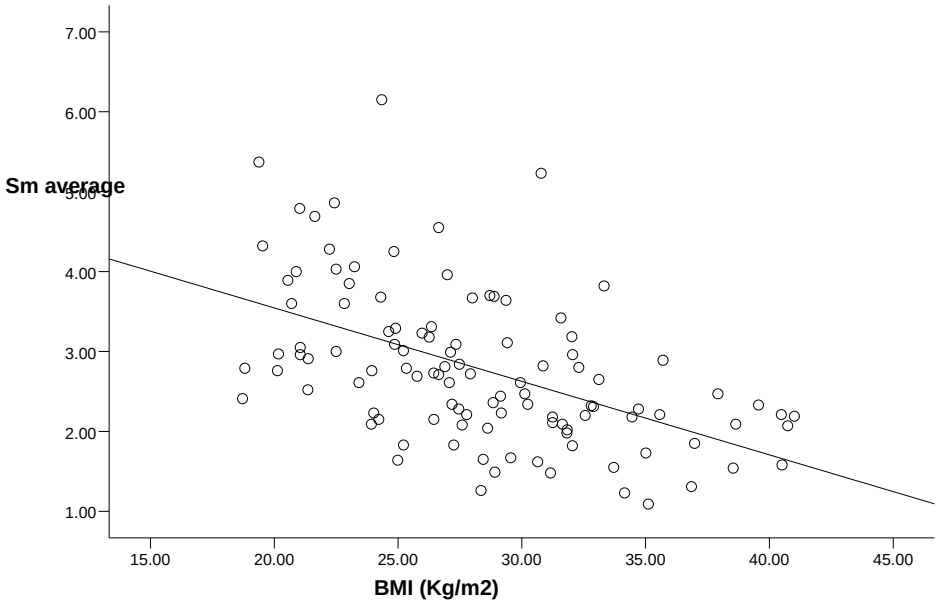


Fig. 3: Correlation between BMI and average of systolic velocity by TVI (r=-0,56;p=0,00)



DISCUSSION

Obesity is a condition where a person has accumulated so much body fats which makes the body weight is well above the normal value. Deposits of fats are apparently not only as passive energy reserves but also having properties which can infiltrate muscular mass, contain more capillary tissues and play a role as endocrine organ which can secrete hormones and mediators, so that the fat accumulation can be suspected as the direct cause of the changes of heart function and structure.[7,8,9] The effect of the duration of obesity, fats infiltration to the myocardial tissue as well as the effect of mediators and hormonal factors like insulin, IGF-1,TNFα, IL-6, PAI1, CRP, Leptin, resistin which are increasing, as well as decreasing adiponectin with body fat addition which may spur the occurrence of mass addition or hypertrophy were suspected to have role in the mechanism of the occurrence of the change of left ventricular structure in obesity[10].

Structural changes in young adult population with obese need to get more attention since some studies showed the relationship between concentric left ventricular remodeling with the increases of morbidity and mortality. Rosen et al investigated the effect of concentric left ventricular remodeling in asymptomatic individuals and found the decrease of both global and regional systolic function of left ventricle, though the results had been adjusted to the blood pressure.[11]Laviereviewed the data of epidemiologyand stated that abnormal geometry including concentric left ventricular remodeling and hypertrophy was not only related with the increasing cardiovascular risk but also having role as a target of therapeutic intervention[12].

The determination of the decrease of systolic function in this study was, mainly, from the decrease of average Systolic velocity by TVI, since all of the sample have normal LVEF. It probably due to the LVEF measuring method was very affected by the volume load while individuals with obesity experiencing the increasing volumes, so that it can be said that the LVEF measuring method is relatively less sensitive to detect the disorder of myocardial contraction speed function occurring at the initial change of systolic dysfunction[4,13,14,15]. This volume effect is expected to be minimized using the methods of tissue Doppler and tissue velocity tracking which are less affected by volume status. So that, it can be said that the tissue velocity tracking method is quite sensitive to detect both systolic and diastolic initial dysfunctions, especially in people with obesity.

CONCLUSION

Obesity is related to the structural, diastolic function and systolic function changes of left ventricles in young adults. Structural changes which are dominant in obese people are concentric remodeling type. No LVEF decrease was found, but by using the method of tissue velocity tracking, there was a significant decrease of initial systolic function found in the obese subjects.

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