Effect of Obesity on Left Ventricular Mass: Results from 320 Multi-slices Computed Tomography

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Objective: *To determine effects of obesity and gender on left ventricular mass in normotensive and hypertensive Thai patients using 320-slice cardiac computed tomography (CT)*

Material and Method: *Left ventricular mass (LVM) obtained from 320-slice coronary CT angiogram was compared in 597 normotensive subjects (175 men [65 obese] and 422 women [133 obese], aged 55±7 years) and 483 hypertensive patients (180 men [104 obese] and 303 women [170 obese], aged 60±7 years). Obesity in Asian population was defined by body mass index (BMI) ≥ 25 kg/m^2 in both genders. LV mass was normalized for body surface area (BSA)and height$^{2.7}$.*

Results: *The upper normal limit of LVM/ height$^{2.7}$ developed from 244 (197 women, 47 men) low risk subjects (non-smoking normal-weight adults free from hypertension, diabetes, coronary artery disease & dyslipidemia) was lower than the established criteria for left ventricular hypertrophy (LVH) (31 versus 44 g/m$^{2.7}$ in women; 36 versus 48 g/m$^{2.7}$ in men). There is statistical difference between men and women in all groups of analysis. Among both hypertensive and normotensive subjects, the prevalence of LVH and LVM/height$^{2.7}$ are higher in the obese group than normal-weight group in both genders (LVM/ height$^{2.7}$ p<0.001; prevalence of LVH – obese versus normal-weight hypertension: 58% versus 34% in women, 43% versus 14% in men; obese versus normal-weight normotension: 35% versus 16% in women, 40% versus 15% in men). The same differences between obese and normal-weight groups were also present when normalizing LVM for height but not with LVM/BSA. Logistic regression analysis revealed that systolic blood pressure and BMI were the main predictors of LVH in the entire population (p<0.001 in both genders). Equations for predicting LVH in men and women were: Risk of LVH = 1/(1+e^{-w}) where w is as follows: w (men) = 0.02* systolic pressure + 0.25*BMI – 9.86, w (women) = 0.03* systolic pressure + 0.17*BMI – 8.82*

Conclusion: *Obesity is an independent stimulus to increase LVM in normo-tensive subjects, and its effect is additive in hypertensive patients. Gender and obesity affect LVM and prevalence of LVH.*

Keywords: *Left ventricular mass, Computed tomographic angiography, Obesity, Hypertension*

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Obesity and hypertension are associated with increased left ventricular (LV) mass. There is evidence that the left ventricular hypertrophy (LVH) is an extremely strong risk factor for cardiovascular morbidity and mortality[1,2].

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Many studies revealed that obesity is associated with LVH, a potential effect to heart failure[3-8]. Majority of publications demonstrated a positive, independent relationship between LV mass and body mass index (BMI). Several studies also reveal additive effects of increasing blood pressure and BMI on LV mass[3,5,6,8]. Therefore, the combination of arterial hypertension and obesity is more steadily associated with LVH than either stimulus alone.

Effects of obesity and hypertension on cardiac structure and function have been extensively studied.

However, there are no previous data, to our knowledge, about impact measurements from 320-slice cardiac CT to show this evidence[9-13]. In addition, there are limited publication data for LV mass and function in Asian population available that may be insufficient for evaluation and management for cardiovascular risk in this population group[14].

To date, cardiac function and myocardial mass assessment have been performed with various noninvasive modalities, such as echocardiography, nuclear medicine, multidetector row helical CT, and MRI. Volume mediated CT scan using 320-slice has been recently released for general use. The unique 16 cm z-axis coverage of the 320-slice CT, allows scanning of the heart at a single moment in time resulting in temporal homogeneous contrast distribution and better contour of intraventricular cavities. The left ventricular parameters measured by cardiac CT are highly reproducible, easy to access, shorter time for study, well-delineated cardiac anatomy and operator-independent difference from echocardiogram. CT scanner is also more available and lower examination cost than MRI which is widely used and gold standard for evaluation of the heart. CT and MRI for measurement of LV mass and function have shown excellent correlation and substitutability with each other[15,16].

The purpose of the present study was to evaluate correlation of obesity, arterial hypertension or their combination to the left ventricular mass and function in separation of gender using 320-slice cardiac MDCT angiography.

Material and Method

Study population

The study population consisted of asymptomatic normotensive and hypertensive patients with intermediate risk (NYHA I) factors for coronary artery disease (CAD) referred to coronary CT angiogram using 320-slice volumetric cardiac CT (Aquilion ONE, Toshiba, Japan) from year 2009 to 2010 at Ramathibodi Hospital, Mahidol University. Hypertensive patients had blood pressure measured by arm cuff and mercury sphygmomanometer above 140/90 mmHg or had been being treated with antihypertensive drug; whereas normotensive subjects had blood pressure consistently below this level. Coronary calcium score was also obtained.

Obesity is defined according to The Asia-Pacific Perspective: Redefining Obesity and its Treatment[17] criteria as a BMI ≥ 25 kg/m^2 in both men and women.

These study population were retrospectively reviewed for left ventricular function and mass.

Data for body weight (kg), height (cm), waist circumference (inch), blood pressure (mmHg), history of current treatment with lipid lowering drug, smoking status, alcoholic consumption, family history of CAD risks (CAD, hypertension and diabetes mellitus) were obtained.

Body surface area (BSA) was calculated with DuBois&DuBois formula[18] (BSA (m^2) = 0.20247 x height (m)$^{0.725}$xweight (kg)$^{0.425}$). Body Mass Index (BMI) was calculated by bodyweight divided by height in square meters.

CTA method

All patients who have initial heart rate more than 70 beats per minute (bpm) were orally administered 50-100 mg metoprolol 1 hour before data acquisition to reduce cardiac motion artifact, unless contraindicated.

MDCT studies were performed using a 320-slice MDCT scanner (Aquilion ONE, Toshiba, Japan) with rotation time of 350 milliseconds and 0.5-mm collimation. The tube voltage and tube current were adapted to patient size (120-135 kVp, 380-500 mA).
Patients were examined with retrospective ECG-triggered, ECG modulated tube current dose reduction methodology scanning a range between 30% and 80% of the cardiac cycle.

The total amount of nonionic contrast media (Ultravist®, 370 mg%) injected into antecubital vein through an eighteen gauge intravenous catheter was 70-90 mL (depending on body weight). The contrast media was administered at a flow rate of 5 mL/sec followed by 20 mL of a saline flush at the same flow rate. Automated bolus tracking was used in order to synchronize the arrival of the contrast media and the scan. After contrast enhancement of the descending aorta was reached to 170 Hounsfield units (HU), the MDCT examination was automatically initiated. After a four second delay, images were obtained during an inspiratory breath hold of approximately 5-10 seconds.

LV function and LV mass analysis

To assess LV function and LV mass, 10 phases of the cardiac cycle were reconstructed at 10% interval from 0% R-R interval to 90% R-R interval and sent to a remote workstation (Vitrea FX version 1.1, Vital Images, Minnesota, USA) for LV function analysis and LV mass calculation. Optimal phase, being the best phase, usually locating at 75% level was reconstructed to give a better picture. Interpretation for coronary stenosis included analysis of axial source images, 3-mm maximum intensity projection (MIP) in axial, right anterior oblique (RAO), left anterior oblique (LAO), post-processed three dimensional reconstruction and multiplanar and curve reformats for each coronary arteries.

Analysis and calculation of LV function and mass were done by senior technologists with more than 5 years experience in cardiac CT. Endocardial and epicardial borders were semiautomatically contoured from the base to the apex on the short axis images and then corrected manually at the base of the heart if images obviously revealed incorrect borders. The papillary muscles were excluded from the LV myocardial mass (Fig. 1).

The four layout screen was an automatic setup allowing the operator to set apical and basal myocardial limits. Correction along axis angulations in three directions – vertical two chambers, four chambers and short axis views were also done in the same manner (Fig. 1).

The systolic and diastolic volumes were calculated automatically from CT volume contouring inside the left ventricular endocardial lining. LV stroke volume was calculated as the difference between LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV). LV ejection fraction (LVEF) was calculated as LV stroke volume divided by LVEDV and then multiplied by 100. Cardiac output was calculated as LV stroke volume time heart rate.

The LV mass was determined from end-diastolic phase as the volumetric contouring of the myocardial area multiplied by specific gravity of the myocardium (1.05 g/mL) (Fig. 2).

Normalization of left ventricular parameters for body size

Heart size differs in subjects of different body size. In a healthy reference group, left ventricular mass in grams was shown to be associated with height in meters.[11] LV mass and volume strongly relate to body size, indicating the need for appropriate adjustment of the left ventricular parameters to the body size[10,19]. Either BSA or height could be used to normalize for these differences. Normalization by height raised to the allometric power of 2.7 was also performed to minimize gender differences in LV mass and to improve the prediction of adverse cardiovascular events in a population with a high prevalence of obesity.[11,19]

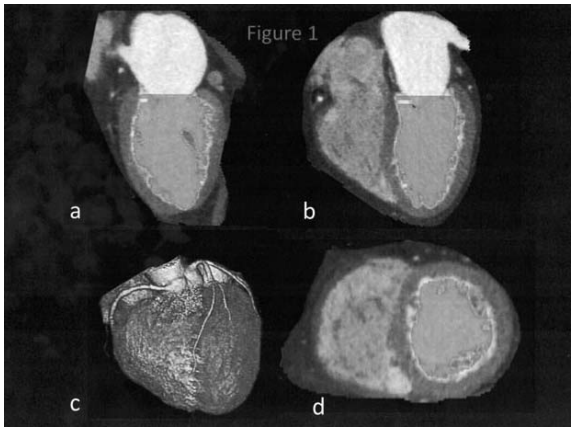

Fig. 1 Demonstrates end-diastolic phase of the vertical long axis, horizontal four chamber and short axis (a, b and d), the red color area inner to the endocardial border represents left ventricular volume. A 3-D image of the heart is demonstrated (c).

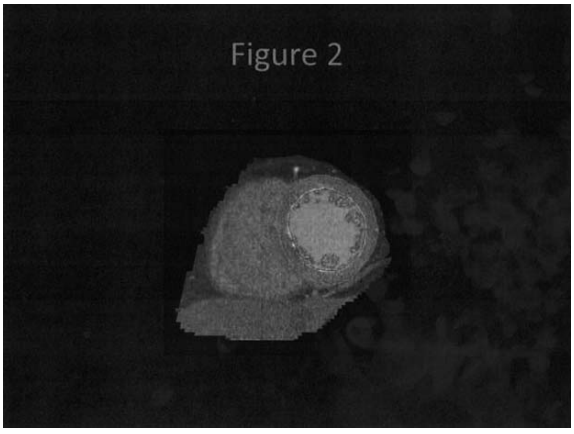

Fig. 2 Demonstrates automatic contour detection along the epicardial (outer line) and endocardial (inner line) borders.The area between these linesrepresents volume of left ventricular mass. The red color area represents left ventricular volume excluding papillary muscle.

Table 1. Patient Characteristics

Characteristics	Normal-weight	Obese	p^a
Normotension, n	399	198	-
Gender female, n (%)	290 (72.7)	133 (67.2)	-
Diabetes mellitus, n (%)	13 (3.3)	22 (11.1)	-
Dyslipidemia, n (%)	122 (30.6)	66 (33.3)	-
Current cigarette smoking, n (%)	44 (11.0)	32 (16.2)	-
Age, y	55.7±7.4	54.7±7.6	0.149
Weight, kg	56.1±7.5	70.9±9.6	<0.0001
Height, cm	159.2±7.4	159.9±7.9	0.317
Waist, inch	33.7±3.4	38.9±3.7	<0.0001
Body mass index, kg/m^2†	22.1±1.9	27.7±2.5	<0.0001
Body surface area (BSA), m^2 ‡	1.57±0.13	1.74±0.15	<0.0001
Systolic blood pressure (SBP), mmHg	116.6±12.3	121.1±11.8	<0.0001
Diastolic blood pressure (DBP), mmHg	71.4±9.9	72.3±10.3	0.277
Heart rate at CT, bpm	58±8	58±7	0.774
Total calcium score (median, min-max)	0 (0-1392)	0 (0-1861)	0.124
Hypertension, n	209	274	-
Gender female, n (%)	133 (63.6)	170 (62)	-
Diabetes mellitus, n (%)	28 (13.4)	61 (22.3)	-
Dyslipidemia, n (%)	116 (55.5)	163 (59.5)	-
Current cigarette smoking, n (%)	34 (16.3)	45 (16.4)	-
Age, y	61.1±7.8	59.3±7.8	0.013
Weight, kg	58.8±7.3	73.3±10.8	<0.0001
Height, cm	160.6±8.0	160.3±8.5	0.704
Waist, inch	35.6±3.5	40.2±4.5	<0.0001
Body mass index, kg/m^2†	22.7±1.6	28.4±2.9	<0.0001
Body surface area (BSA), m^2 ‡	1.61±0.14	1.77±0.17	<0.0001
Systolic blood pressure (SBP), mmHg	136.6±17.3	140.5±17.1	0.014
Diastolic blood pressure (DBP), mmHg	80.2±11.8	82.9±11.0	0.008
Heart rate at CT, bpm	58±7	58±8	0.635
Total calcium score, median (min-max)	12 (0-2614)	14 (0-2992)	0.445
Total fat area, cm^2	308.3±81.1	466.4±101.1	<0.0001
Visceral fat area,cm^2	118.8±43.5	176.5±53.6	<0.0001
Subcutaneous fat area,cm^2	189.6±62.9	289.9±86.3	<0.0001

Values are mean±SD unless indicated otherwise.
†Calculated as the weight in kilograms divided by the square of the height in meters.
‡Calculated with DuBois&DuBois formula.[18]
[a]Normal-weight versus obese

Table 2. Demographic Data in Normal Subjects

Characteristics	Female	Male
n (%)	197 (80)	47 (20)
Age, y	54.0±7.3	55.7±8.3
Weight, kg	53.7±5.7	63.3±6.9
Height, cm	156.3±5.2	167.3±6.1
Waist, inch	33.2±3.6	34.3±2.8
Body mass index, kg/m^2†	22.0±1.9	22.6±1.9
Body surface area (BSA), m^2 ‡	1.52±0.09	1.71±0.11
Systolic blood pressure (SBP), mmHg	116.5±12.5	117.3±12.1
Diastolic blood pressure (DBP), mmHg	70.5±10.1	73.1±10.0
Heart rate at CT, bpm	59±9	58±7
Total calcium score (median, min-max)	0, 0-455	0, 0-1114

Values are mean±SD unless indicated otherwise.
†Calculated as the weight in kilograms divided by the square of the height in meters.
‡Calculated with DuBois&DuBois formula.[18]

Table 3. Left Ventricular Parameters in Normal Population

Parameter	Female	Male	*p*
Ejection fraction (EF), %	66.2±6.5	64.4±7.7	0.097
End diastolic volume (EDV), mL	91.5±17.2	115.0±23.1	<0.0001
End systolic volume (ESV), mL	31.3±9.0	41.0±12.8	<0.0001
Stroke volume (SV), mL	60.2±11.6	74.0±17.3	<0.0001
Cardiac output (CO), L/min	3.5±0.9	4.2±1.1	<0.0001
Left ventricular mass (LVM), g	87.4±17.6	120.9±25.0	<0.0001
LVMI by BSA, g/m^2	57.5±11.3	70.6±14.2	<0.0001
LVMI by height, g/m	55.9±10.9	72.2±14.7	<0.0001
LVMI by height$^{2.7}$, g/m$^{2.7}$	26.2±5.2	30.2±6.5	<0.0001

LVMI = left ventricular mass index, BSA = body surface area

Table 4. Left Ventricular Function and Mass Normalized by Height$^{2.7}$ in Normal Population

‡Indexation parameter by height$^{2.7}$	Female	Male	*p*
LVEFI, %/m$^{2.7}$	19.9±2.7	16.1±2.5	<0.0001
LVEDVI, mL/m$^{2.7}$	27.4±4.9	28.8±6.4	0.17
LVESVI, mL/m$^{2.7}$	9.4±2.6	10.3±3.4	0.098
LVSVI, mL/m$^{2.7}$	18.0±3.4	18.5±4.7	0.5
CI, L/min/m$^{2.7}$	1.0±0.3	1.0±0.3	0.901
LVMI, g/m$^{2.7}$	26.2±5.2	30.2±6.5	<0.0001

‡All left ventricular parameters (ejection fraction, end-diastolic volume, end-systolic volume, stroke volume, cardiac output and left ventricular mass) divided by height$^{2.7}$

Table 5. Left ventricular function and mass in normotensive and hypertensive subjects

Parameters	Normotension			Hypertension		
	Normal-weight	Obese	P^a	Normal-weight	Obese	P^a
Ejection fraction (EF), %	65.4±6.7	65.7±6.2	0.629	66.7±6.1	65.9±6.7	0.148
End diastolic volume (EDV), mL	96.6±20.5	104.9±21.3	<0.0001	99.9±21.1	106.3±22.7	0.002
End systolic volume (ESV), mL	33.8±10.5	36.4±11.1	0.005	33.6±10.4	36.6±11.4	0.003
Stroke volume (SV), mL	62.8±13.8	68.5±13.5	<0.0001	66.4±13.7	69.6±15.2	0.014
Cardiac output (CO), L/min	3.6±0.9	4.0±1.1	<0.0001	3.8±0.9	4.2±3.2	0.074
Left ventricular mass (LVM), g	96.3±25.3	110.4±26.3	<0.0001	107.8±26.0	121.1±30.2	<0.0001
LVMI, g/m†	60.2±14.2	68.7±14.4	<0.0001	66.9±14.5	75.2±16.8	<0.0001
LVMI, g/m^2‡	61.0±13.4	63.1±11.9	0.062	66.6±13.4	68.2±14.1	0.218
LVMI, g/m$^{2.7}$§	27.1±5.7	30.9±5.8	<0.0001	29.8±6.0	33.6±7.0	<0.0001

LVMI = left ventricular mass index
Indexed by †height, ‡body surface area, §height$^{2.7}$

[a]Normal-weight versus obese

Table 6. Prevalence of left ventricular hypertrophy (LVH) in specific groups, according to sex-specific criterion for each indexation

	Normal population		Normotension				Hypertension			
			Normal-weight		Obese		Normal-weight		Obese	
	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men
	(197)	(47)	(290)	(109)	(133)	(65)	(133)	(76)	(170)	(104)
Age (y)	54.0±7.3	55.7±8.3	55.5±7.5	56.1±7.3	54.5±7.6	55.1±7.7	61.3±7.1	60.7±9.0	59.1±7.8	59.5±7.8
BMI (kg/m^2)	22.0±1.9	22.6±1.9	22.0±1.9	22.3±2.1	27.4±2.2	28.1±3.0	22.6±1.7	23.0±1.4	28.5±3.1	28.3±2.4
LVM (%)	17	13	15	11	35	29	29	18	51	43
LVM/BSA (%)	18	13	16	11	15	9	29	13	27	25
LVM/Height (%)	18	15	16	12	35	37	35	17	55	41
LVM/Height$^{2.7}$ (%)	19	17	17	15	35	40	35	13	58	43

BMI = body mass index
BSA = body surface area
LVM = left ventricular mass

Index parameters (e.g., LV mass index) were calculated by dividing each parameter (e.g., LV mass) by BSA, height, and height$^{2.7}$, respectively.

Statistical analysis

Data were analyzed using SPSS 11.5 software (SPSS Inc., Chicago, IL, USA). Data are expressed as mean with standard deviation (mean ± SD) and range. Categories were compared by χ^2statistics.Unpairedt-test was used to analyze quantitative variable between two groups.

Logistic regression analysis was used to determine the gender-specific independent predictors of LV hypertrophy in the entire population sample. For univariate procedures, the null hypothesis was always rejected at a two-tailed $p<0.05$.

Population-specific values for clear-cut definition of LVH were obtained in 244 normal subjects (80% women and 20% men who were normotensive, nondiabetic, with normal renal function [serum creatinine <1.5 mg/dL], no dyslipidemia or current use of lipid-lowering drug, no history of coronary artery disease, non-smoke, and body mass index <25 kg/m^2) drawn from the total population of 1080 patients, using mean ± SD of the distribution of LVM normalized for each measures of body size.

Results

Subject Characteristics

Table 1 showed 1080 subjects, 45% were hypertensive, 44% obese (64% women and 36% men), 43% dyslipidemia, and 12% diabetic.

Hypertensive patients were older than normotensive subjects ($p<0.0001$). Average body mass index was also higher in hypertensive than in normotensive subjects ($p<0.0001$).

Normal Left Ventricular Mass and Function

Table 2 showed subject characteristics of 244 normal populations in men and women.Table 3 presented the mean ± SD of left ventricular function and each index of LVM in the normal population. The upper normal limit of LVM/ height$^{2.7}$ in our study was lower than the established echocardiographic criteria for LVH[20] (31 versus 44 g/m$^{2.7}$ in women; 36 versus 48 g/m$^{2.7}$ in men). The mean LV mass in our study (in both normal men and women subjects) was close to the published data in Asian-American population from MESA study[14] (87 versus 89 gin women; 120 versus 129 g in men).

Men exhibited greater values than women (all $p<0.0001$), except for left ventricular ejection fraction (LVEF). Only LVMI and LVEF remained statistically significant difference (LVMI–men greater than women, $p<0.0001$; LVEFI–women greater than men, $p<0.0001$) after normalization of the parameters by height$^{2.7}$ (Table 4).

Gender Differences for Left Ventricular Mass and Function

Significant differences between genders of entire population were seen for all global measurements of left ventricular function and mass. All LV parameters were statistically significant higher in men than women ($p<0.0001$) except for LVEF which was greater in women than men ($p<0.0001$). The mean LVEF was 64.7±6.6 % in men and 66.3±6.3 % in women ($p<0.0001$). The mean LV mass was 131.2±27.2 g in men and 95.8±21.1 g in women ($p<0.0001$).

When normalized by BSA, these differences remained statistically significant for all parameters ($p<0.0001$) except cardiac output. When indexed by height with allometric power of 2.7, these differences between genders remained statistically significant only in LVMI (28.9±6.2 g/m$^{2.7}$ in women and 32.4±6.8 g/m$^{2.7}$ in men, $p<0.0001$), LVEFI (20.1±2.8%/m$^{2.7}$ in women and 16.1±2.2%/m$^{2.7}$ in men, $p<0.0001$) and LVESVI (9.7±2.7 mL/m$^{2.7}$ in women and 10.1±3.0 mL/m$^{2.7}$ in men, p=0.011).

LV Mass and Function Measurements in Normal-Weight and Obese Subjects

LV mass was higher in obese than in normal-weight subjects in both normotensive (110.4±26.3 g versus 96.3±25.3 g, $p<0.0001$) and hypertensive groups (121.1±30.2 g versus 107.8±26.0 g, $p<0.0001$), with parallel differences in both genders. This difference was confirmed by normalizing LV mass for height$^{2.7}$ (30.9±5.8 g/m$^{2.7}$ versus 27.1±5.7 g/m$^{2.7}$ in normotensive [$p<0.0001$] and 33.6±7.0 g/m$^{2.7}$ versus 29.8±6.0 g/m$^{2.7}$ in hypertensive [$p<0.0001$] subjects) but was statistically insignificant for LV mass/BSA.

Distribution of Left Ventricular Mass and Function in Normal-Weight and Obese Normotensive and Hypertensive Subjects

Table 6 showed the sex-specific prevalence of LVH in relation to type of normalization for body size in the specific group of population. LVH was substantially more prevalent in women than in men with all indexation methods in almost all specific groups except obese normotensive group which higher prevalent in men than in women. The prevalence of LVH substantially increased with normalization for height or height$^{2.7}$ and was lower with normalization for BSA. Nonindexed LVM identified an intermediate prevalence of LVH between that recognized by BSA and those by height-based normalizations.

Normotensive Subjects

The prevalence of LVH using LV mass/height$^{2.7}$-based criteria was 16% in normal-weight normotensive subjects (17% in women and 15% in men), in the range close to a normal population sample (19% in women and 17% in men). In obese normotensive subjects, the prevalence of LVH was higher (35% in women and 40% in men).Using LV mass/BSA criteria, the prevalence of LVH in normal-weight normotensive subjects was 16% in women and 11% in men whereas in obese normotensive subjects was 15% in women and 9% in men.

Hypertensive Patients

In hypertensive patients, LV mass/height$^{2.7}$-based criteria identified 35% women and 13% men of normal-weight, as well as 58% women and 43% men of obese patients as having LVH, a difference that lower detected using LV mass/BSA criteria (29% in women and 13% in men of normal-weight, and 27% in women and 25% in men of obese patients).

Effects of Gender on the Relations of Hypertension and Obesity to Left Ventricular Mass and Function

Effect of obesity on Left Ventricular Mass in Normotensive Women and Men

LV mass was increased in both obese men and women (33.4±6.0 and 29.7±5.3 g/m$^{2.7}$) as compared with normal-weight persons (29.9±6.1 and 26.2±5.3 g/m$^{2.7}$, p<0.0001 in both genders).This difference was not detected if LVM was normalized with BSA (59.1±9.8 versus 57.4±11.4 g/m^2 [p = 0.146] in obese and normal-weight normotensive women; 71.4±11.7 versus 70.6±13.6 g/m^2 [p = 0.704] in obese and normal-weight normotensive men).

Effect of obesity on Left Ventricular Mass in Hypertensive Women and Men

LVM/height$^{2.7}$ was higher in obese hypertensive patients, both among women (32.7±6.6 versus 29.1±5.6 g/m$^{2.7}$, p<0.0001) and men (35.2±7.3 versus 31.2±6.4 g/m$^{2.7}$, p<0.0001).

Predictors of Left Ventricular Hypertrophy

Multiple logistic regression analysis was performed in pooled normotensive and hypertensive men and women using age, systolic and diastolic blood pressure, heart rate and body mass index as continuous variables to determine the independent predictors of LVH. In both men and women, the most potent predictor of LVH was body mass index (p<0.0001 in both genders).Additional predictors in both men and women were high systolic blood pressure (p<0.0001 in both genders). Equation for predicting LVH in men and women were:

$$\text{Estimated probability of LVH} = 1/(1+e^{-w})$$

Where w is as follows:

w (men) = 0.02*systolic pressure + 0.25*BMI – 9.86

w (women) = 0.03*systolic pressure + 0.17*BMI – 8.82

Discussion

Normalization of LVM for body size is widely used to compare individuals with different body builds and to identify groups at high risk for cardiovascular events.

We reported on the normal values of left ventricular function and mass as well as the cut-off values for left ventricular hypertrophy in normal Thai population in both genders using normalization data of LVM and volume taken from 320-slice cardiac CT angiography[21]. The LV function and mass in the present study subjects were lower than the prior published data which were mostly collected in American or European populations. However, it was close to but still lower than the data in the Asian-American subgroup in previous publication[14].This assumes that not only ethnicity but also the environment and socioeconomic status may have effect to LV function and mass.

With this cut-off value, the present study identified higher prevalent of LVH compared to previous report[11].

Normalization of left ventricular mass and function by height with allometric power of 2.7 minimizes genders difference in left ventricular function and mass. Men exhibited greater value of LVMI than women while LVEFI was lower in men compared to women.

Normalization of LVM by BSA underestimated the prevalence of LVH especially in obese subjects. Therefore, if the height$^{2.7}$-based criterion is taken as the basis of cardiovascular disease prevention program, reducing BP is no longer sufficient to eliminate incident events attributable to LVH, and intervention regarding reduction of obesity might also be required. Identification of preclinical LVH in a normal population as high as 19% in women and 17% in men opens up the possibility of targeting primary prevention interventions using our proposed LVH predicting equations to the general preventive measure of reducing causal cardiovascular risk factors.

Limitations and Perspectives

There were several limitations in the present study. We reported data from cross-sectional analytic study. Therefore, our exhibited cut-off value for LVH might not actually relate to true cardiovascular event. We expected to be able to analyze the relation of our reported data to incident of cardiovascular event in the future study. There was small number of male subjects in normal reference group (male 47, female 197), thus the left ventricular mass and function obtained from the present study might not strongly reflected the exact data in normal male population. The ability to properly collect an adequate number of normal male reference samples will allow more accurate quantification of LVH cut-off value and prevalence. The reason for LV mass and function differences between the present study population and other reported studies even in the Asian-American subgroup in MESA study[14] might be related to factors such as socioeconomic status or environment. Further evaluation of the effect of these factors to LV parameters is needed in clinical or research setting.

Conclusion

Obesity is an independent stimulus to increase LVM in normotensive subjects, and its effect is additive in hypertensive patients. LVM/BSA underestimated the prevalence of LVH especially in obese subjects. Male has higher LVM but lower prevalence of LVH as compared to female. Gender and obesity affect to both LVM and prevalence of LVH.

What is already known on this topic?

The measurement of left ventricular parameters especially LVM can be measured by cardiac CT and are highly reproducible.[21] Heart size differs in subjects of different body size and LV mass and volume strongly relate to body size. It has been shown that obesity and hypertension are associated with increased left ventricular(LV) mass[1,2].

What is this study adds?

Obesity is an independent stimulus to increase LVM in normotensive subjects and its effect is additive in hypertensive patients. Gender and obesity affect LVM and prevalence of LVH.

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Potential conflicts of interest

None.

References

1. Kannel WB, Cobb J. Left ventricular hypertrophy and mortality--results from the Framingham Study. Cardiology 1992; 81: 291-8.
2. Benjamin EJ, Levy D. Why is left ventricular hypertrophy so predictive of morbidity and mortality? Am J Med Sci 1999; 317: 168-75.
3. Alpert MA, Lambert CR, Panayiotou H, Terry BE, Cohen MV, Massey CV, et al. Relation of duration of morbid obesity to left ventricular mass, systolic function, and diastolic filling, and effect of weight loss. Am J Cardiol 1995; 76: 1194-7.
4. Avelar E, Cloward TV, Walker JM, Farney RJ, Strong M, Pendleton RC, et al. Left ventricular hypertrophy in severe obesity: interactions among blood pressure, nocturnal hypoxemia, and body mass. Hypertension 2007; 49: 34-9.

5. de la Maza MP, Estevez A, Bunout D, Klenner C, Oyonarte M, Hirsch S. Ventricular mass in hypertensive and normotensive obese subjects. Int J Obes Relat Metab Disord 1994; 18: 193-7.
6. Lauer MS, Anderson KM, Kannel WB, Levy D. The impact of obesity on left ventricular mass and geometry. The Framingham Heart Study. JAMA 1991; 266: 231-6.
7. Morricone L, Malavazos AE, Coman C, Donati C, Hassan T, Caviezel F. Echocardiographic abnormalities in normotensive obese patients: relationship with visceral fat. Obes Res 2002; 10: 489-98.
8. 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; 110: 3081-7.
9. de Simone G, Devereux RB, Roman MJ, Alderman MH, Laragh JH. Relation of obesity and gender to left ventricular hypertrophy in normotensive and hypertensive adults. Hypertension 1994; 23: 600-6.
10. de Simone G, Devereux RB, Daniels SR, Mureddu G, Roman MJ, Kimball TR, et al. Stroke volume and cardiac output in normotensive children and adults. Assessment of relations with body size and impact of overweight. Circulation 1997; 95: 1837-43.
11. de Simone G, Kizer JR, Chinali M, Roman MJ, Bella JN, Best LG, et al. Normalization for body size and population-attributable risk of left ventricular hypertrophy: the Strong Heart Study. Am J Hypertens 2005; 18: 191-6.
12. Lavie CJ, Messerli FH. Cardiovascular adaptation to obesity and hypertension. Chest 1986; 90: 275-9.
13. Okwuosa TM, Hampole CV, Ali J, Williams KA. Left ventricular mass from gated SPECT myocardial perfusion imaging: comparison with cardiac computed tomography. J Nucl Cardiol 2009; 16: 775-83.
14. Natori S, Lai S, Finn JP, Gomes AS, Hundley WG, Jerosch-Herold M, et al. Cardiovascular function in multi-ethnic study of atherosclerosis: normal values by age, sex, and ethnicity. AJR Am J Roentgenol 2006; 186 (6 Suppl 2): S357-65.
15. Bastarrika G, Arraiza M, De Cecco CN, Mastrobuoni S, Ubilla M, Rabago G. Quantification of left ventricular function and mass in heart transplant recipients using dual-source CT and MRI: initial clinical experience. Eur Radiol 2008; 18: 1784-90.
16. Yamamuro M, Tadamura E, Kubo S, Toyoda H, Nishina T, Ohba M, et al. Cardiac functional analysis with multi-detector row CT and segmental reconstruction algorithm: comparison with echocardiography, SPECT, and MR imaging. Radiology 2005; 234: 381-90.
17. World Health Organization. The Asia-Pacific perspective: redefining obesity and its treatment. Manila, Philippine: WHO Western Pacific Region; 2000.
18. DuBois D, DuBois DF. A formula to estimate the approximate surface area if height and weight be known. Arch Int Med 1916; 17: 863-71.
19. de Simone G, Daniels SR, Devereux RB, Meyer RA, Roman MJ, de Divitiis O, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. J Am Coll Cardiol 1992; 20: 1251-60.
20. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005; 18: 1440-63.
21. Jongjirasiri S, Sritara C, Waeosak P, Laothamatas J. Normal data of left ventricular parameters in non-hypertensive patients using 320-slice cardiac CT: a study on intra- and interobserver variability. J Med Assoc Thai 2011; 94: 1053-60.

อิทธิพลของภาวะอ้วนที่มีต่อผลมวลมวลกล้ามเนื้อหัวใจ: ผลการศึกษาจาก เอกซเรย์คอมพิวเตอร์ 320 ไสลด์

สุทธิพงษ์ จงจิระศิริ, พันเลิศ แววศักดิ์, ชนิกา ศรีธารา, จิรพร เหล่าธรรมทัศน์, ศุภขจี แสงเรืองอ่อน

วัตถุประสงค์: ศึกษาวิจัยเพื่อหาความแตกต่างของการทำงานของหัวใจห้องซ้าย (left ventricular function) มวลกล้ามเนื้อของหัวใจห้องซ้ายล่าง (left ventricular mass) โดยเปรียบเทียบในกลุ่มตัวอย่างที่มีภาวะอ้วนและกลุ่มตัวอย่างที่ไม่มีภาวะอ้วนจากกลุ่มที่มีความดันโลหิตปกติและกลุ่มที่มีความดันโลหิตสูง

วัสดุและวิธีการ: ผู้วิจัยและคณะได้ทำการศึกษามวลกล้ามเนื้อของหัวใจห้องซ้ายล่าง (left ventricular mass) จากข้อมูลที่ได้จากการตรวจหัวใจด้วยเอกซเรย์คอมพิวเตอร์ความเร็วสูง (320 ไสลด์) โดยปรียบเทียบ จากกลุ่มที่มี ความดันโลหิตปกติจำนวน 597 ราย (ชาย =175, [ภาวะอ้วน 65]และ หญิง = 422 [ภาวะอ้วน 133], อายุ 55+ 7 ปี) และในผู้กลุ่มที่มีความดันโลหิตสูงจำนวน 483 ราย (ชาย =180, [ภาวะอ้วน 104]และ หญิง = 303 [ภาวะอ้วน 170], อายุ 60+ 7ปี) ภาวะอ้วนของทั้งเพศชายและเพศหญิงของคนเอเชีย ถูกกำหนดเมื่อดัชนีมวลกาย (BMI)> 25 kg/m^2) ข้อมูลของมวลกล้ามเนื้อของหัวใจห้องซ้ายล่าง (left ventricular mass) จะนำมาคำนวณตามพื้นผิวของร่างกาย (body surface area) และความสูง$^{2.7}$

ผลการศึกษา: พบว่าค่าปกติ (upper normal limit) ของค่าของมวลกล้ามเนื้อของหัวใจห้องซ้ายล่าง (left ventricular mass) ในกลุ่มตัวอย่างที่มีความเสี่ยงต่ำ (กลุ่มไม่สูบบุหรี่, ไม่มีความดัน, ไม่มีเบาหวาน,ไม่มีโรคหัวใจ และมีระดับไขมันในเลือดปกติ) เมื่อเทียบกับกลุ่มที่มีความเสี่ยงสูงมีความแตกต่างระหว่างเพศชายและเพศหญิงอย่างมีนัยสำคัญทางสถิติโดยในกลุ่มที่มีความเสี่ยงต่ำมีค่าต่ำกว่าค่าเกณฑ์สำหรับ left venricular hypertrophy (LVH) เมื่อเทียบกับกลุ่มที่มีความเสียงสูง ตามลำดับดังนี้ 31; 44 $g/m^{2.7}$ ในเพศหญิงและ 36 ; 48 $g/m^{2.7}$ ในเพศชายนอกจากนี้ยังพบว่าในกลุ่มตัวอย่างของทั้ง 2 กลุ่มที่มีความดันโลหิตสูงและไม่มีความดันโลหิตสูงพบว่ากลุ่มที่มีภาวะอ้วนจะมีความชุก (prevalence) ของ LVH และ LVM/height$^{2.7}$สูงกว่ากลุ่มที่ไม่มีภาวะอ้วนอย่างมีนัยสำคัญทางสถิติ ($p<0.001$) ทั้งเพศหญิงและชาย โดยในกลุ่มที่มีภาวะอ้วนที่มีความดันโลหิตสูงกับกลุ่มภาวะอ้วนที่มีความดันปกติในเพศหญิงมีความชุกของ LVH และ LVM/height$^{2.7}$ เท่ากับ 58%; 34% และในเพศชายเท่ากับ 43%; 14% ตามลำดับ ขณะที่กลุ่มภาวะอ้วนที่มีความดันปกติเทียบกับกลุ่มที่ไม่มีภาวะอ้วนที่มีความดันปกติ ในเพศหญิงมีค่าเท่ากับ 35% ;16% และ 40% versus 15% ในเพศชายตามลำดับ เมื่อนำมาประมวลด้วย logistgistric regression analysis พบว่า systolic blood pressure และค่า BMI เป็นค่าที่สำคัญที่สุดในการทำนาย ภาวะ LVH ทั้งเพศชายและหญิงโดยสามารถประมวลจากสูตร Risk of LVH = $1/(1+e^{-w})$ โดยที่ w (men) = 0.02* systolic pressure + 0.25*BMI — 9.86c และ w (women) = 0.03* systolic pressure + 0.17*BMI — 8.82

สรุป: กลุ่มภาวะอ้วนเป็นตัวกระตุ้นให้มีการเพิ่มค่าของมวลกล้ามเนื้อของหัวใจห้องซ้ายล่าง (left ventricular mass)ในกลุ่มที่มีความดันปกติ ขณะที่กลุ่มที่มีความดันโลหิตสูงจะเป็นปัจจัยเสริมมากขึ้น เพศและภาวะอ้วนมีผลต่อความชุก (prevalence) ของLVH