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Visceral obesity, but not central obesity, is associated with cardiac remodeling in subjects with suspected metabolic syndrome

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KEYWORDS
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Abstract *Background and aims:* Metabolic syndrome (MetS) is a cluster of multiple risk factors including central obesity that may lead to cardiac damage and cardiovascular events. We investigated whether visceral obesity induces cardiac structural and functional remodeling independently from central obesity and other risk factors in subjects with suspected MetS.
Methods and results: We studied 229 participants with suspected MetS. Visceral fat area (VFA) was measured by bioelectrical impedance analysis. Left ventricular (LV) mass index, early diastolic velocity of mitral annulus (*e'*), and LV global longitudinal strain (GLS) were measured by echocardiography. Subjects were categorized into high and low VFA group (VFA_h and VFA_l). MetS was more prevalent in the VFA_h than in the VFA_l ($p = 0.004$). The VFA_h had a higher waist circumference (WC) than the VFA_l ($p < 0.001$). LV mass index was higher, but *e'* and GLS were lower in the VFA_h than in VFA_l (all $p < 0.05$). VFA was well correlated with blood pressure, fasting blood glucose, triglyceride, high-sensitivity C-reactive protein and adiponectin (all $p < 0.05$). VFA was correlated to LV mass index, *e'*, and GLS (all $p < 0.05$) and was independently associated with GLS after adjustment for other risk factors, including WC ($p = 0.005$).
Conclusions: Visceral obesity assessed by VFA was well correlated with parameters of MetS. Visceral obesity, but not central obesity measured by WC, was independently associated with structural and functional cardiac remodeling in subjects with suspected MetS. It suggests that visceral obesity should be considered as an important risk factor for cardiac damage in dysmetabolic subjects.
Trial registration: NCT02077530 (date of registration: November 1, 2013).

List of Abbreviations: MetS, metabolic syndrome; 2D, two-dimensional; LV, left ventricular; GLS, global longitudinal strain; VFA, visceral fat area; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL, high-density lipoprotein; TG, triglycerides; hs-CRP, high-sensitivity C-reactive protein; VFA_l and VFA_h, the low and high VFA group; *e'*, early diastolic velocity of mitral annulus.

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Introduction

Metabolic syndrome (MetS) is a cluster of multiple risk factors including central obesity that leads to cardiac damage and cardiovascular events [1]. The independent role of obesity in MetS has been debated, as it is strongly associated with other traditional components [2]. Indeed, the adverse impacts of obesity on the structure and function of the heart have been recently reported [3,4].

Visceral adipose tissue is a bioactive organ that secretes several adipokines and is a source of proinflammatory and proatherogenic cytokines [5–7]. Visceral obesity is a potent risk factor for the development of cardiometabolic diseases including hypertension, diabetes mellitus, coronary artery disease, and heart failure [8–10]. Furthermore, visceral obesity has been reported to have a more significant correlation with insulin resistance compared to central obesity assessed by waist circumference (WC) [11]. However, it is still unclear whether visceral obesity associates with cardiac structure and function independently of each component of MetS including central obesity.

Recently, two-dimensional speckle-tracking (2DS) echocardiography has been reported as a useful and sensitive method for the evaluation of cardiac function [12]. Left ventricular (LV) strain measured by 2DS echocardiography has enabled the sensitive evaluation of myocardial deformation [13]. In one community study, LV systolic dysfunction assessed by global longitudinal strain (GLS) reflected early myocardial contractile change and was found to be an independent prognostic factor for cardiovascular events [14].

Therefore, we investigated the association of visceral obesity with cardiac structural and functional remodeling using 2DS echocardiography in subjects with suspected MetS.

Methods

Study population

The Seoul Metabolic Syndrome study is a prospective cohort evaluating the clinical characteristics and outcome of subjects with suspected MetS referred from 25 public health centers in the Seoul metropolitan area. Detailed protocols have been reported in previously published papers [15]. A total of 229 consecutive participants who agreed to undergo both conventional and 2DS echocardiography were enrolled in this study. Exclusion criteria included a previous history of stroke, angina pectoris, myocardial infarction, or any revascularization procedure. At the first visit, demographic data, medical history, and concomitant medications were investigated, and physicians performed a basic physical examination. Anthropometric data including body weight, height, WC, body mass index (BMI) and systolic and diastolic blood pressure (SBP and DBP) were collected. General obesity was defined as BMI $\geq$ 25 kg/m^2. Written informed consent was obtained from all the participants, and this study was approved by the institutional review board of Korea University Anam Hospital.

Laboratory parameters

Blood samples were collected from each participant after fasting for 8 h before the first visit. Fasting blood glucose and lipid profile including total cholesterol, triglycerides (TG), high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein cholesterol were measured. High-sensitivity C-reactive protein (hs-CRP), apolipoprotein A1, apolipoprotein B, and adiponectin were measured using the immunoturbidimetry assay.

Metabolic syndrome components

MetS was evaluated according to the updated National Cholesterol Education Program/Adult Treatment Panel III criteria for Asian Americans [16]. A subject who had at least three of the following components was defined as having MetS: 1) central obesity: WC $\geq$ 90 cm for men and $\geq$80 cm for women; 2) hypertension: SBP $\geq$ 130 mmHg or DBP $\geq$ 85 mmHg; 3) fasting blood glucose $\geq$100 mg/dL; 4) abnormal HDL: HDL cholesterol level $<$40 mg/dL for men and $<$50 mg/dL for women; and 5) abnormal TG: TG level $\geq$150 mg/dL.

Dual bioelectrical impedance analysis for visceral fat measurement

VFA and subcutaneous fat area were measured using dual bioelectrical impedance analysis at the umbilicus level (Omron HDS-2000). Trained nurses used this device according to the protocol provided by the manufacturer. Previously, VFA measured by dual bioelectrical impedance analysis has been reported to show a good correlation with VFA measured by computed tomography [5,17]. Subjects were divided into high VFA and low VFA groups (VFA_h and VFA_l) according to the median VFA value used in this study.

Echocardiographic study

Both conventional and 2DS echocardiography were conducted in all patients using a commercially available echocardiographic system (Vivid-E9, Vingmed-General Electric, Horten, Norway) with an M5Sc transducer. According to the recommendation by the American Society of Echocardiography, conventional echocardiographic views were obtained [18]. Chamber quantification was performed using 2D echocardiography images, while LV end diastolic volume, LV end systolic volume, and LV ejection fraction were measured by biplane Simpson's method from the apical four- and two-chamber view. Left atrial volume index and LV mass were calculated using the formula recommended by the American Society of Echocardiography [18]. LV mass index was normalized by height$^{2.7}$ [19]. Early diastolic velocity of mitral inflow and late diastolic velocity were evaluated by pulsed wave Doppler in the apical four-chamber view. Early diastolic velocity (*e'*) of the septal and lateral mitral annulus was derived by pulsed-wave tissue Doppler image [20].

2DS echocardiography was performed in each subject according to the previously reported protocol [18]. The second-harmonic B-mode images of three standard apical views of the LV were obtained and stored digitally in digital cineloop format. Images of three consecutive cardiac cycles were obtained while holding the breath. LV global longitudinal strain (GLS) was calculated as the mean longitudinal peak negative strain from 18 apical segments and analyzed using EchoPac-PC software (version 201.67.0).

Statistical analysis

All data are presented as means ± SD for continuous variables or frequencies (percentages) for categorical variables. The differences in clinical, laboratory, and echocardiographic variables between the VFA_h and VFA_l were compared using the chi-square test or Student's *t*-test. The correlation between the VFA and clinical, laboratory, and echocardiographic parameters was evaluated by correlation analysis. The mean GLS value was compared according to the presence of MetS components. Multiple linear regression analysis was performed to determine the association between VFA and myocardial function (lateral *e'* velocity and GLS) after adjustment for age, sex, general obesity, WC, hypertension, anti-hypertensive medication, fasting glucose level, triglyceride level, HDL level, LV mass index and LV ejection fraction. Multicollinearity was tested by variance inflation factor using all covariates of the final model. SPSS version 24.0 for Windows (IBM, NY, USA) was used to perform the statistical analysis. A two-sided p-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Table 1 shows the baseline clinical and laboratory characteristics of the study population. The mean age was 56.4 ± 4.5 years, and 115 (50.2%) subjects were male. A total of 85 (37.1%) subjects had MetS. Twelve (5.4%) subjects were taking antihypertensive medications, while 21 (9.2%) subjects were taking statins.

When divided by the median VFA (76 cm^2), 113 patients had a high VFA and 116 patients had a low VFA, and there was a significant difference in the VFA between the two groups ($p < 0.001$). Comparing the VFA_h and VFA_l groups, there was no difference in the mean age, but there were more males in the VFA_h. BMI, WC, SBP and DBP were all

Table 1 Clinical and laboratory characteristics.

	Total (n = 229)	VFA_l (n = 116)	VFA_h (n = 113)	P value
Age (y)	56.4 ± 4.5	56.6 ± 4.0	56.2 ± 5.0	0.430
Male (n, %)	115 (50.2%)	35 (30.2%)	80 (70.8%)	<0.001
BMI (kg/m^2)	25.6 ± 2.8	24.3 ± 2.2	26.9 ± 2.8	<0.001
General Obesity (n, %)	127 (55.5%)	40 (34.5%)	87 (77.0%)	<0.001
WC (cm)	87.4 ± 7.63	83.2 ± 6.1	91.8 ± 6.5	<0.001
SBP (mmHg)	123.8 ± 15.0	120.3 ± 13.9	127.4 ± 15.3	<0.001
DBP (mmHg)	75.9 ± 9.8	73.8 ± 9.6	77.9 ± 9.6	0.001
Pulse rate (BPM)	71.2 ± 10.5	70.8 ± 9.6	71.6 ± 11.4	0.585
VFA (cm^2)	78.5 ± 28.5	55.8 ± 14.4	101.7 ± 18.9	<0.001
SFA (cm^2)	186.7 ± 46.6	186.1 ± 48.8	187.3 ± 44.5	0.849
Metabolic syndrome				
HTN	88 (38.4%)	35 (30.2%)	53 (46.9%)	0.007
IFG	89 (38.9%)	35 (30.2%)	54 (47.8%)	0.005
Central obesity	153 (66.8%)	66 (56.9%)	87 (77.0%)	0.001
High TG	84 (36.7%)	33 (28.4%)	51 (45.1%)	0.006
Low HDL	66 (28.8%)	35 (30.2%)	31 (27.4%)	0.378
Number of components	2.1 ± 1.1	1.8 ± 1.1	2.4 ± 1.1	<0.001
≥3 Number of components	85 (37.1%)	33 (28.4%)	52 (42.0%)	0.004
Medications				
Antihypertensive (n, %)	12 (5.4%)	6 (5.3%)	8 (7.4%)	0.177
OHA (n, %)	2 (0.9%)	2 (1.8%)	0 (0.0%)	0.256
Statins (n, %)	21 (9.2%)	9 (8.1%)	12 (11.1%)	0.300
Laboratory findings				
FBG (mg/dL)	99.3 ± 13.0	96.3 ± 11.0	102.3 ± 14.1	<0.001
TC (mg/dL)	198.5 ± 31.5	200.7 ± 34.2	196.1 ± 28.5	0.274
HDL (mg/dL)	52.1 ± 13.5	54.1 ± 13.2	50.1 ± 13.7	0.025
TG (mg/dL)	146.6 ± 78.4	125.7 ± 65.5	168.0 ± 84.8	<0.001
TG/HDL ratio	3.1 ± 2.3	2.5 ± 1.6	3.8 ± 2.7	<0.001
Apolipoprotein AI (mg/dL)	142.6 ± 25.5	142.6 ± 24.7	142.4 ± 26.5	0.953
Apolipoprotein B (mg/dL)	101.2 ± 22.4	101.9 ± 24.9	100.6 ± 19.7	0.662
Adiponectin (ng/mL)	10.09 ± 6.25	11.94 ± 6.83	8.19 ± 4.96	<0.001
Hs-CRP (mg/dL)	1.30 ± 2.19	0.96 ± 1.45	1.64 ± 2.72	0.018

Abbreviations: VFA and SFA, visceral and subcutaneous fat area; VFA_l and VFA_h, the low and high VFA group; BMI, body mass index; WC, waist circumference; SBP and DBP, systolic and diastolic blood pressure; HTN, hypertension; IFG, impaired fasting glucose; TG, triglycerides; HDL, high-density lipoprotein; OHA, oral hypoglycemic agents; FBG, fasting blood glucose; TC, total cholesterol; hs-CRP, high sensitivity C-reactive protein.

higher in the VFA_h than in the VFA_l; however, the subcutaneous fat area did not differ between the groups.

The VFA_h had a higher prevalence of MetS components, with the exception of low HDL. The prevalence of MetS was also higher in the VFA_h. In terms of laboratory characteristics, the VFA_h showed a higher TG/HDL ratio and hs-CRP and a lower adiponectin level compared to the VFA_l.

Visceral fat area and its association with clinical and laboratory parameters

VFA showed a good correlation with traditional MetS components and other clinical parameters. In terms of clinical parameters, VFA was related to obesity parameters including BMI and WC. VFA was also well correlated with SBP and DBP but was not associated with age. Among the laboratory parameters, VFA was positively correlated with fasting blood glucose, TG, TG/HDL ratio, and hs-CRP and negatively correlated with HDL and adiponectin (Table 2).

The association of visceral fat area with cardiac structural and functional remodeling

In the total subjects, the mean LV mass index value was 33.1 ± 7.7 $g/m^{2.7}$, and 12 patients (4.3%) had LV concentric remodeling and 7 (2.5%) patients had LV hypertrophy in these subjects. However, LV mass index was larger in the VFA_h than in the VFA_l.

Table 2 Correlation between VFA and other parameters.

	Coefficient	P value
Clinical parameters		
Age	−0.072	0.280
Sex	0.442	<0.001
BMI	0.584	<0.001
WC	0.683	<0.001
SBP	0.251	<0.001
DBP	0.238	<0.001
Laboratory parameters		
FBG	0.245	<0.001
TG	0.265	<0.001
HDL	−0.158	0.017
TG/HDL	0.251	<0.001
Hs-CRP	0.166	0.012
Adiponectin	−0.324	<0.001
Echocardiographic parameters		
RWT	0.086	0.196
LV mass index	0.213	0.001
LAVI	−0.057	0.389
LVEF	0.001	0.992
Septal *e'*	−0.129	0.052
Lateral *e'*	−0.218	0.001
E/*e'*	0.108	0.105
LV GLS	−0.405	<0.001

Additional abbreviations: RWT, relative wall thickness. LV, left ventricular; LAVI, left atrial volume index; EF, ejection fraction; *e'*, septal early mitral tissue velocity; E, early diastolic mitral inflow velocity; GLS, global longitudinal strain. Other abbreviations are described in Table 1.

Although LV ejection fraction, E/e' ratio, LA volume index were not different between the groups, E-velocity, septal and lateral *e'* velocity as parameters of diastolic function, and LV GLS as systolic function were more significantly impaired in the VFA_h than in the VFA_l (Table 3). VFA was significantly related to LV mass index (Fig. 1a) and was also correlated with lateral *e'* velocity and LV GLS (Table 2, Fig. 1b).

Comparing LV GLS according to the presence of MetS, central obesity defined by WC criteria, and visceral obesity defined by VFA, a significant difference in the LV GLS value was observed only in the groups divided by VFA (−17.2% ± 2.0% vs −15.3% ± 2.5%, p = 0.001) (Fig. 2).

In multiple linear regression analysis with clinical and other metabolic risk factors including WC, VFA was significantly associated with LV GLS even after adjustment for other cardiovascular risk factors including WC (β = −0.019, p = 0.005) (Table 4).

Discussion

To our knowledge, this is the first study to demonstrate that visceral obesity is independently associated with cardiac structural and functional remodeling in a middle-aged population with suspected MetS but no apparent cardiovascular diseases. In addition, we demonstrated that: 1) The VFA_h tended to have more MetS and its components compared to the VFA_l; 2) Even though the LV ejection fraction was not different between groups, LV mass index was increased for cardiac structural remodeling and *e'* velocity and LV GLS were reduced for cardiac functional remodeling in the VFA_h compared to the VFA_l; and 3) VFA was well correlated with SBP, DBP, dyslipidemia, hs-CRP, and adiponectin.

Visceral obesity and myocardium

While the exact pathogenic mechanism by which visceral obesity may cause cardiac damage remains unclear, various mechanisms have been suggested. With excessive visceral adipose tissue accumulation, macrophages infiltrate visceral adipose tissue and induce visceral adipose tissue to secrete proinflammatory cytokines [21]. These various proinflammatory cytokines including plasminogen activator inhibitor type-1, tumor necrosis factor-a, and interleukin 1 and 6, have local and systemic effects on the myocardium. Epicardial adipose tissue, as a localized visceral adipose tissue in the heart, may directly influence the myocardium by a paracrine effect [22]. Systemic inflammation may cause endothelial dysfunction including in the coronary artery and then induce cardiac damage through myocardial ischemia [3]. Previous experimental studies reported the role of visceral adipose tissue as an active source of adipokines [6,7]. Circulating leptin may change the fatty acid metabolism of myocardium and induce myocardial hypertrophy, fibrosis, and cardiac dysfunction [23]. Adiponectin is negatively correlated with BMI and protects the heart from adverse cardiac remodeling [24]. In patients with visceral obesity, heme

Table 3 Echocardiographic characteristics.

	Total (n = 229)	VFA_l (n = 116)	VFA_h (n = 113)	P value
Pulse rate (/min)	70.9 ± 11.5	69.7 ± 11.3	72.1 ± 11.6	0.120
RWT	0.309 ± 0.067	0.307 ± 0.063	0.312 ± 0.072	0.562
LV mass index ($g/m^{2.7}$)	33.1 ± 7.7	31.7 ± 6.7	34.5 ± 8.4	0.007
LV concentric remodeling (n, %)	12 (4.3%)	3 (2.6%)	9 (8.0%)	0.167
LV hypertrophy (n, %)	7 (2.5%)	3 (2.6%)	4 (3.5%)	
LAVI (mL/m^2)	33.1 ± 10.7	33.8 ± 9.8	32.4 ± 11.4	0.304
EF (%)	59.1 ± 6.0	59.2 ± 6.3	59.1 ± 7.0	0.950
E (cm/s)	54.6 ± 13.4	56.9 ± 14.1	52.1 ± 12.3	0.007
DT (ms)	190.8 ± 43.9	187.6 ± 44.9	194.2 ± 42.7	0.269
Septal e′ (cm/s)	6.3 ± 1.6	6.5 ± 1.5	6.1 ± 1.6	0.028
Lateral e′ (cm/s)	8.7 ± 2.1	9.1 ± 2.0	8.3 ± 2.1	0.004
E/e′ ratio	7.5 ± 1.9	7.5 ± 1.8	7.5 ± 2.0	0.897
LV GLS (%)	−16.3 ± 2.4	−17.2 ± 2.0	−15.3 ± 2.5	<0.001

Abbreviation: DT, deceleration time. Other abbreviations are described in Table 2.

oxygenase-1, an anti-oxidative enzyme, was lower in visceral adipose tissue than in subcutaneous adipose tissue, and it was negatively correlated with insulin resistance defined by the homeostasis model assessment of insulin resistance [25]. Visceral obesity-related insulin resistance increases myocardial fatty acid metabolism and results in lipotoxicity in the myocardium [26,27]. However, in our study, although VFA was associated with hs-CRP, adiponectin, and the TG/HDL ratio, we failed to demonstrate the independent relationship of these markers with cardiac remodeling.

In 1151 participants who were free of cardiovascular disease, VFA by computed tomography was independently associated with LV concentricity [28]. In patients with severe aortic stenosis, VFA measured by bioelectrical impedance analysis was independently associated with impaired LV GLS; however, no previous study has investigated the association between VFA and LV GLS in a population without cardiovascular diseases. In our study of 229 participants with suspected MetS but no apparent cardiovascular diseases, VFA was associated with LV structural remodeling and both diastolic and systolic dysfunction.

In early stage of LV diastolic dysfunction, the e' velocity declines as ventricular stiffness increases, and LV filling and LA pressure may not be changed yet. As LV diastolic dysfunction progress with aging and cardiometabolic disturbance, LV filling pressure represented by E/e' ratio and LA pressure rises and LA size increases [20]. In this study, VFA_h had lower e' velocity compared to VFA_l, however, E/e' ratio and LA volume were not different between groups who did not have apparent cardiovascular diseases or heart failure.

Risk stratification by visceral fat area in population without cardiovascular diseases

In this study, subjects with suspected MetS were enrolled. The prevalence of hypertension and impaired fasting glucose was 38.4% and 38.9%, respectively. The prevalence of previously diagnosed hypertension and diabetes was only 5.4 and 0.9%, respectively. In this type of a population with low to intermediate cardiovascular risk, it is essential to stratify the high-risk group that may require an early prevention strategy. In our group, the median value of VFA that was used to allocate the groups was 76 cm^2. This value is consistent with the value established in a previous study that was able to predict two or more cardiovascular risk factors in 2870 middle aged Japanese employees who underwent a health check-up [29]. BMI, as an indicator of general obesity, shows a U-shaped association with morbidity and mortality, whereas VFA measured by bioelectrical impedance analysis shows a linear association [30]. In addition, bioelectrical

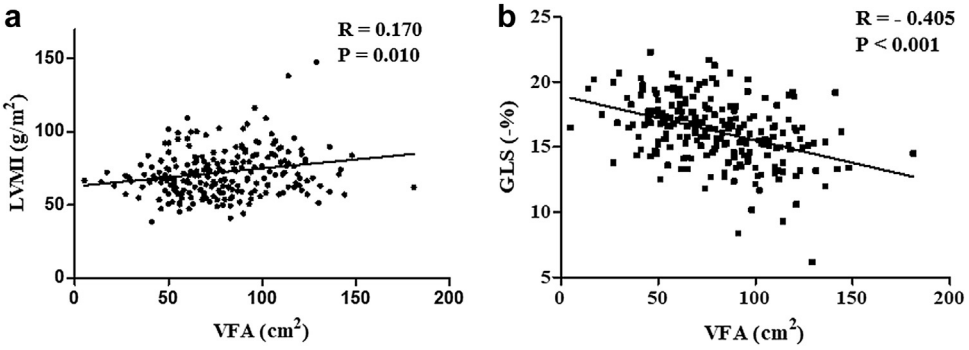

Figure 1 The association of VFA with LV mass index and LV GLS. VFA is significantly correlated with LV mass index (a) and LV GLS (b). VFA; visceral , LV: left ventricular, LVMI; left ventricular mass index, GLS; global longitudinal area.

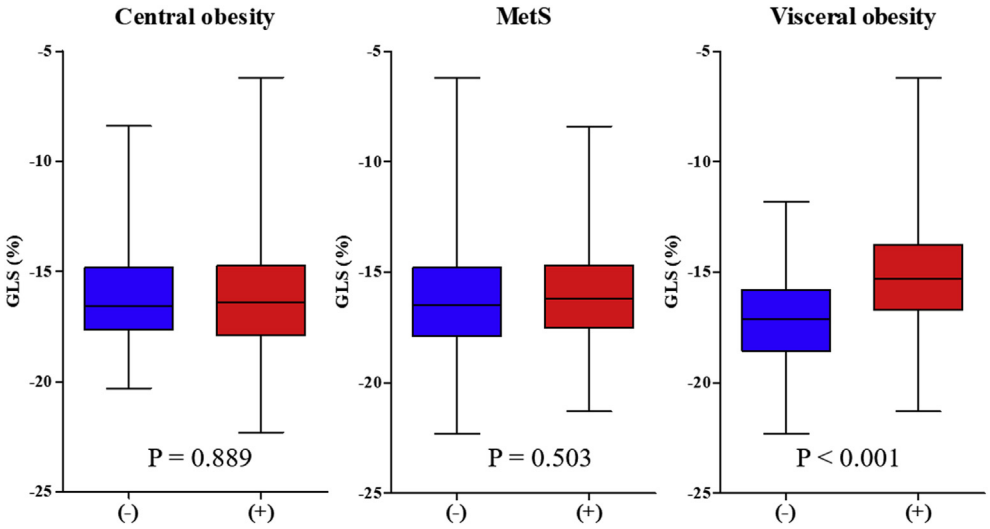

Figure 2 Difference of LV GLS according to the presence of MetS, central obesity defined by WC and visceral obesity defined by VFA. A significant difference in the LV GLS value was observed only in the groups divided by visceral obesity. MetS; metabolic syndrome, VFA; visceral fat area, WC; waist circumference.

Table 4 The impact of VFA on myocardial dysfunction represented by LV GLS using multiple linear regression analysis after adjustment for age, sex, general obesity, waist circumference, hypertension, anti-hypertensive medication, fasting blood glucose, triglyceride, high-density lipoprotein levels, LV mass index and LV EF.

	β	Standardized β	95% CI		p	VIF
Age	−0.014	−0.026	−0.075	0.047	0.654	1.086
Sex	−1.144	−0.237	−1.793	−0.494	0.001	1.517
General obesity	−0.183	−0.038	−0.881	0.515	0.606	1.732
WC	0.004	0.013	−0.052	0.060	0.888	2.609
VFA	−0.019	−0.229	−0.033	−0.006	0.005	2.080
HTN	−0.412	−0.083	−1.024	0.200	0.186	1.276
Anti-hypertensive medication	1.064	0.126	0.079	2.049	0.034	1.152
FBG	0.060	0.012	−0.518	0.638	0.839	1.143
TG	0.000	0.013	−0.003	0.004	0.841	1.270
HDL	0.013	0.073	−0.009	0.035	0.235	1.239
LV mass index	−0.059	−0.188	−0.097	−0.022	0.002	1.181
LV EF	0.091	0.248	0.050	0.132	<0.001	1.031

β, regression coefficient; CI, confidence interval; VIF, variance inflation factor. Other abbreviations are described in Tables 1 and 2

impedance analysis showed the best correlation with VFA by computed tomography when compared with anthropometric measurements, including BMI, WC, and waist-hip ratio [31]. VFA by bioelectrical impedance analysis is also simple, cost-effective, and safe, with no radiation exposure [5]. In the present study, the highest difference in LV GLS was observed only according to the presence of visceral obesity and not MetS or central obesity measured by WC. Therefore, VFA may represent a novel marker to stratify subjects with subclinical and early cardiac remodeling in middle-aged populations with low to intermediate cardiovascular risk.

Limitations

Our study had several considerable limitations. First, we indirectly measured the amount of visceral obesity by dual impedance bioelectrical analysis instead of computed tomography. Although the precise measurement of visceral obesity by computed tomography has several advantages in evaluating the exact amount and distribution of visceral obesity and its localized effect, a good correlation between VFA measured by the two methods has been shown [17], and VFA measurement is a simple and cost-effective method for a general population study [5]. Second, increased LV mass index and impaired LV GLS are early markers of any myocardial disease such as myocardial ischemia, myocardial fibrosis, and heart failure; however, in this study, the association between visceral obesity and clinical cardiovascular events was not evaluated. Finally, this kind of cross-sectional study could not confirm the causality. The Seoul Metabolic Syndrome study is an on-going prospective study scheduled for 10 years of follow-up. Long-term data including newly developed or clinical cardiac dysfunction will be included in future investigations.

Conclusion

Visceral obesity assessed by VFA was well correlated with parameters of MetS. Visceral obesity, but not central obesity measured by WC, was independently associated

with structural and functional cardiac remodeling in subjects with suspected MetS. It suggests that visceral obesity should be considered as an important risk factor for cardiac damage in dysmetabolic subjects.

Conflicts of interest

None.

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Data sharing statement: Data of Seoul Metabolic Syndrome study are not publicly accessible due to Korean personal information projection act. Dataset generated and analyzed during this study are available from the corresponding author on reasonable request.

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