Correlation between Aortic Stiffness and Visceral Fat Determined by Magnetic Resonance Imaging

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Background and Objective: *Arterial stiffening is an independent predictor for cardiovascular events. Studies using a variety of different techniques have shown that visceral fat accumulation may be related to aortic stiffening. However, there are limited data available about the study using magnetic resonance imaging (MRI). The aim of the present study is to assess the correlation of visceral fat to the degree of aortic stiffness as determined by MRI.*

Material and Method: *The present study examined 95 subjects (age 69.14 ± 9.76 years, female 50.5%, waist circumference 93 ± 11 cm) who underwent cardiac MRI examination. Using MRI, aortic stiffness was measured as aortic pulse wave velocity (PWV) by distance divided by time delay between mid-ascending and mid-descending aorta. Body fat measures were evaluated as abdominal visceral fat volume (visceral fat), pericardial fat volume (visceral fat) and abdominal subcutaneous fat volume (subcutaneous fat). Pearson correlation analysis was performed to determine the correlation between aortic stiffness and each measure of the body fat.*

Results: *Mean PWV 11.41 ± 5.30 m/s, pericardial fat 17.37 ± 4.60 ml, abdominal visceral fat 470.85 ± 181.12 ml and abdominal subcutaneous fat 617.57 ± 214.70 ml. No correlation was found between PWV and each measure of body fat as follows; (1) pericardial fat volume to PWV (r = -0.025, p-value = 0.808), (2) abdominal visceral fat volume to PWV (r = 0.068, p-value = 0.520), (3) abdominal subcutaneous fat volume to PWV (r = -0.001, p-value = 0.992), (4) total abdominal fat volume to PWV (r = 0.038, p-value = 0.719), (5) total visceral fat volume to PWV (r = 0.066, p-value = 0.528). There was also no correlation found between PWV and visceral fat grouped in tertiles.*

Conclusion: *There was no significant correlation between visceral fat volumes and aortic stiffness.*

Keywords: *aortic stiffness, pulse wave velocity, visceral fat, magnetic resonance imaging*

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Cardiovascular disease is the leading cause of death in the general population. The pathogenesis is the process of atherosclerosis, but also increasingly linked to other pathogenic process like diffuse arteriosclerosis (stiffening of large arteries)[1].

A decrease in the compliance of the arterial system, termed arterial stiffness, results in increased cardiac workload. In recent years, there has been much interest in the relationship between central arterial stiffness and cardiovascular disease. Its clinical determinants were studied in extensive work. The central arteries stiffness causes hemodynamic alterations that could lead to cardiovascular events. Data from several studies showed that aortic stiffness was independent predictor for all-cause and cardiovascular mortalities, fatal and non-fatal coronary events, and fatal strokes in the general population and patients with essential hypertension[2-4], type 2 diabetes mellitus[5], end-stage renal disease[6,7] and in elderly patients[8,9]. As changes can be detected before clinically apparent vascular disease, arterial stiffness may act as a marker for the development of future atherosclerotic disease.

There are many methods to assess arterial stiffness[1]. Pulse wave velocity (PWV) is a technique that can provides information about the distensibility of the vessel being studied. It is the speed which the forward blood flows along the aorta through the vascular tree. PWV will be increased when the aorta become stiffening or poor compliance. PWV can be measured by various different methods, both invasive and non-invasive. The most commonly use tool is

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ultrasonography. However, this tool has limitation for the evaluation of large arteries, such as the aorta, due to poor acoustic penetration, especially in obese patients. More recently, an alternative method of measuring PWV has been described using a Cardiovascular Magnetic Resonance Imaging (MRI) technique, which can accurately assess aortic stiffness because it provides cross-sectional images of the aorta with high spatial and temporal resolution which is not limited by imaging plane or body size[10].

Arterial stiffening increases with age and is known to increase with certain disease and condition including genetic predisposition, hypertension, diabetes mellitus, hypercholesterolemia end-stage renal disease and inflammation[11]. Some data suggest that body fat distributions are also related to arterial stiffness[12]. There are several studies using a variety of different techniques which have shown that visceral fat accumulation may be related to aortic stiffening. However, there are limited data about the study of aortic stiffness using MRI. Therefore, the aim of the present study was to assess the correlation of visceral fat to the degree of aortic stiffness determined by MRI.

Material and Method

Study population and design

This was a cross-sectional, single center study. Ninety-five subjects were recruited among patients who underwent cardiac MRI at Siriraj Hospital for the detection of myocardial ischemia and viability. Inclusion criteria were: male or female patient age 18 or above who will undergo Cardiac MRI at Siriraj Hospital. Exclusion criteria were as follows: unable to perform MRI due to ferromagnetic prosthesis or claustrophobia; pregnant; aortic aneurysm. The present study protocol was approved by the ethic committee and each subject gave written informed consent to participate. Demographic data included cardiovascular risk factor were obtained. The primary objective was to verify correlation between visceral fat and aortic stiffness. The secondary objective was to identify which part of visceral fat is correlated with aortic stiffness (abdominal visceral fat vs. pericardial fat).

Anthropometric measurements

Body weight and height were measured using standardized equipment. Waist circumference was measured at the site of the midpoint between the lowest rib and the top of iliac crest. Body mass index (BMI) was calculated from the formula: (body weight in kilograms)/(height in meters)^2.

MRI analysis of aortic stiffness

PWV was measured as an indicator of aortic stiffness. Cardiac MRI was performed using a 1.5-T Phillips Achieva XR scanner (Phillips Medical System, Best, the Netherlands). Cardiac axis was located by ECG-triggered non breath hold blackblood single shot sequentive 100 slides. The scanning parameters were: echo time (TE) 20 ms; repetition time (TR) 1,800 ms; refocusing flip angle 90°; slice thickness 8 mm; field of view in x axis (FOVx) 240 to 360 mm; field of view in y axis (FOVy) 250 to 280 mm; typical matrix size 118 x 115; and typical acquired spatial resolution 1.59-1.86 x 2.17-2.43 mm.

PWV was assessed by using velocity encoded MRI (VE-MRI) technique as the through plane flow in the mid ascending and descending aorta at the level of pulmonary trunk with the following scanning parameters: TE 3.6 ms; TR 5.3 ms; Refocusing flip angle 12°; slice thickness 8 mm; FOVx 320 mm; FOVy 270 mm; typical matrix size 160 x 132; typical acquired spatial resolution 2.0 x 2.04 mm; temporal resolution 10 to 12 ms; and velocity encoding 170 cm/s.

Image analysis

Cardiovascular imaging software (Extended workspace) was used to draw the contours of the mid-ascending and -descending aorta. The flow at these 2 levels was obtained in all phases of the cardiac cycle. And then, the corresponding flow time curve was created. The arrival time of pulse wave was measured as the point of interception of linear extrapolation of baseline and steep early systolic stage. From the reconstructed sagittal view corresponding to the same level as VE-MRI image obtained, the centerline was drawn from the level of the mid-ascending aorta to the mid-descending aorta to obtained the aortic path length[10](Fig. 1). The PWV between the mid-ascending and mid-descending aorta was calculated according to the following formula:

$PWV = \Delta x/\Delta y$ (m/s)

where Δx = aortic path length between the mid-ascending and mid-descending aorta

Δy = time delay

A higher pulse wave velocity indicated a stiffer aorta.

Assessment of pericardial fat

Using ECG gated breath-holds with a balanced turbo-field echo MR sequence, pericardial fat was quantified. The scanning parameters are: TE 1.60 msec; TR 3.2 msec; flip-angle 50°; slice thickness 8 mm; field

of view, 400 x 400 mm[13]. Pericardial fat was assessed in the four-chamber view, with the plane of mitral and tricuspid valves as margins. The contours around the pericardial fat were drawn manually at end systole and multiplied by the thickness of the slice to yield a volume (Fig. 2).

Assessment of abdominal fat

Abdominal fats were quantified by a turbo spin echo imaging MR protocol. The abdominal visceral and subcutaneous fat volumes were measured at the level of the intervertebral space between L4 and L5. One transverse image was acquired during one breath hold. The scanning parameters are: TE 11 msec; TR, 168 msec; flip angle, 90°; slice thickness, 10 mm[13] (Fig. 3). The total abdominal fat volume was calculated by sum of the abdominal visceral and subcutaneous fat volume.

The total visceral fat volume was determined by the sum of pericardial fat volume and abdominal visceral fat volume. Parameters of fat volumes were divided by BSA to calculate fat volume index. Intraobserver and interobserver reproducibility of PWV, pericardial fat, abdominal visceral fat and abdominal subcutaneous fat was performed in 15 randomly selected patients.

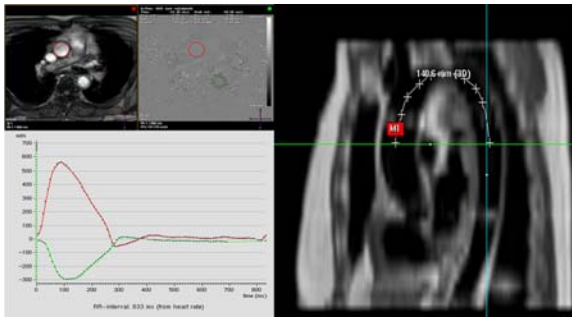

Fig. 1 Assessment of time delay and aortic path length between mid ascending and descending aorta (Left Upper). Velocity-encoded MRI acquired at mid-ascending (dark circles) and mid-descending thoracic aorta (pale circles) (Left Lower). Corresponding measurement of flow at 2 sites (mid-ascending as black line and mid-descending thoracic aorta as dotted line) (Right). Measurement of aortic path length from multiplanar reconstruction

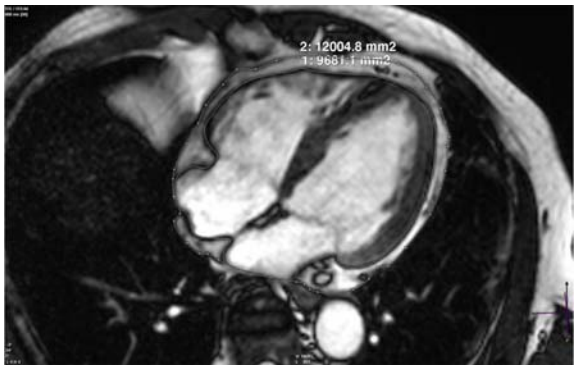

Fig. 2 Measurement of pericardial fat volume in four-chamber view from ECG gated breath-holds with a balanced turbo-field echo MR sequence (1 = area within the visceral pericardium, 2 = area within the parietal pericardium, pericardial fat volume in ml was calculated by the different between area 2 and area 1 multiplied by the thickness of the slice)

Statistical analysis

The statistical analyses were performed by SPSS version 13.0. Continuous data were expressed as mean ± SD and median (Min:Max). Dichotomous data were presented as numbers and percentages. Pearson's correlation coefficient was used to compare 2 continuous variables. The association of normally distributed variables determined with use of the Student t-test to compare mean between two groups and nonnormally distributed variables with use of the Mann-Whiney U-test. Compared for continuous data between multiple groups values by one-way ANOVA and Kruskal-Wallis test. For assessment of agreement within and between observers, a Bland-Altman plot was performed and intraclass correlation coefficients (ICC) were calculated. P-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Patient characteristics are shown in Table 1. There were 95 patients included in this study with mean age 69.14 ± 9.76 years, female 50.5%. Average left ventricular ejection fraction was 64.83 ± 14.75%. Table 2 showed association of PWV with various

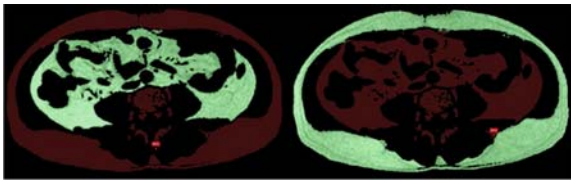

Fig. 3 Measurement of abdominal visceral fat (white color in left panel) and abdominal subcutaneous fat (white color in right panel)

Table 1. Clinical Characteristics of the study patient (n = 95)

Variable	Number (%) or mean ± SD
Age (yrs)	69.14 ± 9.76
Men/Women	47 (49.5)/ 48 (50.5)
Height (cm)	156.94 ± 9.35
Weight (kg)	63.87 ± 11.26
Waist circumference (cm)	92.87 ± 11.36
Body mass index (kg/m^2)	25.92 ± 3.96
Body surface area (m^2)	1.66 ± 0.18
Underlying disease	
Diabetes mellitus	41 (44.6)
Hypertension	72 (79.1)
Coronary artery disease	30 (32.3)
Chronic kidney disease	27 (29.7)
Peripheral arterial disease	8 (8.8)
Medications	
Aspirin	52 (61.2)
Clopidogrel	18 (21.2)
Beta blocker	53 (61.6)
Angiotensin-converting enzyme inhibitor	19 (22.1)
Angiotensin receptor blocker	26 (30.2)
Calcium channel blocker	29 (34.1)
Statin	57 (67.9)
Sulfonylurea	18 (21.2)
Metformin	26 (30.6)
Thiazolidinedione	4 (4.7)

Continuous data represent as mean ± SD. Categorical data represent as number (percent). Coronary artery disease was defined as post myocardial infarction, or positive coronary angiogram or stable angina pectoris

baseline characteristics. Age, history of hypertension, CKD and the use of beta blocker had significant associations with PWV. MRI parameters are shown in Table 3.

Correlation analysis

No correlation was found between PWV and each of body fat measures by MRI. There was also no correlation found between PWV and each measure of fat volume index (Table 4). In addition, the correlations between PWV and all fat volumes grouped in tertiles were also not significant (Table 5).

Reproducibility of PWV measurements

There were good intraobserver and interobserver reproducibility of abdominal subcutaneous fat, pericardial fat and interobserver reproducibility of abdominal visceral fat but not good for intraobserver reproducibility of abdominal visceral fat, and intraobserver and interobserver reproducibility of PWV (Table 6).

Discussion

The present study had 2 major findings. First, with regard to the primary objective of this current study, no correlation was found between visceral fat volumes and aortic stiffness measured as aortic pulse wave velocity determined by MRI. Second, there was statistically significant coefficient correlation between aortic stiffness and aging, hypertension and chronic kidney disease which supports previous reports in the literature emphasizing the correlation between aortic stiffness and these variables.

There were potential explanations for our findings that no correlation was observed with visceral fat. Previous studies that show the positive correlation of aortic stiffness and visceral fat used a different technique from our study for assessment. Sutton-Tyrrell et al[11] evaluated aortic stiffness by PWV measured from simultaneous Doppler flow signals obtained from the right carotid and right femoral arteries by use of nondirectional transcutaneous Doppler flow probes. And body fat measures were evaluated with computed tomography. Another study from Resnick et al[14] used MRI for evaluate visceral fat and aortic stiffness but with a different technique by assessing aortic distensibility while the present study measured PWV as an indicator of aortic stiffness.

The patient characteristic in these studies were also different. The authors participants were Asian only with mean age 69.14 ± 9.76 years. Participants of previous study from Sutton-Tyrrell et al[11] were 40% black and older (mean age 74 years). And the present study from Resnick et al[14] included only an essential hypertension population which might increase possibility of aortic stiffening.

The reason that the present study showed no correlation between PWV and visceral fat may be due to 2 main reasons; first, many of these patients were on medications that could have influenced PWV and second, the technique for measurement of PWV by MRI may require experience since the intraobserver and interobserver agreement of this technique was not very good in the present study.

The present study had some potential limitations. First, the authors studied only patients who underwent cardiac MRI for the detection of myocardial

Table 2. PWV of Clinical Characteristics of the study patient (n = 95)

Variable	PWV					p-value
	n	Mean	SD	Median	Min:Max	
Age (yrs)						
<66	29	9.88	5.03	7.82	5.10:27.77	0.038*
66-73	33	11.75	5.10	10.54	5.47:28.78	
>73	31	12.48	5.58	11.23	3.92:24.23	
Men/Women						
Male	45	11.55	5.08	10.54	5.47:27.77	0.801
Female	48	11.27	5.55	10.15	3.92:28.78	
Height (cm)						
< 151	28	12.05	5.86	10.80	3.92:28.78	0.211
151-160	39	10.28	4.45	8.74	5.10:24.23	
> 160	26	12.41	5.72	10.42	5.57:27.77	
Weight (kg)						
< 58	26	11.95	6.37	10.11	5.76:28.78	0.988*
58-68	38	11.00	4.39	10.56	3.92:22.29	
> 68	29	11.46	5.48	10.25	4.38:27.77	
Waist circumference (cm)						
< 90	28	11.14	5.02	9.58	5.10:22.07	0.939*
90-96	32	11.17	4.71	10.40	5.53:27.77	
>96	29	11.77	6.31	10.97	3.92:28.78	
Body mass index (kg/m^2)						
≤ 23	21	12.43	5.58	10.74	6.39:22.07	0.451*
23.1-27	33	10.07	3.50	9.99	5.10:17.78	
> 27	37	12.07	6.39	10.97	3.92:28.78	
Body surface area (m^2)						
≤ 1.5	11	10.33	5.52	8.49	5.86:21.37	0.757
1.51-1.7	46	11.44	5.49	10.40	3.92:28.78	
> 1.7	34	11.73	5.25	11.08	4.38:27.77	
Underlying disease						
Diabetes mellitus						
No	50	10.61	5.17	9.58	3.92:27.77	0.077
Yes	40	12.62	5.43	11.08	5.47:28.78	
Hypertension						
No	19	9.17	2.33	9.10	5.75:15.12	0.001
Yes	70	12.21	5.76	10.98	3.92:28.78	
Coronary artery disease						
No	62	10.68	4.59	10.11	3.92:24.23	0.076
Yes	29	13.11	6.46	11.23	5.53:28.78	
Chronic kidney disease						
No	62	10.44	4.27	10.04	3.92:24.23	0.012
Yes	27	14.12	6.68	13.31	4.38:28.78	
Peripheral arterial disease						
None	81	11.36	5.38	10.26	3.92:28.78	0.278
Yes	8	13.53	5.04	10.86	8.63:21.05	
Ejection Fraction (%)						
< 59	17	11.74	4.09	10.86	6.75:20.24	0.921
59-72	18	12.21	5.94	11.21	5.47:28.78	
> 72	17	11.51	5.41	10.54	4.38:24.23	
Medications						
Aspirin						
No	33	12.19	5.50	10.57	3.92:24.23	0.463
Yes	51	11.30	5.28	10.74	4.38:28.78	

Table 2. Cont.

Variable	PWV					p-value
	n	Mean	SD	Median	Min:Max	
Clopidogrel						
None	66	11.23	5.30	10.15	3.92:28.78	0.172
Yes	18	13.18	5.44	11.28	5.76:27.77	
Beta blocker						
None	32	10.33	5.39	8.56	4.38:27.77	0.023#
Yes	53	12.35	5.23	11.20	3.92:28.78	
Calcium channel blocker						
None	56	11.26	5.23	10.26	3.92:27.77	0.356
Yes	28	12.42	5.61	11.35	5.10:28.78	
ACEI or ARB						
No	44	11.52	6.13	9.79	3.92:28.78	0.463#
Yes	42	11.59	4.41	10.92	5.47:22.07	
Statin						
No	27	12.54	5.23	11.27	5.86:24.23	0.308
Yes	56	11.24	5.46	10.56	3.92:28.78	
Sulfonylurea						
None	67	11.59	5.62	10.57	3.92:28.78	0.834
Yes	17	11.89	4.27	10.86	6.75:22.07	
Metformin						
None	59	10.98	5.12	10.17	3.92:27.77	0.078
Yes	25	13.23	5.67	11.22	5.47:28.78	
Thiazolidinedione						
None	81	11.61	5.43	10.54	3.92:28.78	0.739
Yes	3	12.67	2.72	11.22	10.97:15.81	

ACEI = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blocker. # Mann Whitney U-test, * Kruskal-Wallis test

Table 3. Cardiac MRI parameters

Parameters	Mean ± SD
Pulse wave velocity (m/s)	11.41 ± 5.30
Pericardial fat (ml)	17.37 ± 4.60
Abdominal visceral fat (ml)	470.85 ± 181.12
Abdominal subcutaneous fat (ml)	617.57 ± 214.70
Total abdominal fat (ml)	1,088.42 ± 317.48
Total visceral fat (ml)	488.22 ± 182.87

ischemia and viability, not randomly selected from the general population. These patients may have had some predisposing conditions, particularly cardiac risk factors. Because of the cross-sectional design, it is hard to generalize to other patient group. Second, many patients in our study received medication that might have effects on structure and function of the arterial walls, included angiotensin-converting enzyme inhibitors[15], angiotensin receptor blockers[16], beta-blockers[17], statins[18]. Some of these medications were not on hold before testing; therefore, there could be effect on the difference of aortic stiffness in patients who received these medications and who were not receiving these agents.

The present study, however, had some strength. First, we used PWV by MRI technique for the evaluation of aortic stiffness. PWV is a well-accepted index of arterial stiffness with high reproducibility, a without the assumption of central pressure[19]. Second, the authors used MRI to assess visceral fat volume. MRI is considered a reproducible technique for fat quantification. A single MRI scan of abdomen is sufficient to distinguish and quantify visceral versus subcutaneous fat without the accompanying radiation of CT scanning.

Perspectives

Previous studies have shown that visceral fat

Table 4. Pearson's correlation of PWV and visceral and subcutaneous fat

Variables	Correlation coefficient	p-value
Pericardial fat volume to PWV	- 0.025	0.808
Abdominal visceral fat volume to PWV	0.068	0.520
Abdominal SC fat volume to PWV	- 0.001	0.992
Total abdominal fat volume to PWV	0.038	0.719
Total visceral fat volume to PWV	0.066	0.528
Pericardial fat volume index to PWV	- 0.026	0.802
Abdominal visceral fat volume index to PWV	0.104	0.323
Abdominal SC fat volume index to PWV	- 0.018	0.865
Total abdominal fat volume index to PWV	0.045	0.669
Total visceral fat volume index to PWV	0.102	0.331

SC = subcutaneous, PWV = pulse wave velocity

Table 5. Correlation of PWV and fat volume grouped in tertiles

Variable	PWV					p-value
	n	Mean	SD	Median	Min:Max	
Pericardial fat						0.426*
Tertile 1	30	12.00	6.13	10.43	4.38:28.78	
Tertile 2	32	10.82	5.57	9.63	3.92:27.77	
Tertile 3	30	11.54	4.16	10.91	5.76:22.32	
Abdominal visceral fat						0.65
Tertile 1	30	10.68	4.87	8.83	5.76:22.07	
Tertile 2	33	11.62	5.26	10.84	5.10:27.77	
Tertile 3	30	11.90	5.83	10.92	3.92:28.78	
Abdominal SC fat						0.467*
Tertile 1	30	12.14	5.72	10.80	5.47:28.78	
Tertile 2	32	10.51	5.41	8.95	4.38:27.77	
Tertile 3	31	11.63	4.78	11.20	3.92:24.33	
Total abdominal fat						0.547*
Tertile 1	30	10.92	4.99	8.90	5.47:22.07	
Tertile 2	32	11.68	6.15	10.40	5.10:28.78	
Tertile 3	31	11.60	4.75	11.20	3.92:24.23	
Total visceral fat						0.591*
Tertile 1	30	10.56	4.83	8.49	5.76:22.07	
Tertile 2	32	11.70	5.30	10.88	5.10:27.77	
Tertile 3	30	11.97	5.80	10.92	3.92:28.78	

SC = subcutaneous, PWV = pulse wave velocity, * Kruskal-Wallis test

is associated with increased central arterial stiffness. The present study does not confirm this association, but it is difficult to conclude from this finding alone that visceral fat volumes have no correlation with aortic stiffness. PWV determined by MRI is a new technique, and a larger patient population needs to be evaluated by using this method including such patients as those with metabolic syndrome who have abnormal distribution of visceral fat, in order to demonstrate the application of this method.

As aortic stiffness can be detected before the appearance of clinically apparent cardiovascular disease, it is likely that, in the future, measurement of arterial stiffness will become an increasingly important

Table 6. Bland-Altman analysis and intraclass correlation coefficients (ICC) of the intraobserver and interobserver agreement of PWV, pericardial fat, abdominal visceral fat and abdominal subcutaneous fat

Variables	Bias	Lower limit of agreement	Upper limit of agreement	ICC
Intraobserver-PWV	1.6	-9.4	12.6	0.599
Interobserver-PWV	0.9	-10.0	11.9	0.634
Intraobserver-pericardial fat	-0.3	-5.6	5.0	0.910
Interobserver-pericardial fat	-0.6	-6.0	4.7	0.904
Intraobserver-abdominal visceral fat	-39.3	-299.9	221.3	0.573
Interobserver-abdominal visceral fat	7.1	-53.2	67.4	0.974
Intraobserver-abdominal SC fat	-8.6	-73.5	56.2	0.988
Interobserver-abdominal SC fat	-1.9	-42.4	38.6	0.995

SC = subcutaneous, PWV = pulse wave velocity

part of the cardiovascular risk assessment and may possibly also improve the monitoring of therapy.

Conclusion

The authors cannot demonstrate the significant correlation between visceral fat volumes and aortic stiffness measured as aortic pulse wave velocity determined by MRI in the present study.

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Potential conflicts of interest

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การศึกษาความสัมพันธ์ระหว่างปริมาณไขมันในอวัยวะภายในกับภาวะหลอดเลือดแดงใหญ่แข็งโดยการตรวจด้วยคลื่นแม่เหล็กไฟฟ้า

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วัตถุประสงค์: *ภาวะหลอดเลือดแดงใหญ่แข็งเป็นสิ่งบ่งชี้อย่างหนึ่งของการเกิดโรคหัวใจและหลอดเลือดในอนาคต โดยจะเกิดมากขึ้นตามอายุที่เพิ่มขึ้น มีข้อมูลจากการศึกษาด้วยคลื่นเสียงสะท้อน และเอกซเรย์คอมพิวเตอร์ว่าการกระจายตัวของไขมันในร่างกายก็มีผลต่อภาวะหลอดเลือดแดงใหญ่แข็งด้วยเช่นกัน การศึกษานี้มีวัตถุประสงค์เพื่อหาความสัมพันธ์ ระหว่างปริมาณไขมันในอวัยวะภายในกับภาวะหลอดเลือดแดงใหญ่แข็งโดยวิธีการตรวจด้วยคลื่นแม่เหล็กไฟฟ้า*

วัสดุและวิธีการ: *เป็นการศึกษาแบบ cross-sectional study คัดเลือกผู้เข้าร่วมการศึกษาจากผู้ที่มารับการตรวจหัวใจด้วยคลื่นแม่เหล็กไฟฟ้าที่โรงพยาบาลศิริราช ช่วงระยะเวลาตั้งแต่ ตุลาคม พ.ศ. 2553 ถึง กุมภาพันธ์ พ.ศ. 2554 ผู้เข้าร่วมการศึกษาจะได้รับการตรวจหัวใจด้วยคลื่นแม่เหล็กไฟฟ้า และประเมินภาวะหลอดเลือดแดงใหญ่แข็งด้วยวิธีวัด pulse wave velocity (PWV), การตรวจปริมาณไขมันรอบหัวใจด้วย และการตรวจปริมาณไขมันในช่องท้องด้วยคลื่น แม่เหล็กไฟฟ้าแล้วหาความสัมพันธ์ระหว่างปริมาณไขมันในอวัยวะภายใน กับภาวะหลอดเลือดแดงใหญ่แข็ง*

ผลการศึกษา: *ผู้ป่วยเข้าร่วมการศึกษา 95 คน มีอายุเฉลี่ย 69.14 $\pm$ 9.76 ปี, เพศหญิงร้อยละ 50.5, เส้นรอบเอว 93 $\pm$ 11 เซนติเมตร, ค่าเฉลี่ยของ PWV 11.41 $\pm$ 5.30 เมตรต่อนาที, ปริมาณไขมันรอบหัวใจ 17.37 $\pm$ 4.60 มิลลิลิตร, ปริมาณไขมันในช่องท้อง 470.85 $\pm$ 181.12 มิลลิลิตร, ปริมาณไขมันใต้ผิวหนัง 617.57 $\pm$ 214.70 มิลลิลิตร ไม่พบความสัมพันธ์อย่างมีนัยสำคัญระหว่างปริมาณไขมันในอวัยวะ ในตำแหน่งต่าง ๆ กับภาวะหลอดเลือดแดงใหญ่แข็งทั้งไขมันรอบหัวใจ ($r = -0.025$, p-value = 0.808), ไขมันในช่องท้อง ($r = 0.068$, p-value = 0.520) และไขมันใต้ผิวหนัง ($r = -0.001$, p-value = 0.992) ไม่พบความสัมพันธ์อย่างมีนัยสำคัญระหว่างปริมาณไขมัน ในอวัยวะภายในกับภาวะหลอดเลือดแดงใหญ่แข็งเมื่อแบ่งระดับปริมาณไขมันในอวัยวะภายในเป็น 3 ระดับเช่นกัน*

สรุป: *ไม่พบความสัมพันธ์ระหว่างปริมาณไขมันในอวัยวะภายในกับภาวะหลอดเลือดแดงใหญ่แข็งโดยการตรวจด้วยคลื่นแม่เหล็กไฟฟ้าในการศึกษานี้*