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ORIGINAL SCIENTIFIC PAPER

Evaluation of the relationship between para-aortic adipose tissue and ascending aortic diameter using a new method

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ABSTRACT

Background: Para-aortic adipose tissue (PAT) is the local adipose tissue that externally surrounds the aorta. It contributes significantly to aortic atherosclerosis and enlargement. Studies conducted with computed tomography and magnetic resonance have shown that individuals with aortic aneurysm had more PAT than healthy individuals. In this study, we measured PAT using transthoracic echocardiography (TTE). The aim of this study is to investigate the possible relationship of TTE measured PAT with ascending aortic width.

Methods: PAT was defined as the hypoechoic space in front of ascending aortic 2 cm above the sinotubular junction at the end of the systole. Patients were divided into 2 groups according to the presence of dilatation in the ascending aorta using Roman's classification (aortic size index, ASI). ASI of less than 21 was considered no aortic dilation and an ASI of 21 mm/m2 or greater was considered to have aortic dilation.

Results: A total of 321 unselected patients were divided into the ascending aortic dilatation (AAD) group ($n = 96$) and the normal ascending aorta diameter group ($n = 225$ patients). PAT was significantly higher in the AAD group compared with the non-ADD group (0.9 (0.48) vs. 0.7 (0.91) mm, $p < 0.0001$). Univariate and multivariate logistic regression analysis revealed that PAT (OR: 3.005, 95%CI (1.445–6.251)) were significantly associated with AAD.

Conclusions: This is the first study which evaluated PAT measured by TTE. We found a significant association between PAT measured by TTE and ascending aorta width.

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KEYWORDS
Para-aortic adipose tissue; local adipose tissue; ascending aortic dilatation; aortic size index; aortic aneurysm; transthoracic echocardiography

Introduction

Ascending aortic aneurysm is characterised by a larger than normal ascending aorta and is associated with increased mortality [1]. Systemic factors, such as atherosclerosis triggered by inflammation, and local factors, such as deficiencies of structural proteins, e.g. elastin and collagen in the aortic wall play a role in the pathogenesis of ascending aortic dilatation (AAD) [1–4]. Para-aortic adipose tissue (PAT) is the local adipose tissue surrounding the aorta. In addition to protecting the aorta against trauma, it has been hypothesised that PAT contributes significantly to aortic atherosclerosis and enlargement *via* its secretion of bioactive molecules such as adiponectin and growth factors [5]. The fact that PAT had been found to be associated with aortic calcification and peripheral artery disease supports this hypothesis [6,7]. Perivascular adipose tissue has been found to be an important predictor of abdominal aortic aneurysm [8]. A study conducted using computed tomography (CT) demonstrated that the density of the perivascular adipose tissue around the aneurysm sac was higher in individuals with abdominal aortic aneurysm than in healthy individuals [9]. PAT has also been evaluated using magnetic resonance imaging (MRI) or CT in other studies. However, to our knowledge there are no studies in the literature examining measurement of para-aortic adipose tissue using transthoracic echocardiography (TTE), which is in fact an easier and more accessible method. Therefore, the aim of this study was to measure PAT using TTE and to investigate the relationship between the thickness of the PAT and the width of the ascending aorta.

Material and method

The study included all patients over the age of 18 who applied to the cardiology outpatient clinic of our

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hospital and underwent TTE. Patients who had undergone previous cardiac surgery, e.g. coronary artery bypass surgery and heart valve replacement, severe aortic valve regurgitation and stenosis, patients with bicuspid and rheumatic aortic valve disease, Marfan syndrome, Ehlers-Danlos syndrome, familial thoracic aortic aneurysm syndrome, Turner syndrome, other connective tissue disorders, infectious diseases, restrictive and hypertrophic cardiomyopathy, renal failure requiring dialysis, malignancy or patients that were pregnant were excluded from this study (Figure 1). Roman's classification (aortic size index (ASI)), a body surface area (BSA)-adjusted classification, was used to diagnose AAD [10]. Accordingly, ASI was calculated using the following formula of ASI = Ascending aortic diameter (mm)/Body surface area (m^2)' [11], and ASI values of $\geq$ 21 mm/m^2 were deemed to indicate AAD [11].]

Patients were categorised into two groups according to ASI. The AAD group consisted of patients with ASI of 21 mm/m^2 or greater and the non-AAD group patients ASI lower than21mm/m^2. Gender, age, body mass index (BMI), body surface area, and histories of coronary artery disease and diabetes mellitus were recorded. BMI was calculated using the following formula: 'BMI = weight (kg)/height^2 (m). Body surface area (BSA) was calculated using the formula of BSA (m^2) = ([Height (cm) × Weight (kg)]/3600)$^{1/2}$'. This study was approved by the institutional ethics committee and conducted in accordance with the principles set out in the Declaration of Helsinki.

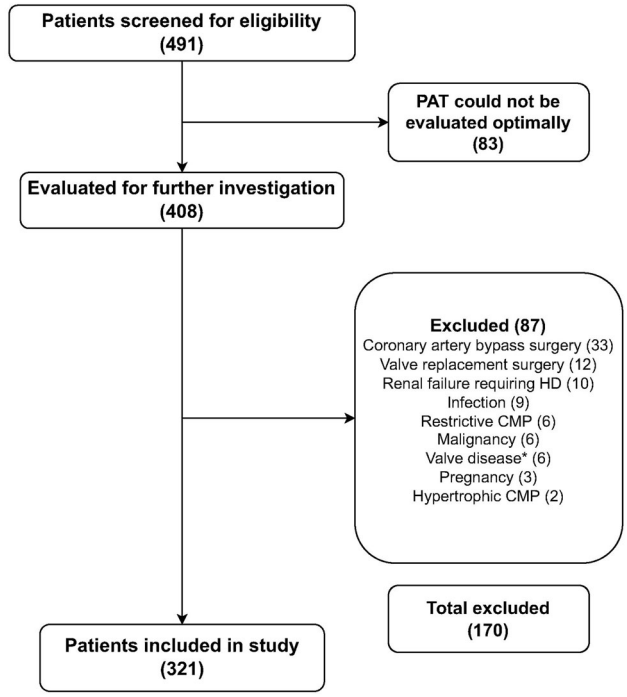

Figure 1. Flow-chart of patient selection.

Measurement of Para-aortic adipose tissue

Transthoracic echocardiography (TTE) was carried out using a Philips Epiq 7c S5-1 Probe system. Each patient underwent two-dimensional TTE as per the recommendations of the European Association of Echocardiography [12]. The parasternal long axis view was used to view the proximal AAD. The distance between leading edge to leading edge of the aortic lumen perpendicular to the long axis 2 cm above the sinotubular junction at the end of the diastole in views showing the largest aortic diameter was used to determine AAD[13]. Additionally, we defined PAT as the hypoechoic space in front of the ascending aorta 2 cm above the sinotubular junction at the end of the systole (Figure 2). Values were measured in three cardiac cycles and the mean of these measurements were taken into consideration. There was perfect interobserver harmony between the two operators who measure PAT. Intraclass correlation coefficient (ICC) of PAT measurement was calculated as 0.917 (95% Confidence Interval (CI), 0.724–0.977). A radiologist performed tissue analysis with computed tomography imaging whether the hypodense space in front of ascending aortic 2 cm above the sinotubular junction is adipose tissue. The mean attenuation (Hounsfield Units, HU) of adipose tissue within PAT was noted with adipose tissue thresholds set at −190 to −30 HU [13]. The mean Hounsfield Units (HU) of PAT was found to be −70.16 HU (Minimum −93 HU, Maximum -46HU) (Figure 3).

Statistical analysis

Data were analysed using the SPSS 23 (IBM Statistical Package for Social Sciences version 23) software package. Categorical variables were expressed using frequency distributions and numerical variables using descriptive statistics (mean ± standard deviation). The Kolmogorov–Smirnov test was used to determine whether the data conformed to the normal distribution. Subsequently, a parametric test was used to analyse normally distributed data and data that did not confirm to the normal distribution were analysed using a nonparametric test. The independent samples *t*-test and Mann–Whitney *U* test were used to check whether there was a difference between the measurements of the two independent groups. Additionally, multivariate logistic regression analyses were conducted to assess the relationship between PAT and AAD. In multivariate regression models, the effect size was adjusted for variables

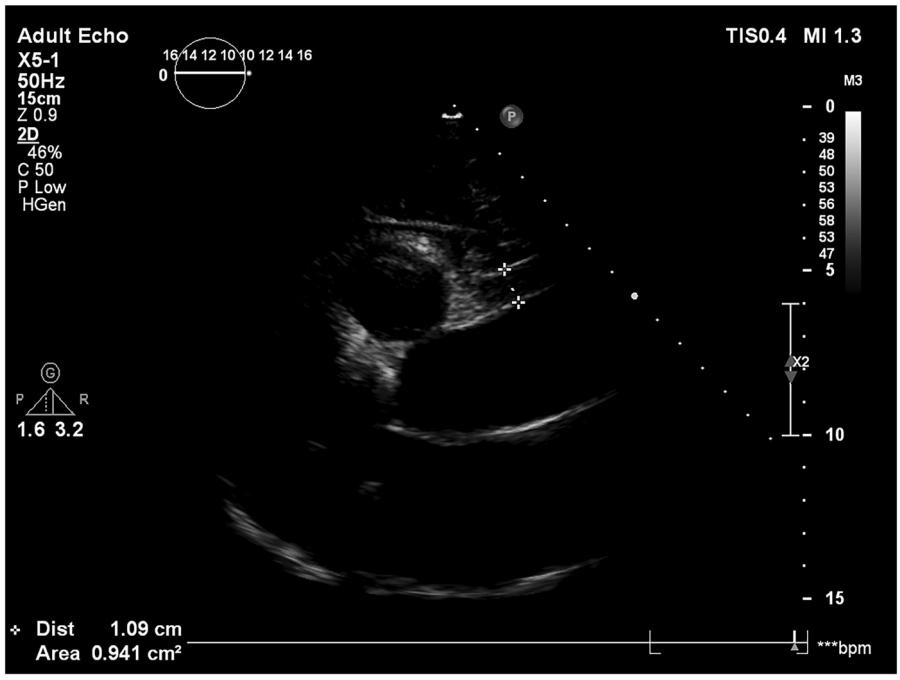

Figure 2. Para-aortic adipose tissue measurement with transthoracic echocardiography.

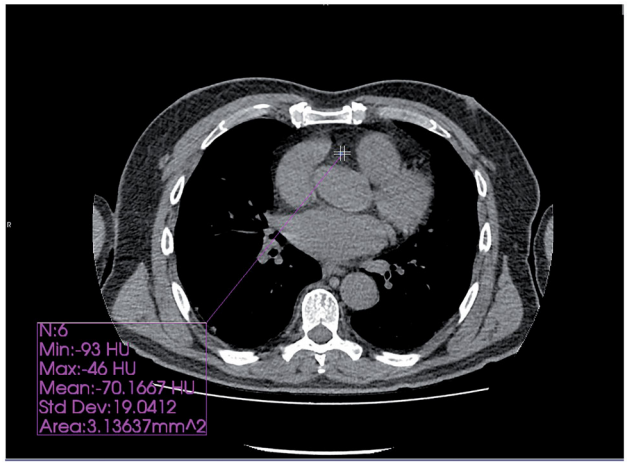

Figure 3. Confirmation of para-aortic tissue as adipose tissue by computed tomography.

with a significance level ≤ 0.10 in the univariate analysis. Adjusted odds ratios (ORs) and their corresponding confidence intervals (CI) were given. 2-tailed probability (p) values of$<$ 0.05 were considered statistically significant.ICC was used to determine the in-class reliability of PAT measurement (95% CI).

Results

A total of 491 patients were screened within the scope of this study. Of these, 83 patients were excluded as a PAT image of sufficient quality could not be obtained using TTE. An additional 87 patients were excluded from the study based on one or more of the other exclusion criteria. Thus, the study included a total of 321 patients (Figure 1). Patients were categorised into two groups: the AAD group ($n = 96$) and the non-AAD group($n = 225$). Baseline characteristics and echocardiographic and biochemical parameters are given in Tables 1 and 2. The average age of patients with AAD was greater than those without (64.7 ± 11.7 vs. 57.3 ± 13.1 years, $p < 0.001$) and a higher percentage were female (69% vs. 54%, $p = 0.019$). BMI and BSA values of patients with AAD were lower than those of patients without AAD ($p < 0.001$) (Table 1). Additionally, PAT was significantly higher in the patients with AAD as compared to patients without AAD (0.9 [0.48] vs. 0.7 [0.91]cm, $p < 0.0001$). Biochemical analyses revealed that the estimated glomerular filtration rates(eGFR)of patients with AAD were lower than those of the patients without AAD (89[15.3]mL/min vs. 95 [20] mL/min, $p < 0.0001$).

Pearson correlation analysis revealed a weakly significant linear relationship in the positive direction between PAT and the echocardiography parameters of left ventricular end-diastolic width ($r = 0.110$; $p < 0.05$), left ventricular end-systolic width ($r = 0.152$; $p < 0.01$), and left atrial width ($r = 0.143$; $p < 0.05$). Additionally, a moderately significant linear relationship in the positive direction was found between PAT and aortic diastolic width ($r = 0{,}369$; $p < 0.001$), aortic systolic width ($r = 0.360$; $p < 0.001$), and waist circumference ($r = 0.363$; $p < 0.001$). Lastly, a weekly significant linear

relationship in the negative direction was found between PAT and the blood parameters of total cholesterol (r=-0.142; $p < 0.05$) and triglyceride (r=-0.118; $p < 0.05$).

Furthermore, univariate, and multivariate logistic regression analyses revealed that age (OR: 1.028, 95%CI [1.002–1.054]), BMI (OR: 0.885, 95%CI [0.827–0.947[), gender (OR:0.381, 95%CI [0.207–0.700]), left ventricular mass index (OR: 1.026, 95%CI [1.008–1.044]), and PAT (OR: 3.005, 95%CI [1.445–6.251]) were associated with AAD Tables 3 and 4. ROC curve analysis was performed to predict AAD (Table 5). PAT 8 mm had 66% sensitivity and 57% specificity [AUC = 0.642, $p < 0.000$, 95% CI (0.555–0.692)] to predict the presence of AAD.

Table 1. Baseline characteristics of the study groups with and without ascending aortic dilatation.

	Aortic Dilatation		
	No ($N = 225$)	Yes ($N = 96$)	p Value
Age (year)	57.3 ± 13.1	64.7 ± 11.7	<0.000
Weight (kg)	80 (17)	67 (17)	<0.000
Height (cm)	165 (11)	159 (12)	<0.000
Body surface area (m^2)	1.9 (0.24)	1.7 (0.22)	<0.000
Body mass index (kg/m^2)	29 (6.1)	27.2 (6.6)	<0.000
Obesity, (n, %)	102 (45.3)	28 (29.2)	0.006
Paraaortic adipose tissue, mm	0.7 (0.91)	0.91 (0.48)	<0.000
Female gender (n, %)	122 (54.2)	66 (68.8)	0.019
Hypertension (n, %)	148 (65.8)	66 (68.8)	0.698
Diabetes mellitus (n, %)	66 (29.3)	28 (29.2)	1.000
Coronary artery disease (n, %)	29 (12.9)	14 (14.6)	0.721
Hyperlipidemia (n, %)	26 (11.6)	9 (9.4)	0.697
Smoking (n, %)	46 (20.4)	13 (13.5)	0.159
RAS inhibitors (n, %)	112 (49.8)	51 (53.1)	0.627
Statins (n, %)	27 (12)	14 (14.6)	0.584
Calcium channel blockers (n, %)	19 (8.4)	11 (11.5)	0.407
Beta blockers (n, %)	49 (21.8)	24 (25)	0.562
Acetylsalicylic acid (n, %)	52 (23.1)	27 (28.1)	0.396
Left ventricular Hypertrophy (n, %)	36 (16)	38 (39.6)	0.000

Continuous variables are normally distributed showed Mean ± standard deviation; continuous variables are not normally distributed showed as median (interquartile range); categorical variables are presented as number (percentage).

Discussion

Untreated and unmonitored aortic aneurysm may result in aortic dissection leading to mortality. The aetiology of aortic dilatation is multifactorial such as hypertension, infections, genetic factors, Marfan syndrome, bicuspid aortic valve, Ehler-danlos syndrome and idiopathic conditions [14]. In this context, PAT, as a paracrine organ, also may play an important role in the aetiology of aortic dilatation through the cytokines it secretes [15], which has been reported to result in more aortic function, width, and atherosclerosis [16,17]. PAT measurement can be used safely in the follow-up of aortic dilatation [18]. Various studies in the literature have discussed the measurement of PAT using CT and MRI. PAT measurement has not received the attention it deserves in clinical practice since measurements are predominately performed using CT and MRI, which are commonly requested for other indications, However, such methods require the use of special software and requires expensive equipment and time to perform. Conversely, TTE is available in almost every healthcare facility around the globe. We found that PAT measured using TTE is an important predictor of ascending aortic width.

Table 2. Laboratory and echocardiographic findings.

	Aortic Dilatation		
	No ($N = 225$)	Yes ($N = 96$)	p Value
Urea (mg/dl)	30 (14)	32 (16)	0.115
Creatinine (mg/dL)	0.8 (0.3)	0.84 (0.3)	0.232
Aspartate Aminotransferase (U/L)	19 (8)	19 (7)	0.740
Alanine Aminotransferase (U/L)	17.5 (10)	16 (8)	0.112
Glomerular filtration rate ($mL/min/1.73m^2$)	95(20)	89 (15.3)	<0.000
Total cholesterol (mg/dL)	188 (50)	190 (47)	0.522
Triglyceride (mg/dL)	135 (81)	132 (84)	0.821
Low-density lipoprotein (mg/dL)	110 (47)	114 (45)	0.465
High-density lipoprotein (mg/dL)	45 (15)	43 (11)	0.571
Glucose (mg/dL)	103 (29)	106 (26)	0.294
Left atrial diameter (mm)	36 (1)	36 (1)	0.030
Left ventricular end-diastolic diameter (mm)	46 (6)	47 (4)	0.630
Left ventricular end-systolic diameter (mm)	28 (5)	28 (5)	0.481
Interventricular septal thickness (mm)	10 (0)	10 (1)	0.017
Posterior wall thickness (mm)	10 (0)	10 (0)	0.166
E (cm/sn)	70 (10)	70 (10)	0.726
A (cm/sn)	80 (15)	83 (10)	0.063
Left ventricular ejection fraction (%)	65 (7.6)	64 (7.2)	0.512
Left ventricular mass index (gr/m^2)	82 (20)	93.5 (22)	<0.000
Left ventricular hypertrophy (n, %)	36 (16)	38 (39.6)	<0.000
Normal left ventricular geometry (n, %)	68 (30.2)	16 (16.7)	0.012
Concentric remodelling (n, %)	121 (53.8)	42 (43.8)	0.113
Concentric hypertrophy (n, %)	13 (5.8)	17 (17.7)	0.001
Eccentric hypertrophy (n, %)	23 (10.2)	21 (21.9)	0.008

Continuous variables are presented as median (interquartile range).

Table 3. Univariate analysis for ascending aortic dilatation.

	β	*p* Value
Age (year)	0.046	<0.0001
Body mass index (kg/m^2)	−0.098	<0.0001
Female gender (%)	−0.619	0.016
Obesity (%)	−0.700	0.007
Para aortic adipose tissue, mm	1.020	0.001
Hypertension (%)	0.135	0.605
Diabetes mellitus (%)	−0.008	0.976
Hyperlipidemia (%)	−0.233	0.567
Smoking (%)	−0.495	0.147
Medications (%)		
Angiotensin converting enzyme inhibitor	0.134	0.583
Calcium channel blocker	0.339	0.397
İnsulin	−0.397	0.449
Oral antidiabetic	−0.028	0.933
Creatinine (mg/dL)	0.114	0.518
Glomerular filtration rate (mL/min/1.73m^2)	−0.011	0.030
Total cholesterol (mg/dL)	0.000	0.973
Triglyceride (mg/dL)	0.000	0.829
Low density lipoprotein (mg/dL)	0.001	0.820
High density lipoprotein (mg/dL)	0.007	0.564
Glucose (mg/dL)	0.001	0.506
Left atrial diameter (mm)	0.081	0.025
LV ejection fraction (%)	−0.007	0.714
LV mass index (gr/m^2)	0.040	<0.000
E (cm/sn)	0.000	0.972
A (cm/sn)	0.014	0.078
Left ventricle hypertrophy (%)	1.235	<0.000
LV geometry (%)		
Normal	−0.773	0.013
Concentric remodelling	−0.403	0.101
Eccentric hypertrophy	0.900	0.007
Concentric hypertrophy	1.255	0.001

SBP: indies systolic blood pressure; DBP: diastolic blood pressure; LV: left ventricle; β; Regression coefficient.

Table 4. Multivariate analysis for ascending aortic dilatation.

		95% CI		
	β	OR	Lower	Upper
Age	0.027	**1.028**	1.002	1.054
A	0.013	1.013	0.996	1.031
BMI	−0.122	**0.885**	0.827	0.947
Gender	−0.965	**0.381**	0.207	0.700
Para aortic adipose tissue	1.100	**3.005**	1.445	6.251
Left atrial diameter	0.033	1.034	0.931	1.148
Left Ventricular Mass Index	0.026	**1.026**	1.008	1.044
Glomerular filtration rate	−0.003	0.997	0.986	1.007

CI: indicates confidence interval; OR: Odds ratio; β: Regression coefficient.

Table 5. ROC Curve parameters for ascending aortic dilatation.

Risk factor	AUC (95%)	Cut off value	*p* value	Sensitivity	Specificity
PAT	0.64 (0.555-692)	8 mm	< 0.000	66	57

PAT: Para aortic adipose tissue, AUC: Area under curve, CI: Confidence interval.

In the present study, an association was found between PAT thickness measured by TTE and coronary artery disease, hypertension, diabetes mellitus, and chronic kidney disease (Table 6). In accordance with the current study, it has been reported that there is a relationship between PAT volume measured by CT and coronary artery disease and related risk factors. These results support that there may be an association between pat thickness assessed by TTE and PAT volume assessed by CT [19,20].

PAT measured using TTE can be used as accessible parameter in the follow-up of aortic aneurysms. A follow-up study conducted in Japan using CT reported that para-aortic adipose tissue measured from the periphery of the abdominal aorta is associated with aortic dilatation and dilatation progression [21].

Although the mechanism of the relationship between PAT and aortic dilatation has not been fully elucidated, there is abundant data which indicate that PAT contributes to aortic remodelling through the growth factor, cytokines, interleukins, and adipokines it secretes due to its proximity to the aorta [9,22–25]. Increased production of inflammatory cytokines in the periaortic adipose tissue surrounding the aorta and significantly more inflammatory cell infiltration in the adipose tissue surrounding the atherosclerotic aorta compared to the normal aorta has been reported [26,27]. In the current study, we found PAT to be the strongest predictor of aortic width in multidirectional regression analysis. Similarly, the Framingham Heart Study, which involved the CT scans of 3000 patients, revealed a relationship between aortic enlargement and perivascular adipose tissue. Furthermore, a subgroup analysis conducted on 965 patients of the total 3000 patients, reported that this relationship also involved adipokines and resistin [16,28].

PAT measurements from the ascending aorta region were taken using TTE in the current study. In a study conducted with 1492 patients, Chun-Ho Yun *et al.* measured PAT using CT from the same region and found that the size of the PAT from this region was associated with subclinical atherosclerosis, systemic inflammation, metabolic syndrome, and many cardiovascular risk factors [29].

In line with relevant results in the literature [30,31], we found that age and left ventricular hypertrophy are important predictors of aortic enlargement. Additionally, low body mass index and female gender were found to be associated with increased aortic width. Consistent with our data, there are numerous studies reporting that aortic enlargement is more common in women than in men [32]. But there is a paradoxical effect of BMI on aortic width. Because we used aortic size index which was defined dividing ascending aortic diameter by BSA. Therefore, a false negative relationship was found between aortic dilatation and BMI. To correct this, the diameter of the ascending aorta was divided into length instead of the BSA. After normalised aortic diameter with height, a positive correlation was found between aortic diameter and BMI ($p = 0.003$). In accordance with our findings, there are accumulated evidence indicating a positive relationship between larger BMI and aortic enlargement [32,33]. Similarly, in a study conducted using CT, aortic dilatation and aortic enlargement velocity were

Table 6. Baseline characteristics of the study groups according to para aortic adipose tissue.

	Para aortic adipose tissue		
	≤ 8 mm (163)	> 8 mm (158)	*p* Value
Age (year)	55.3 ± 13.1	63.9 ± 11.7	**<0.000**
Weight (kg)	75 (19)	78.5 (17)	0.155
Height (cm)	165 (12)	163.5 (15)	0.132
Body surface area (m^2)	1.9 (0.28)	1.9 (0.26)	0.411
Waist Circumference	100 (19)	110 (17)	**<0.000**
Glomerular filtration rate (mL/min/1.73m^2)	96 (22)	89 (15)	**<0.000**
Total cholesterol (mg/dL)	191 (54)	190 (48)	0.216
Triglyceride (mg/dL)	134 (74)	133 (87)	0.540
Low-density lipoprotein (mg/dL)	110 (47)	113 (45)	0.960
High-density lipoprotein (mg/dL)	45 (15)	43 (14)	0.180
Glucose (mg/dL)	102 (20)	107 (46)	**0.003**
Left ventricular ejection fraction (%)	65.6 (7.4)	64 (8.8)	**0.019**
Left ventricular mass index (gr/m^2)	84 (21)	89 (22)	**0.022**
Female gender (n, %)	92 (56.4)	96 (60.8)	0.252
Hypertension (n, %)	82 (50.3)	132 (83.5)	**<0.000**
Diabetes mellitus (n, %)	33 (20.2)	61 (38.6)	**<0.000**
Coronary artery disease (n, %)	8 (4.9)	35 (22.2)	**<0.000**
Chronic kidney disease (n, %)	32 (19.6)	47 (29.7)	**0.039**
Hyperlipidemia (n, %)	15 (9.2)	20 (12.7)	0.372

Continuous variables are normally distributed showed Mean ± standard deviation; continuous variables are not normally distributed showed as median (interquartile range); categorical variables are presented as number (percentage).
Bold values are statistically significant.

reported to be associated with age and body surface area [34]. Moreover, the 4-year and 16-year follow-up data of the Framingham Heart Study revealed that increased age and body surface area were closely associated with the development of aortic remodelling [35].

Limitations of the study

There are some limitations to this study, such as the study's cross-sectional methodology, recorded interobserver differences, and the fact that PAT measurement could not be performed in every patient is due to insufficient image quality. In addition, PAT was measured only from around the ascending aorta and as a length measurement. The relationship between PAT, measured by TTE, and PAT volume, measured by CT and MR, was not evaluated. Finally, our results did not demonstrate the effect of PAT on aortic remodelling or the adipokinin, growth factors, etc. secreted by PAT as a paracrine organ. We do not have follow-up results. Therefore, PAT may predict the presence of an aortic aneurysm. However, no assumptions can be made regarding the development of an aortic aneurysm. Therefore, further study is necessary to corroborate its results and studies using larger samples would be valuable.

Conclusion

This study is the first measuring para-aortic adipose tissue from the ascending aorta using TTE. In addition, PAT measured by TTE was found to be the most important predictor of ascending aorta dilatation.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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