

Evaluation of the Relationship between Para-aortic Adipose Tissue and Ascending Aortic Diameter using a New Method

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Abstract

Purpose: 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 for the first time 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 21mm/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.

Conclusion: Our results showed an association between PAT measured by transthoracic echocardiography and ascending aorta width. PAT appears to be an important follow-up parameter in patients at risk of developing aortic aneurysm.

Introduction

Ascending aortic aneurysm is characterized 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 hypothesized that PAT contributes significantly to aortic atherosclerosis and enlargement via its secretion of bioactive molecules such as adiponectins 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 hospital between January 2018 and September 2018 and underwent TTE. Patients who had undergone previous cardiac surgery, e.g., coronary artery bypass surgery and heart valve replacement, 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 (Figure1). 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 $\geq 21mm/m^2$ were deemed to indicate AAD [11].]

Patients were categorized into two groups according to ASI. The AAD group consisted of patients with ASI of $21mm/m^2$ or greater and the non-AAD group patients ASI lower than $21mm/m^2$. 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) / height² (m). Body surface area (BSA) was calculated using the formula of BSA (m^2) = ([Height (cm) x 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.

Transthoracic Echocardiography

Transthoracic echocardiography (TTE) was carried out using a GE Healthcare Vivid S5 7S-RS 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 the inner edges 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 [12]. 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).

Statistical Analysis

Data were analyzed 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 \pm 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 analyze normally distributed data and data that did not conform to the normal distribution were analyzed 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 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, 84 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 categorized 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, $p < 0.0001$).

Table 1
Baseline characteristics of the study groups with and without 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.).

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 remodeling (n, %)	121 (53.8)	42 (43.8)	0.113
Concentric hypertrophy (n, %)	13 (5.8)	17 (17.7)	0.001

Continuous variables are presented as median (interquartile range).

Aortic Dilatation

Eccentric hypertrophy (n, %)	23 (10.2)	21 (21.9)	0.008
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Continuous variables are presented as median (interquartile range).

Table 3
Univariate analysis for 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
Insulin	-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

	β	P value
Left ventricle hypertrophy (%)	1.235	<0.000
LV geometry (%)		
Normal	-0.773	0.013
Concentric remodeling	-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 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.

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 weakly 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.

Discussion

Untreated and unmonitored aortic aneurysm may result in aortic dissection leading to mortality. The etiology of aortic dilatation is multifactorial. In this context, PAT, as a paracrine organ, affects aortic dilatation through the cytokines it secretes [13], which has been reported to result in more aortic function, width, and atherosclerosis [14, 15]. PAT measurement can be used safely in the follow-up of aortic dilatation [16]. Various studies in the literature have discussed the measurement of PAT using CT and MRI. PAT measurement via transthoracic echocardiogram has not received the attention it deserves in the 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. To the best of our knowledge, this is the first study in which PAT measurement was performed using TTE. We found that PAT measured using TTE is an important predictor of ascending aortic width.

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 [17].

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 remodeling through the growth factor, cytokines, interleukins, and adipokines it secretes due to its proximity to the aorta [18–22]. 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 [23, 24]. 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 [14, 25].

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 [26].

In line with relevant results in the literature [27, 28], we found that age and left ventricular hypertrophy are important predictors of aortic enlargement. Additionally, low body surface area and female gender were found to be associated with increased aortic width. Similarly, in a study conducted using CT, aortic dilatation and aortic enlargement velocity were reported to be associated with age and body surface area [29]. 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 remodeling [30].

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 due to insufficient image quality. In addition, PAT was measured only from around the ascending aorta and as a length measurement. Finally, our results did not demonstrate the effect of para-aortic adipose tissue on aortic remodeling or the adipokines, growth factors, etc. secreted by PAT as a paracrine organ. Therefore, further study is necessary to corroborate its results and studies using larger samples would be valuable.

Conclusion

This study is the first in the literature measuring para-aortic adipose tissue from the ascending aorta using TTE. In addition, PAT was found to be the most important predictor of aortic width. In conclusion, PAT measurement can be an important follow-up parameter in patients at risk of developing aortic aneurysm.

Declarations

Conflict interest : None

References

1. Erbel, R., et al., *2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC)*. Eur Heart J, 2014. **35**(41): p. 2873-926.
2. Isselbacher, E.M., *Thoracic and abdominal aortic aneurysms*. Circulation, 2005. **111**(6): p. 816-28.
3. Urabe, G., et al., *Structural analysis of adventitial collagen to feature aging and aneurysm formation in human aorta*. J Vasc Surg, 2016. **63**(5): p. 1341-50.

4. Peshkova, I.O., G. Schaefer, and E.K. Koltsova, *Atherosclerosis and aortic aneurysm - is inflammation a common denominator?* FEBS J, 2016. **283**(9): p. 1636-52.
5. Yun, C.H., et al., *Pericardial and thoracic peri-aortic adipose tissues contribute to systemic inflammation and calcified coronary atherosclerosis independent of body fat composition, anthropometric measures and traditional cardiovascular risks.* Eur J Radiol, 2012. **81**(4): p. 749-56.
6. Fox, C.S., et al., *Periaortic fat deposition is associated with peripheral arterial disease: the Framingham heart study.* 2010. **3**(5): p. 515-519.
7. Lehman, S.J., et al., *Peri-aortic fat, cardiovascular disease risk factors, and aortic calcification: the Framingham Heart Study.* 2010. **210**(2): p. 656-661.
8. Freiberg, M., et al., *Abdominal aortic aneurysms, increasing infrarenal aortic diameter, and risk of total mortality and incident cardiovascular disease events: 10-year follow-up data from the Cardiovascular Health Study.* 2008. **117**.
9. Dias-Neto, M., et al., *High density of periaortic adipose tissue in abdominal aortic aneurysm.* 2018. **56**(5): p. 663-671.
10. Roman, M.J., et al., *Two-dimensional echocardiographic aortic root dimensions in normal children and adults.* Am J Cardiol, 1989. **64**(8): p. 507-12.
11. Davies, R.R., et al., *Novel measurement of relative aortic size predicts rupture of thoracic aortic aneurysms.* Ann Thorac Surg, 2006. **81**(1): p. 169-77.
12. Sicari, R., et al., *The use of pocket-size imaging devices: a position statement of the European Association of Echocardiography.* Eur J Echocardiogr, 2011. **12**(2): p. 85-7.
13. Turkmen, K., et al., *Peri-aortic fat tissue and malnutrition-inflammation-atherosclerosis/calcification syndrome in end-stage renal disease patients.* Int Urol Nephrol, 2013. **45**(3): p. 857-67.
14. Thanassoulis, G., et al., *Periaortic adipose tissue and aortic dimensions in the Framingham Heart Study.* J Am Heart Assoc, 2012. **1**(6): p. e000885.
15. Watts, S.W., et al., *A New Function for Perivascular Adipose Tissue (PVAT): Assistance of Arterial Stress Relaxation.* Sci Rep, 2020. **10**(1): p. 1807.
16. Yun, C.H., et al., *The association among peri-aortic root adipose tissue, metabolic derangements and burden of atherosclerosis in asymptomatic population.* J Cardiovasc Comput Tomogr, 2016. **10**(1): p. 44-51.

17. Yamaguchi, M., et al., *Clinical Significance of Increased Computed Tomography Attenuation of Periaortic Adipose Tissue in Patients With Abdominal Aortic Aneurysms*. Circ J, 2021.
18. Li, X., et al., *Perivascular adipose tissue-derived extracellular vesicle miR-221-3p mediates vascular remodeling*. FASEB J, 2019. **33**(11): p. 12704-12722.
19. Sakaue, T., et al., *Perivascular Adipose Tissue Angiotensin II Type 1 Receptor Promotes Vascular Inflammation and Aneurysm Formation*. Hypertension, 2017. **70**(4): p. 780-789.
20. Dias-Neto, M., et al., *High Density of Periaortic Adipose Tissue in Abdominal Aortic Aneurysm*. Eur J Vasc Endovasc Surg, 2018. **56**(5): p. 663-671.
21. Lee, M.H., et al., *Perivascular adipose tissue inhibits endothelial function of rat aortas via caveolin-1*. PLoS One, 2014. **9**(6): p. e99947.
22. Moe, K.T., et al., *Tumor necrosis factor-alpha induces aortic intima-media thickening via perivascular adipose tissue inflammation*. J Vasc Res, 2013. **50**(3): p. 228-37.
23. Chatterjee, T.K., et al., *Proinflammatory phenotype of perivascular adipocytes: influence of high-fat feeding*. 2009. **104**(4): p. 541-549.
24. Henrichot, E., et al., *Production of chemokines by perivascular adipose tissue: a role in the pathogenesis of atherosclerosis?* 2005. **25**(12): p. 2594-2599.
25. Piacentini, L., et al., *Gene-expression profiles of abdominal perivascular adipose tissue distinguish aortic occlusive from stenotic atherosclerotic lesions and denote different pathogenetic pathways*. Sci Rep, 2020. **10**(1): p. 6245.
26. Yun, C.H., et al., *Quantification of peri-aortic root fat from non-contrast ECG-gated cardiac computed tomography*. Data Brief, 2015. **5**: p. 995-8.
27. Kauhanen, S.P., et al., *High prevalence of ascending aortic dilatation in a consecutive coronary CT angiography patient population*. Eur Radiol, 2020. **30**(2): p. 1079-1087.
28. Argan, O., et al., *Epicardial adipose tissue is a predictor of ascending aortic dilatation in hypertensive patients, but not paracardial adipose tissue*. BMC Cardiovasc Disord, 2020. **20**(1): p. 142.
29. Chang, H.W., et al., *Diameter and growth rate of the thoracic aorta-analysis based on serial computed tomography scans*. J Thorac Dis, 2020. **12**(8): p. 4002-4013.
30. Lam, C.S., et al., *Aortic root remodeling over the adult life course: longitudinal data from the Framingham Heart Study*. Circulation, 2010. **122**(9): p. 884-90.

Figures

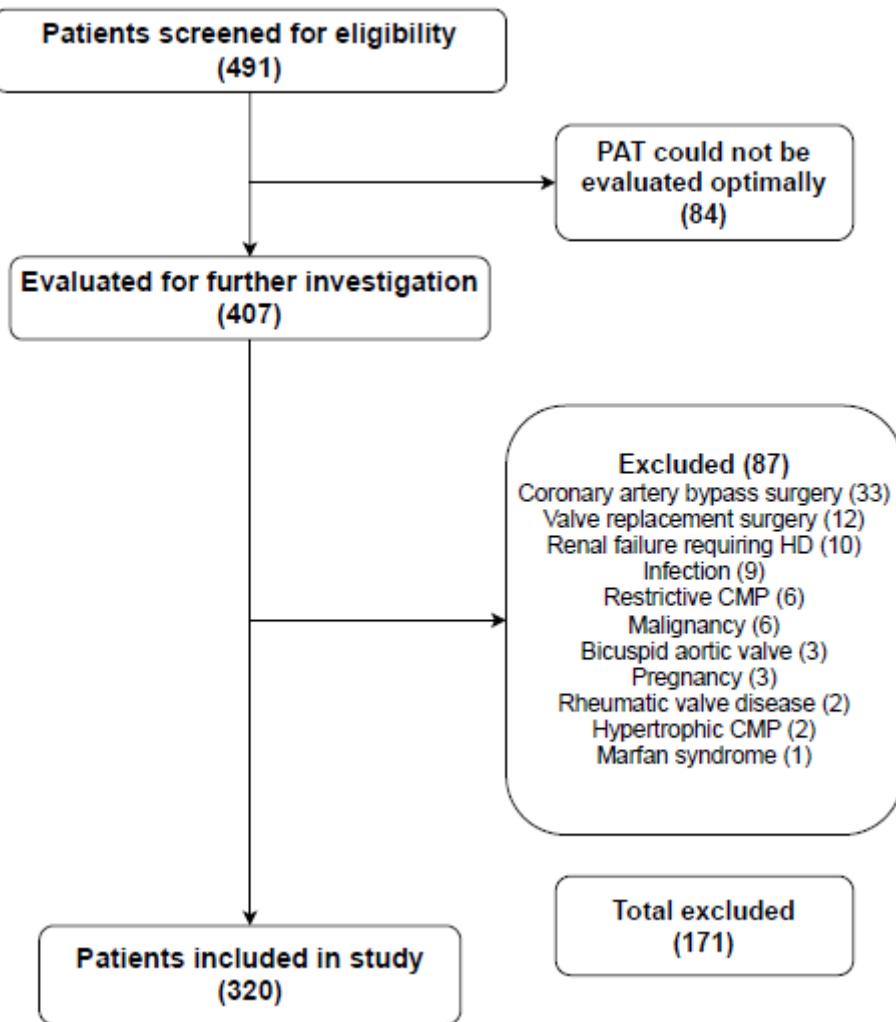


Figure 1

Flow-chart of patient selection



Figure 2

Para-aortic adipose tissue measurement with transthoracic echocardiography

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