Original Article | Cardiovascular Imaging

eISSN 2005-8330
https://doi.org/10.3348/kjr.2023.0339
Korean J Radiol 2023;24(10):974-982

Korean Journal of Radiology
KJR

Assessing Abdominal Aortic Aneurysm Progression by Using Perivascular Adipose Tissue Attenuation on Computed Tomography Angiography

Shuai Zhang[1]*, Hui Gu[1]*, Na Chang[2], Sha Li[3], Tianqi Xu[3], Menghan Liu[4], Ximing Wang[1]

[1]Department of Radiology, Shandong Provincial Hospital Affiliated to Shandong First Medical University, Shandong University, Jinan, Shandong, China
[2]Department of Medical Technology, Jinan Nursing Vocational College, Jinan, Shandong, China
[3]Department of Clinical Medicine, Shandong Provincial Hospital, Shandong University, Jinan, Shandong, China
[4]Depertment of Health Management, The First Affiliated Hospital of Shandong First Medical University, Jinan, Shandong, China

Objective: Recent studies have highlighted the active and potential role of perivascular adipose tissue (PVAT) in atherosclerosis and aneurysm progression, respectively. This study explored the link between PVAT attenuation and abdominal aortic aneurysm (AAA) progression using computed tomography angiography (CTA).

Materials and Methods: This multicenter retrospective study analyzed patients with AAA who underwent CTA at baseline and follow-up between March 2015 and July 2022. The following parameters were obtained: maximum diameter and total volume of the AAA, presence or absence of intraluminal thrombus (ILT), maximum diameter and volume of the ILT, and PVAT attenuation of the aortic aneurysm at baseline CTA. PVAT attenuation was divided into high (> -73.4 Hounsfield units [HU]) and low (≤ -73.4 HU). Patients who had or did not have AAA progression during the follow-up, defined as an increase in the aneurysm volume > 10 mL from baseline, were identified. Kaplan–Meier and multivariable Cox regression analyses were used to investigate the association between PVAT attenuation and AAA progression.

Results: Our study included 167 participants (148 males; median age: 70.0 years; interquartile range: 63.0–76.0 years), of which 145 (86.8%) were diagnosed with AAA accompanied by ILT. Over a median period of 11.3 months (range: 6.0–85.0 months), AAA progression was observed in 67 patients (40.1%). Multivariable Cox regression analysis indicated that high baseline PVAT attenuation (adjusted hazard ratio [aHR] = 2.23; 95% confidence interval [CI], 1.16–4.32; P = 0.017) was independently associated with AAA progression. This association was demonstrated within the patients of AAA with ILT subcohort, where a high baseline PVAT attenuation (aHR = 2.23; 95% CI, 1.08–4.60; P = 0.030) was consistently independently associated with AAA progression.

Conclusion: Elevated PVAT attenuation is independently associated with AAA progression, including patients of AAA with ILT, suggesting the potential of PVAT attenuation as a predictive imaging marker for AAA expansion.

Keywords: Abdominal aortic aneurysm; Perivascular adipose tissue; Intraluminal thrombus; Aneurysm progression; CT angiopraphy

Received: April 13, 2023 **Revised:** June 6, 2023 **Accepted:** July 11, 2023

*These authors contributed equally to this work.
Corresponding author: Ximing Wang, PhD, Department of Radiology, Shandong Provincial Hospital Affiliated to Shandong First Medical University, Shandong University, No. 324, Jingwu Road, Jinan 250021, Shandong, China
• E-mail: wangximing369@yeah.net
Corresponding author: Menghan Liu, PhD, Depertment of Health Management, The First Affiliated Hospital of Shandong First Medical University, No. 16766, Jingshi Road, Jinan 250014, Shandong, China
• E-mail: lmh141216@163.com

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INTRODUCTION

Abdominal aortic aneurysm (AAA), which is defined as an aortic enlargement with a diameter of ≥ 3 cm, is a life-threatening condition [1,2]. The prevalence of AAA ranges from 1.2% to 3.3% in men aged > 60 years, and the mortality rate of ruptured AAA is estimated to be as high as 81% [2,3]. AAA can be dangerous if not detected early; it increases over time and can rupture, causing life-threatening bleeding. Therefore, early diagnosis is crucial.

Clinically, the maximum diameter of AAA, which is widely accepted as the main parameter for predicting AAA progression [4,5], can be detected by computed tomography angiography (CTA), abdominal and pelvic ultrasonography, and magnetic resonance imaging. However, using maximum diameter measurements to predict rapid growth has limited efficacy, as some smaller AAAs grow rapidly, whereas large AAAs may grow more slowly.

In recent years, the analysis of additional aneurysm characteristics for diagnosing AAA, such as intraluminal thrombus (ILT), vessel wall pressure, and aneurysm wall inflammation, has attracted considerable attention [6-9]. Aortic wall inflammation plays a significant pathophysiological role in the progression of AAA [9]. Previous studies have demonstrated perivascular adipose tissue (PVAT) and vascular inflammation to have a reciprocal relationship. In addition, the perivascular fat attenuation index on CTA has recently been recommended as a quantitative measure of vascular inflammation [10,11]. Several researchers have studied the association between PVAT and atherosclerosis and found that PVAT could be helpful in cardiovascular and cerebrovascular risk prediction and stratification [12,13]. With reference to AAA, Kugo et al. [14] have reported that PVAT can promote AAA progression. Moreover, Dias-Neto et al. [15] found that perivascular fat attenuation on CTA was associated with the presence of AAA. Additionally, several studies have investigated AAA, aortoiliac occlusive disease, and healthy individuals using PVAT attenuation on CTA [15,16], and concluded that PVAT could have an effect on aortic pathophysiology. However, the association between the PVAT attenuation on CTA and AAA progression is not fully elucidated. Thus, our study aimed to explore the link between PVAT attenuation and AAA progression using CTA.

MATERIALS AND METHODS

Study Population

The institutional review board of Shandong Provincial Hospital Affiliated to Shandong First Medical University approved this study (SWYX: No. 2023-308) and waived the requirement for informed consent. This retrospective study included patients with AAA from four hospitals between March 2015 and July 2022. The inclusion criteria were as follows: 1) patients with AAA who underwent abdominal CTA examination and 2) follow-up computed tomography (CT) scans at least 6 months after the initial CTA examination. The exclusion criteria were as follows: 1) endovascular or open aortic repair at the initial CTA scan or within the imaging interval; 2) other aortic diseases that could affect image analysis, such as aortic dissection and aortic occlusion; 3) incomplete clinical data; and 4) poor image quality.

The patient selection flowchart is shown in Figure 1. Clinical and follow-up data, including physical examination and images, were collected from the medical records. The follow-up endpoints were the time to the first detection of AAA progression and the time to the last follow-up if the AAA was stable.

CT Imaging Protocol

All abdominal CT images were obtained using multi-detector CT scanners (Somatom Force, Siemens Healthcare; Somatom Definition Flash, Siemens Healthcare; Ingenuity CT, Philips; Optima CT660, GE Healthcare; Discovery 750, GE Healthcare). For enhanced CT scanning, 100–120 mL contrast medium (Omnipaque-350; GE Healthcare) was injected at a speed of 4.5 mL/s. The detailed CT protocol is shown in the Supplementary Material.

Image Analysis

All the images were reviewed by two radiologists (SZ and HG, with 7 and 10 years of experience in vascular imaging, respectively) who were blinded to the patients' clinical information. Any disagreement regarding the assessment was resolved by consensus. The following parameters were collected at baseline and follow-up CT: maximum diameter and total volume of the AAA, presence or absence of ILT, maximum diameter and volume of the ILT, and PVAT attenuation of the aortic aneurysm. Images were reviewed and the diameters of the AAA and ILT were measured using a post-processing workstation (Syngo. via; Siemens Force). Measurements of the maximum AAA diameter were

 https://doi.org/10.3348/kjr.2023.0339

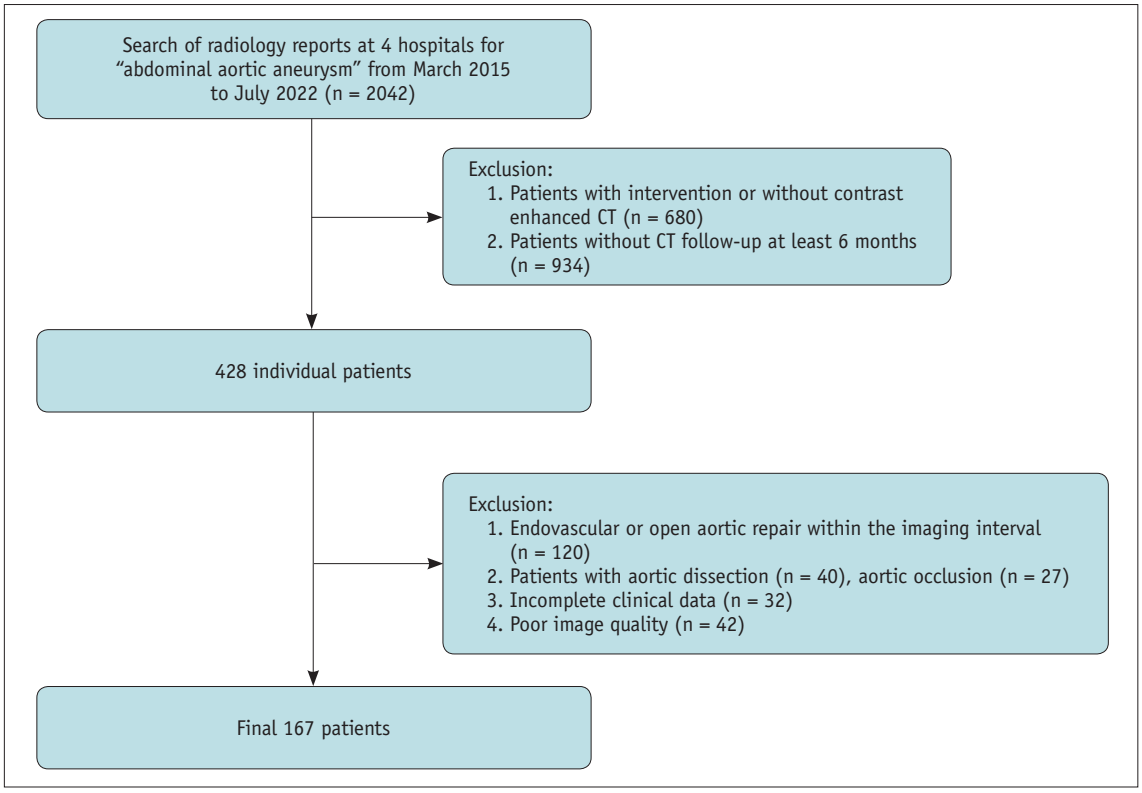

Fig. 1. Flow diagram illustrates the study design and study population. A total of 2042 patients with abdominal aortic aneurysm were initially enrolled, of which 1875 patients were excluded. Finally, 167 patients were included. CT = computed tomography

performed on maximum cross-sections perpendicular to the long axis of the aorta using a multiplanar reconstruction method [6,17]. The maximum diameter of the ILT was measured on the maximum thrombus cross sections perpendicular to the long axis of the aorta. The superior and inferior borders of the AAA were defined as the loss of parallelism of the aortic wall to the end of aortic dilatation. AAA segmentation was performed by manually placing regions of interest slice-by-slice at the aortic border from the upper to the lower boundaries of the AAA. ILT segmentation was performed using a similar method. After segmentation, the AAA and ILT volumes were computed by summing the volumes of the segmented voxels using a dedicated ITK-SNAP software (version 3.8.0, open source, http://www.itksnap.org). PVAT segmentation was performed using semi-automatic commercial perivascular fat analysis software (Version 1.0, Lianying Technology). The regions of interest were manually segmented into the cross-sectional areas of the AAA. Contouring was performed within the border of the AAA, and the adjacent normal tissues were carefully avoided. Thereafter, PVAT was automatically segmented, and PVAT attenuation measurements were automatically generated by the software by calculating the mean of all the fat pixels within a 5 mm range around the AAA (Fig. 2). PVAT attenuation was determined by quantifying the weighed perivascular fat attenuation after adjusting for the technical parameters based on the attenuation histogram of perivascular fat within the range of -190 to -30 Hounsfield units (HU). Thus, the PVAT attenuation was the average of all the weighed perivascular fat attenuations obtained in the range of -190 to -30 HU. The longitudinal measurement range of the PVAT attenuation was the upper and lower boundaries of the AAA, and the transverse measurement range was 5 mm around the aortic wall. All the fat density measurements were reported in HU. The AAA volume change was calculated as follows: AAA volume at follow-up and AAA volume at baseline. An increase in the AAA volume > 10 mL was defined as the progression [7].

Statistical Analysis

The Shapiro–Wilk test was used to assess the normality of the data distribution. Continuous variables are presented as mean ± standard deviation or median (interquartile range), and categorical variables are presented as percentages. Univariable and multivariable Cox regression analyses were used to calculate the hazard ratios (HRs) and the

https://doi.org/10.3348/kjr.2023.0339

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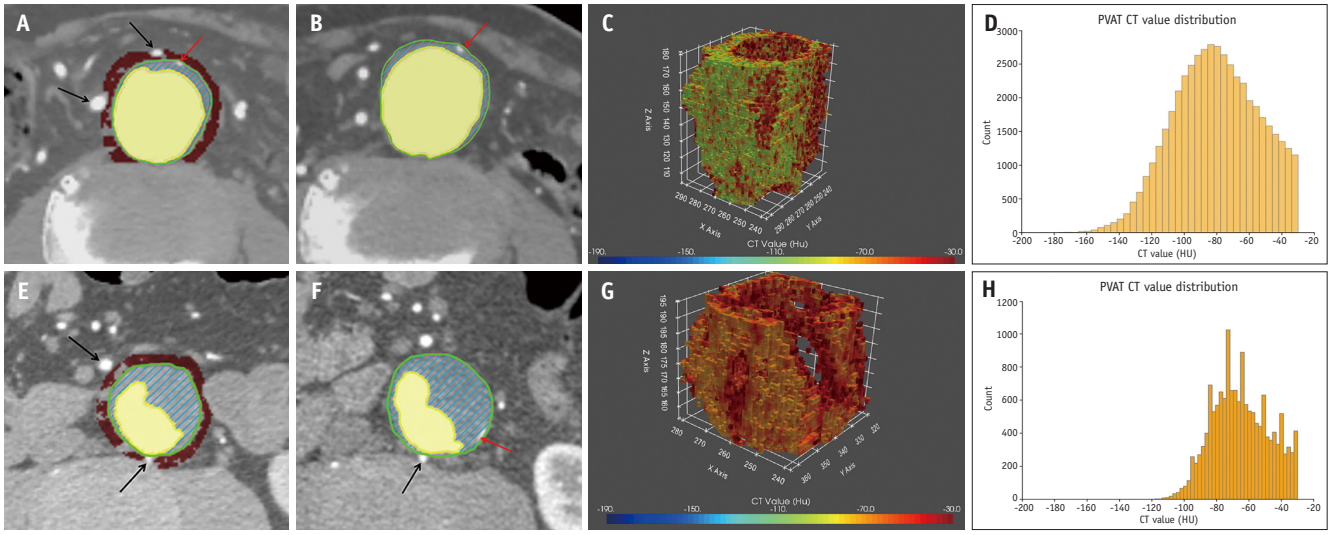

Fig. 2. Examples of PVAT attenuation in AAA progression and non-progression. **A, B:** Axial contrast-enhanced computed tomography images show that the aneurysm grew from 48.0 mL to 52.5 mL over 1.9 years at a growth rate of 1.2 mL/y, with corresponding PVAT attenuation range and color presentation. **C, D:** The histogram and pixel diagram of PVAT attenuation at the baseline of AAA non-progression. **E, F:** Axial contrast-enhanced CT images show that the aneurysm grew from 59.5 mL to 88.4 mL over 0.8 years at a growth rate of 36.1 mL/y. **G, H:** The histogram and pixel diagram of PVAT attenuation at the baseline of AAA progression. The pixel diagram shows the three-dimensional position distribution of pixels with different PVAT attenuation. Different colors indicate different densities. The histogram shows the number distribution of pixels with different PVAT attenuation. The PVAT attenuation (-62.9 HU) in AAA progression was lower than that (-75.7 HU) in AAA non-progression. The yellow regions indicate enhanced aortic lumen, the dark red regions indicate periaortic fat tissue, the black arrows indicate vascular side branches, the red arrows indicate calcification, the blue diagonal line regions indicate intraluminal thrombus, and the green circles indicate aortic wall **(A, B, E, F)**. PVAT = perivascular adipose tissue, AAA = abdominal aortic aneurysm, CT = computed tomography

corresponding 95% confidence intervals (CIs) of the factors associated with AAA progression. Youden's index was used to determine the cutoff value for dichotomization of PVAT attenuation. According to the cutoff value, patients were divided into high (> -73.4 HU) and low (≤ -73.4 HU) PVAT attenuation groups. AAA progression-free survival was evaluated using Kaplan–Meier curves, and the log-rank test was used to determine the differences between the Kaplan-Meier curves. Patient outcomes were collected based on changes in AAA volume between the baseline and follow-up CTA. The relationship between PVAT attenuation and the AAA maximal diameter was tested using Spearman's correlation coefficient. Analyses were performed for all the patients and for a subgroup of patients with ILT.

We randomly selected 50 patients to compare the PVAT attenuation measurements based on non-contrast CT and CTA. The Student's *t*-test for paired-sample *t*-tests was used to analyze the paired differences between PVAT attenuation measured using non-contrast CT and CTA. Inter-observer reproducibility was assessed in additional 50 randomly chosen patients using intraclass correlation coefficients and kappa statistics. $P < 0.05$ was considered statistically significant. All the statistical analyses were performed using SPSS (version 22.0; IBM).

RESULTS

Patient Characteristics

A total of 167 patients (148 males; median age, 70.0 years; interquartile range, 63.0–76.0 years) were included in the final analysis. The median follow-up was 11.3 months (range, 6.0–85.0 months). Among these, 67 patients (40.1%) showed AAA progression. The detailed clinical data of all the patients with AAA are listed in Table 1.

Predictors for AAA Progression

Univariable Cox regression identified the maximal diameter (HR = 1.05; 95% CI, 1.03–1.07; $P < 0.001$), total aneurysm volume (HR = 1.00; 95% CI, 1.00–1.01; $P = 0.022$), ILT maximal diameter (HR = 1.05; 95% CI, 1.02–1.08; $P = 0.002$), ILT volume (HR = 1.01; 95% CI, 1.00–1.01; $P = 0.032$), and high PVAT attenuation at baseline (HR = 3.83; 95% CI, 2.24–6.53; $P < 0.001$) as significant inputs, which were subsequently entered into the multivariable analysis (Table 2). Multivariable Cox regression analysis revealed that maximal diameter (adjusted HR = 1.11; 95%

https://doi.org/10.3348/kjr.2023.0339

Table 1. Baseline clinical characteristics of patients with abdominal aortic aneurysm

Parameter	All patients (n = 167)
Age, yr	70.0 (63.0–76.0)
Sex, male	148 (88.6)
Hypertension	120 (91.9)
Diabetes	41 (24.6)
Hyperlipidemia	61 (36.5)
Smoking history	101 (60.5)
Anti-hypertensive treatment	129 (77.2)
Lipid-lowering treatment	69 (41.3)
Interval between CTA scans, month	11.3 (6.7–26.4)

Values are presented as medians (interquartile ranges) or number of patients (%).
CTA = computed tomography angiography

CI, 1.04–1.19; $P = 0.001$) and high PVAT attenuation at baseline (adjusted HR = 2.23; 95% CI, 1.16–4.32; $P = 0.017$) were independent predictors of AAA progression (Table 2).

The Kaplan–Meier curves for AAA progression-free survival stratified according to PVAT attenuation are shown in Figure 3. For all the patients with AAA, AAA progression-free survival was significantly lower in patients with high PVAT attenuation than in those with low PVAT attenuation (log-rank $P < 0.001$).

Spearman's correlation analysis revealed that PVAT attenuation was positively correlated with the maximal AAA diameter ($r = 0.624$, $P < 0.001$). Paired sample *t*-test showed no significant difference ($P = 0.139$) between PVAT attenuation (non-contrast CT vs. CTA, -83.4 ± 9.1 HU vs. -82.9 ± 9.9 HU) measured from non-contrast CT and CTA in 50 patients.

AAA with ILT Subcohort

A total of 145 patients (86.8%) had AAA with ILT, which constituted the AAA with ILT subcohort. Among them, 60 (41.4%) patients had AAA progression. Univariable and multivariable Cox regression analyses further suggested that maximal diameter (adjusted HR = 1.10; 95% CI, 1.03–1.18; $P = 0.007$) and high PVAT attenuation at baseline (adjusted HR = 2.23; 95% CI, 1.08–4.60; $P = 0.030$) were independent predictors of AAA progression (Table 2). For patients with AAA with ILT, the Kaplan–Meier curves for the progression of AAA showed that AAA progression-free survival was significantly lower in patients with high PVAT attenuation than in those with low PVAT attenuation (log-rank $P < 0.001$) (Fig. 3).

Table 2. Univariable and multivariable Cox regression analyses of risk factors for AAA progression in whole cohort and in AAA with ILT subcohort

Parameter	Whole cohort (n = 167)				AAA with ILT subcohort (n = 145)			
	Univariable analysis		Multivariable analysis		Univariable analysis		Multivariable analysis	
	HR (95% CI)	*P*	aHR (95% CI)	*P*	HR (95% CI)	*P*	aHR (95% CI)	*P*
Age, yr†	1.01 (0.99–1.03)	0.496			1.00 (0.97–1.03)	0.979		
Sex (male vs. female)*	1.92 (0.83–4.46)	0.129			2.11 (0.76–5.85)	0.151		
Hypertension (present vs. absent)*	0.91 (0.53–1.55)	0.726			0.87 (0.49–1.52)	0.619		
Diabetes (present vs. absent)*	0.81 (0.44–1.49)	0.491			0.79 (0.41–1.53)	0.488		
Hyperlipidemia (present vs. absent)*	1.13 (0.66–1.95)	0.658			1.24 (0.69–2.27)	0.476		
Smoking history (present vs. absent)*	1.09 (0.66–1.82)	0.727			1.22 (0.70–2.13)	0.482		
Anti-hypertensive treatment (present vs. absent)*	1.05 (0.60–1.84)	0.870			0.92 (0.51–1.66)	0.788		
Lipid-lowering treatment (present vs. absent)*	1.06 (0.63–1.76)	0.830			1.03 (0.59–1.79)	0.913		
Maximal diameter, mm†	1.05 (1.03–1.07)	< 0.001	1.11 (1.04–1.19)	0.001	1.05 (1.03–1.08)	< 0.001	1.10 (1.03–1.18)	0.007
Total aneurysm volume, mL†	1.00 (1.00–1.01)	0.022	0.99 (0.98–1.00)	0.180	1.00 (1.00–1.01)	0.010	0.99 (0.98–1.00)	0.130
ILT (present vs. absent)*	1.53 (0.70–3.36)	0.290						
ILT maximal diameter, mm†	1.05 (1.02–1.08)	0.002	1.02 (0.98–1.07)	0.331	1.05 (1.02–1.08)	0.004	1.01 (0.97–1.05)	0.678
ILT volume, mL†	1.01 (1.00–1.01)	0.032	1.00 (0.99–1.01)	0.438	1.01 (1.00–1.01)	0.058		
PVAT attenuation (high vs. low)*	3.83 (2.24–6.53)	< 0.001	2.23 (1.16–4.32)	0.017	3.89 (2.17–6.95)	< 0.001	2.23 (1.08–4.60)	0.030

*For categorical variables with categories in parentheses, the former was compared with the latter (the reference) to calculate HRs and 95% CIs with the Cox regression analysis,
†For continuous variables, an increase by 1 unit was considered when calculating HRs and 95% CIs with the Cox regression analysis.
AAA = abdominal aortic aneurysm, ILT = intraluminal thrombus, HR = hazard ratio, CI = confidence interval, aHR = adjusted hazard ratio, PVAT = perivascular adipose tissue

https://doi.org/10.3348/kjr.2023.0339

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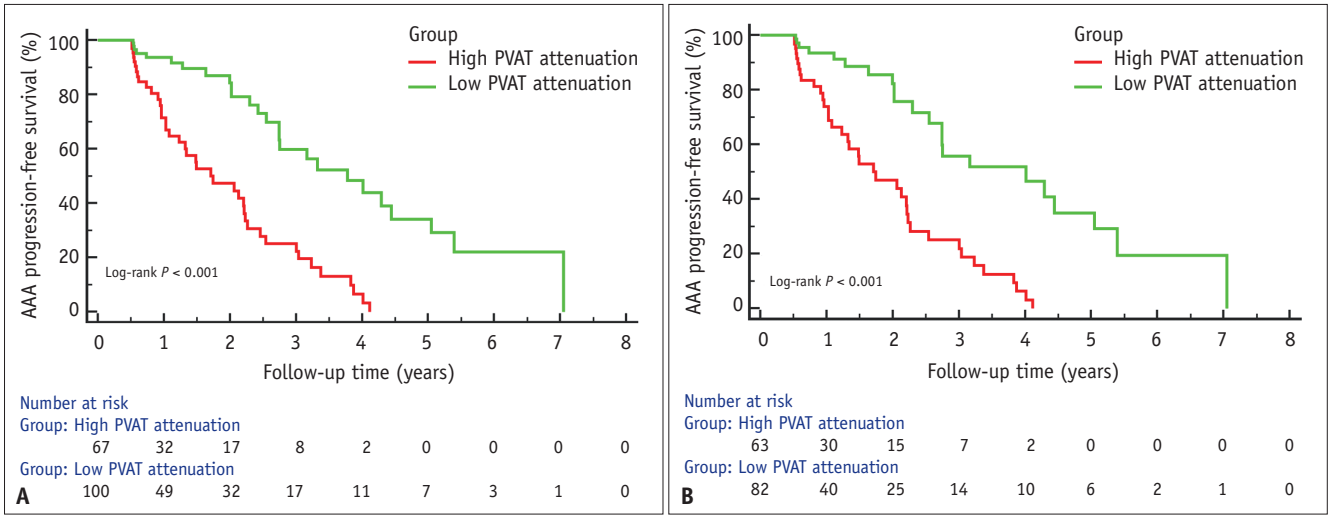

Fig. 3. Kaplan–Meier curves of AAA progression-free survival in the entire cohort **(A)** and AAA with ILT subcohort **(B)**. The patients with high PVAT attenuation had a lower AAA progression-free survival rate than those with low PVAT attenuation in the whole cohort and AAA with the ILT subcohort (both log-rank $P < 0.001$). AAA = abdominal aortic aneurysm, ILT = intraluminal thrombus, PVAT = perivascular adipose tissue

Inter-observer Agreement

The intraclass correlation coefficient of maximal diameter, total aneurysm volume, ILT maximal diameter, ILT volume, and PVAT attenuation were 0.91 (95% CI, 0.89–0.93), 0.87 (95% CI, 0.84–0.90), 0.90 (95% CI, 0.87–0.92), 0.84 (95% CI, 0.80–0.87), and 0.86 (95% CI, 0.82–0.89), respectively, and the Cohens kappa coefficient of the presence of ILT was 0.94 (95% CI, 0.87–0.99) for inter-observer agreement.

DISCUSSION

Previous studies have shown that PVAT plays an important role in the progression of AAA [14,15]. Non-invasive detection of PVAT attenuation in asymptomatic participants is of great importance. Therefore, we investigated the association between attenuation of PVAT and AAA progression. We found that PVAT attenuation was an independent predictor of AAA progression, even in the AAA-with-ILT subcohort. Thus, PVAT attenuation may be a potential imaging marker for predicting AAA progression.

The maximum AAA diameter is the most common measurement used to predict the risk of AAA rupture. Previous studies have determined AAA diameter to be an independent predictor of AAA progression and rupture [4,5,18]. This is consistent with the findings of our study, where maximal diameter (adjusted HR = 1.11; 95% CI, 1.04–1.19; $P = 0.001$) was an independent predictor of AAA progression. However, its accurate measurement is hindered by certain factors such as aortic tortuosity and interobserver variability [18]. Moreover, this method does not account for morphological variations such as saccular aneurysms [18].

Several studies have demonstrated that PVAT can serve as a marker of vascular inflammation; moreover, it has already been used in coronary and carotid atherosclerosis [19,20]. Oikonomou et al. [12] revealed that the perivascular fat attenuation index on coronary CTA has predictive value for major adverse cardiac events. Moreover, Zhang et al. [13] found that an increase in the attenuation density of the carotid PVAT on CTA was related to the risk of cerebrovascular symptoms. In this study, we investigated the association between PVAT and AAA progression and found that PVAT attenuation (adjusted HR = 2.23; 95% CI, 1.16–4.32; $P = 0.017$) was an independent predictor in AAA progression, which is consistent with Yamaguchi's study [16]. In addition, several studies have reported that PVAT attenuation in patients with AAA was higher than that in patients with aortoiliac occlusion and healthy individuals [15]. Gaibazzi et al. [21] demonstrated a significant positive correlation between perivascular fat attenuation and the ascending aortic aneurysm diameter. However, unlike previous studies, we not only investigated the predictive value of PVAT attenuation by multicenter analysis, but also performed a subanalysis of AAA with an ILT subcohort.

Over the years, other features associated with AAA progression have been discussed, including ILT, aortic wall

https://doi.org/10.3348/kjr.2023.0339

stress, and aortic wall inflammation [6-9]. ILT is common in patients with AAAs [22,23]. In the present study, ILT was commonly observed in the AAA group (86.8%). Previous studies have demonstrated that ILT could accelerate AAA progression by inducing hypoxia and releasing pro-inflammatory substances, which could also cause increased inflammatory infiltrates in the PVAT [23,24]. These mechanisms suggest a potential association between PVAT attenuation and AAA progression. Therefore, we performed a subanalysis in AAA using the ILT subcohort, and found that high PVAT attenuation at baseline (adjusted HR = 2.23; 95% CI, 1.08–4.60; P = 0.030) was still an independent predictor of AAA progression in the ILT subcohort.

Under pathological conditions, PVAT promotes inflammation and oxidative stress, suppresses the release of vasoprotective adipocyte-driven relaxation factors and increases the secretion of paracrine factors [9,10]. Therefore, these substances could cause an increased occurrence of metabolic diseases and lead to functional disturbance of the PVAT and AAA progression [25,26]. The formation and progression of AAA are accompanied by aortic wall inflammation, which can lead to inflammatory infiltrates and deposition of collagen fibers in the PVAT [11,27]. There are several possible explanations for the positive correlation between PVAT attenuation and AAA progression.

Open surgery or endovascular repair is the most effective method for treating large AAAs (diameter > 55 mm); however, insufficient evidence exists concerning the benefit of small AAA [8,28]. Therefore, several guidelines recommend elective repair or follow-up, as appropriate for smaller AAAs [28]. Several recent studies have proposed pharmacological treatments, including blood pressure control, improvement of the blood lip, and inhibition of thrombosis and inflammation, as methods to treat small aneurysms [29]. Furthermore, some studies have shown that certain methods can reduce the inflammatory and oxidative status of the PVAT and help alleviate AAA [30]. Therefore, if PVAT attenuation is confirmed as a risk factor for AAA progression in larger trials, mediators of PVAT development may be novel targets for AAA treatment.

Considering the effect of attenuation within the aorta on the PVAT attenuation measurements, we randomly selected 50 patients to compare the measurements of PVAT attenuation based on non-contrast CT and CTA using a paired-sample t-test. A paired-sample t-test showed no significant difference (P = 0.139) between PVAT attenuation measured using non-contrast CT and CTA. In addition, a previous study investigated the quantification of PVAT in contrast-enhanced CTA and non-contrast CT and demonstrated no significant difference in PVAT attenuation measured on contrast-enhanced CTA and non-contrast CT [20], which is consistent with our findings. Further studies based on noncontrast CT are warranted to completely elucidate the effect of attenuation within the aorta.

The present study has several limitations. First, it possesses limitations inherent to retrospective time-to-event analyses, including heterogeneity in the patient follow-up and insufficient follow-up intensity. Second, although this was a multicenter study, our sample size was relatively small, especially for patients with AAA without ILT. Therefore, a larger patient cohort with a longer follow-up period is warranted. Third, given the effects of the peripheral organs on PVAT, the specific measurement ranges for PVAT are not certain. Our study adopted a transverse measurement range of 5 mm around the aortic wall based on previous studies [16]. However, further histological evidence is warranted to validate these results. Finally, we performed a three-dimensional analysis of the AAA features, which may not be as easily measured in the clinic as two-dimensional data but are considered more accurate and beneficial [31].

In conclusion, our study demonstrated that PVAT attenuation is an independent predictor of AAA progression in the entire AAA cohort and in the AAA with ILT subcohort. Therefore, PVAT attenuation may be a potential marker for predicting AAA progression. These findings provide novel insights into the prediction and treatment of AAA progression.

Supplement

The Supplement is available with this article at https://doi.org/10.3348/kjr.2023.0339.

Availability of Data and Material

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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Author Contributions

Conceptualization: Shuai Zhang, Menghan Liu. Data curation: Shuai Zhang, Menghan Liu, Na Chang. Formal analysis: Shuai Zhang, Menghan Liu, Na Chang. Funding acquisition: Shuai Zhang, Menghan Liu, Na Chang. Investigation: Shuai Zhang, Sha Li. Methodology: Shuai Zhang, Sha Li, Tianqi Xu, Hui Gu. Project administration: Shuai Zhang, Sha Li, Tianqi Xu. Resources: Shuai Zhang, Sha Li, Tianqi Xu, Hui Gu, Ximing Wang. Software: Tianqi Xu, Hui Gu, Ximing Wang. Supervision: Hui Gu, Ximing Wang. Validation: Shuai Zhang, Hui Gu, Ximing Wang. Visualization: Shuai Zhang, Hui Gu, Ximing Wang. Writing—original draft: Shuai Zhang, Ximing Wang. Writing—review & editing: Shuai Zhang, Ximing Wang.

ORCID IDs

Shuai Zhang
https://orcid.org/0000-0002-9631-0411
Hui Gu
https://orcid.org/0000-0002-5216-9436
Na Chang
https://orcid.org/0009-0000-4106-1721
Sha Li
https://orcid.org/0000-0002-9415-1576
Tianqi Xu
https://orcid.org/0009-0000-2533-0485
Menghan Liu
https://orcid.org/0009-0001-6054-2472
Ximing Wang
https://orcid.org/0000-0001-5136-8057

Funding Statement

This study was supported by the supported by the National Science Foundation for Scientists of China (82271993, 81871354).

Acknowledgments

The authors thank Mr. Jianqing Sun from the United Imaging Intelligence (Beijing) Co., Ltd. for providing us with software support and post-processing guidance.

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https://doi.org/10.3348/kjr.2023.0339