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Association of Estimated Pulse Wave Velocity with Abdominal Aortic Calcification: A Large Cross-Sectional Study

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Abstract

Objectives: There is evidence that pulse wave velocity (PWV) can predict the occurrence of abdominal aortic calcification (AAC), while the association between estimated PWV (ePWV) and AAC has not been reported, so our study aimed to analyze the association between ePWV and AAC.

Methods: The study enrolled 3140 adults between the ages of 40 and 80 who participated in the 2013–2014 National Health and Nutrition Examination Survey. Using multivariate logistic regression analysis, multivariate linear regression and receiver operating characteristic (ROC) curve to evaluate the association between ePWV and AAC.

Results: The ePWV was significantly higher in participants with AAC compared with those without AAC. And ePWV had a high correlation with age and AAC (correlation coefficient = 0.906 and 0.332, both $P < 0.001$). Individuals in high ePWV group had significantly higher percentage of AAC compared to low ePWV group (OR = 2.971, 95% CI 2.529–3.490, $P < 0.001$) in the crude model. After adjusting for all confounding variables, ePWV was still significantly higher (Model 3, OR = 1.962, 95% CI 1.612–2.389, $P < 0.001$). While after adjusting for all confounding variables plus age (Model 4), ePWV, when as a categorical variable, was no longer significantly positively associated with AAC. Additionally, the ROC curve indicated that both ePWV and age had some diagnostic value for AAC (AUC = 0.690, $P < 0.001$; AUC = 0.708, $P < 0.001$).

Conclusions: In the age range of 40–80 years, ePWV did have an association with AAC but did not have predictive power beyond age.

Keywords: Pulse wave velocity, Estimated pulse wave velocity, Abdominal aortic calcification, NHANES, Arterial stiffness

1 Introduction

In recent years, growing studies have focused on the pathological mechanism of vascular calcification. However, most previous studies on vascular calcification mainly focused on coronary artery calcification, but with the development of biomarkers and the improvement of calcification assessment tools, the studies on abdominal aortic calcification (AAC) have gradually increased [1]. In the studies of the connection between cardiovascular diseases, chronic kidney disease, diabetes and osteoporosis, arterial calcification, particularly AAC, has a a pivotal position. AAC can be evaluated by several imaging tools, including dual-energy X-ray absorptiometry (DXA) and lateral abdominal X-ray [2, 3]. Each tool has its own disadvantages and advantages, among which DXA is a relatively fast, safe, easy to obtain and inexpensive way to evaluate AAC [4]. At present, the 8-point

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semiquantitative score (AAC-8) and 24-point semiquantitative score (AAC-24) from DXA are mainly used to evaluate the severity of AAC in clinical studies [4, 5]. AAC is an actively regulated pathological process, which is usually related to smoking, age, inflammation and metabolic dysregulation [2, 6]. Previous studies have shown that AAC is associated with coronary artery disease [7, 8], stroke [9], fracture [10], cardiovascular diseases mortality [7], all-cause mortality [11]. Therefore, it is essential to explore and intervene the controllable risk factors of AAC to prevent premature cardiovascular diseases and death.

The pulse wave velocity (PWV), as a noninvasive and easily available indicator, has been widely used to screen for arterial stiffness [12–14]. There is evidence that PWV is related to subclinical and clinical cardiovascular diseases, including AAC, and could independently predict future adverse cardiovascular events [15–19]. Therefore, PWV can be used as an effective measuring tool to screen participants with high risk factors of cardiovascular metabolism in general population. However, in a large-scale population health survey, it is still a time-consuming and labor-intensive work to measure PWV by a relatively expensive measuring tool. Accordingly, the method of estimating PWV based on the combination formula of age and mean blood pressure (MBP) was developed, namely ePWV [20]. And previous study showed that ePWV was highly correlated with PWV in the internal model building queue and the external verification queue, and could be used as a reliable alternative marker of PWV [20]. Since then, increasing scholars paid attention to the association between ePWV and cardiovascular diseases, and found that ePWV was independently associated with cardiovascular and cerebrovascular diseases and all-cause mortality [21–26]. And some studies also showed that compared with measured PWV, ePWV had similar or higher predictive value for cardiovascular events [27, 28].

However, as far as we know, although there is evidence that PWV is related to AAC [17], it has certain limitations because it is impossible to obtain PWV in epidemiological studies post hoc. Therefore, we used ePWV, an alternative index highly related to PWV, among adults from the National Health and Nutrition Examination Survey (NHANES) to explore the correlation with AAC, and to evaluate whether ePWV is an independent predictor of AAC.

2 Methods

2.1 Study Population

In this cross-sectional observational study, we followed the methods of Wang et al. in 2022, that is, multivariate logistic regression analysis, correlation analysis, forest map and receiver operating characteristic (ROC) [29]. After excluding participants with missing age, blood pressure and AAC score parameters, we finally enrolled 3140 adults between the ages of 40 and 80 who participated in the 2013–2014 NHANES, the specific details of which were available elsewhere [30]. The protocol was approved by the NCHS Research Ethics Review Board (Protocol #2011-17) and in accord with the Declaration of Helsinki, all participants signed written informed consent. The data cleaning algorithm was shown in Fig. 1.

2.2 Data Collection and Definitions

Using standardized interview questionnaire to collect the demographic information of each participant, including age, sex, race, smoking, hypertension and diabetes. We divided race into five groups: Mexican American, non-Hispanic White, non-Hispanic Black, other Hispanic and other races. We divided smoking into two groups according to whether smoked at least 100 cigarettes in a lifetime: absent and present. According to ADAs diabetes diagnostic criteria, diabetes was defined as self-reported diagnosis, or taking hypoglycemic drugs, or fasting plasma glucose (FPG) $\geq$ 7.0 mmol/L, or hemoglobin A1c (HbA1c) $\geq$ 6.5% [31]. Hypertension was defined as the systolic blood pressure (SBP) $\geq$ 140 mmHg and/or diastolic blood pressure (DBP) $\geq$ 90 mmHg or taking antihypertensive drugs [32]. Using standardized procedures to measure height, weight, SBP and DBP of each participant, and the body mass index (BMI, kg/m^2) was determined by a method, that is, weight (kg)/height (m)^2. Using the difference between SBP and DBP to calculate pulse pressure (PP). Using standardized operational procedures to collect blood sample of each participant for measurement of blood markers, including triglycerides (TGs), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), blood urea nitrogen (BUN), creatinine (CR), uric acid (UA), alkaline phosphatase (ALP), FPG, HbA1c, total calcium (Tca) and adjusted calcium (Adj-Ca). The Adj-Ca (mg/dL) = measured calcium (mg/dL) + 0.8 × albumin (g/dL) + 3.2 [33].

The ePWV was determined by using the formula described by Greve et al. [27], which was derived from the reference value of Collaboration Cohort [20]. The ePWV was estimated on the basis of age and MBP, that is, $ePWV = 9.587 - 0.402 \times age + 4.560 \times 10^{-3} \times age^2 - 2.621 \times 10^{-5} \times age^2 \times MBP + 3.176 \times 10^{-3} \times age \times MBP - 1.832 \times 10^{-2} \times MBP$. MBP was calculated as DBP + 0.4(SBP − DBP) [20]. In our seemingly healthy population, all participants were divided into two groups according to the median of ePWV: high ePWV ($\geq$ 8.98 m/s) and low ePWV (< 8.98 m/s).

NHANES professionals firstly detected calcium deposits in the abdominal aorta by DXA, and then scored the

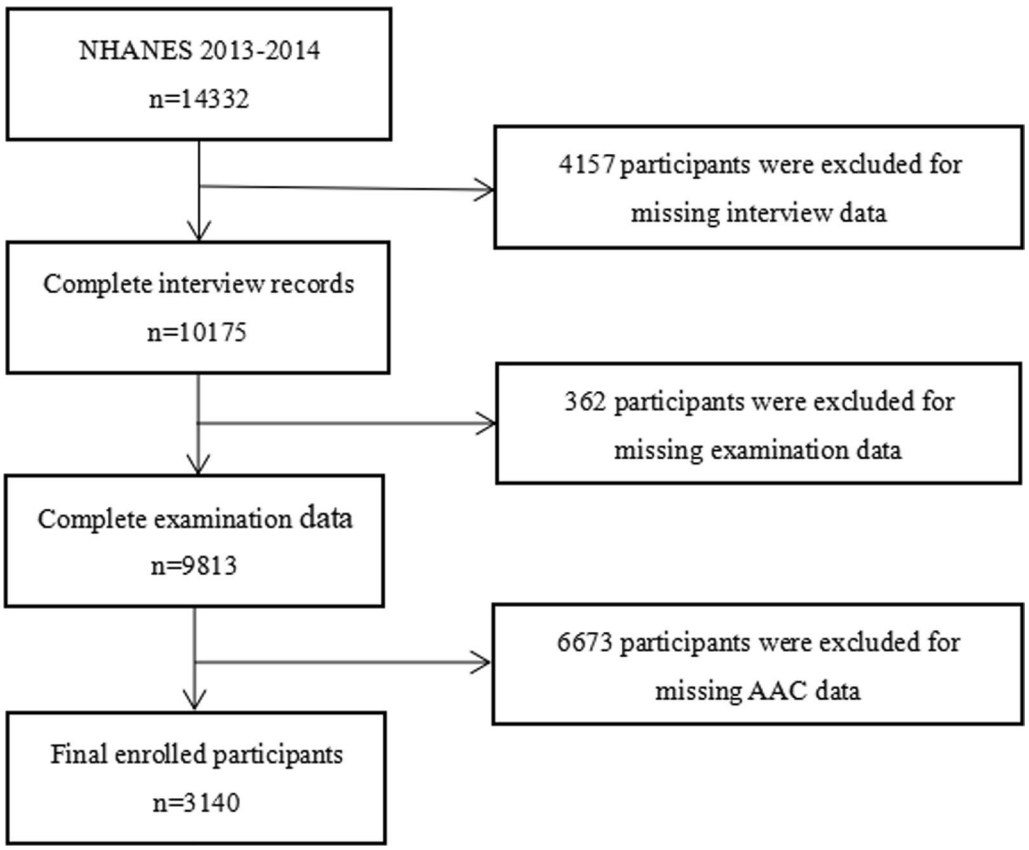

Fig. 1 Flow chart of selected participants. *NHANES* National Health and Nutrition Examination Survey, *AAC* abdominal aortic calcification

degree of calcification by using the semi-quantified Kauppila scoring system, which included 8-point and 24-point systems, detailed scoring criteria could be found in other literatures [4, 5]. In this study, we chosed a 24-point scale to determine the severity of AAC and divided all participants into two groups: non-AAC group (AAC score = 0) and AAC group (AAC score > 0).

2.3 Statistical Analysis

Continuous variables were showed as mean ± standard deviation or median (quartiles: Q1, Q3), categorical variables were showed as numbers (percentages). Using the independent-sample *t*-test or Mann–Whitney *U* test and Pearson chi-square test or fisher's exact test to assess the differences between groups. Using the Pearson correlation or spearman's rank to assess the correlations between ePWV and other covariates. Using the multivariate logistic regression and multivariate linear regression with four models to explore the association between ePWV and AAC. Crude model: unadjusted; Model 1: adjusted for race, smoking, hypertension and diabetes; Model 2: adjusted for race, smoking, hypertension, diabetes, BMI, TGs, TC, HDL-C, BUN, CR, UA, FPG, HbA1c and Adj-Ca; Model 3: adjusted for race, smoking, hypertension, diabetes, BMI, TGs, TC, HDL-C, BUN, CR, UA, FPG, HbA1c, Adj-Ca, SBP and DBP. Model 4: adjusted for variables included in Model 3 plus age. We also performed ROC analysis to evaluate the diagnostic performance of ePWV and other parameters for AAC. All Statistical tests were conducted by using MedCalc 19.1 and SPSS 26.0. A two-tailed *P* value < 0.05 was defined as statistically significant.

3 Results

3.1 Characteristics of Study Participants

The 3140 individuals (median age: 58.0 years; 48.3% men) included in this study were classified into two groups on the basis of the presence or absence of AAC: non-AAC (AAC score = 0) and AAC group (AAC score > 0). There were significant differences in races between groups ($P < 0.001$). The ePWV was higher in individuals with AAC than those without AAC ($P < 0.001$). Individuals with AAC were older, had higher percentages of smoking, diabetes and hypertension, higher levels of SBP and PP, but lower levels of BMI and DBP than those without AAC ($P < 0.001$). In terms of blood markers, TGs, BUN,

CR, UA, FPG, HbA1c, Tca and Adj-Ca were higher, while TC and HDL-C were lower among individuals with AAC than those free from AAC ($P<0.05$) (Table 1).

3.2 Associations Between ePWV and Covariates

In addition, we used the pearson correlation or spearman's rank analyses to test the associations between ePWV (as a continuous variable) and other covariates. The results showed that ePWV was positively related to age, smoking, diabetes, hypertension, SBP, DBP, PP, MBP, TGs, HDL-C, BUN, CR, UA, FPG, HbA1c, ALP, Tca, Adj-Ca and AAC, but negatively related to TC and LDL-C ($P<0.05$) (Table 2).

3.3 Associations Between ePWV and AAC

As shown in Table 3 and Fig. 2, multivariate logistic regression analyses showed that when ePWV was viewed as categorical variable, Individuals in high ePWV group had significantly higher percentage of AAC compared to low ePWV group (OR = 2.971, 95% CI 2.529–3.490, $P<0.001$) in the crude model. After gradually adjusting for the confounding variables, the risk of participants who suffered from AAC declined step by step in high ePWV group, but it was still significantly higher than that of participants with low ePWV (Model 1, 2 and 3: OR = 2.503, 95% CI 2.109–2.970, $P<0.001$; OR = 2.271, 95% CI 1.903–2.711, $P<0.001$; OR = 1.962, 95% CI 1.612–2.389, $P<0.001$; respectively). Additionally, multivariate linear regression analysis showed that after adjusting for the covariables contained in Model 3, the AAC score increased by 0.606 points for each additional unit of ePWV ($\beta = 0.606$, 95% CI 0.529–0.684, $P<0.001$) (Table 4). While after adjusting for all confounding variables plus age (Model 4), ePWV, when as a categorical

Table 1 Baseline characteristics of participants with and without AAC

Variables	Total population	Non-AAC ($n=2193$)	AAC ($n=947$)	*P* value
Age (years)	58.0 (48.0, 68.0)	55.0 (46.0, 64.0)	66.0 (56.0, 75.0)	< 0.001
Male	1518 (48.3%)	1045 (47.7%)	473 (49.9%)	0.237
Race				< 0.001
Non-Hispanic white	1375 (43.8%)	869 (39.6%)	506 (53.4%)	
Non-Hispanic black	620 (19.7%)	465 (21.2%)	155 (16.4%)	
Mexican–American	412 (13.1%)	316 (14.4%)	96 (10.1%)	
Other Hispanic	298 (9.5%)	225 (10.3%)	73(7.7%)	
Other races	435 (13.9%)	318 (14.5%)	117 (12.4%)	
Smoking history	1452(46.2%)	940 (42.9%)	512 (54.1%)	< 0.001
Diabetes	648 (20.6%)	403 (18.4%)	245 (25.9%)	< 0.001
Hypertension	1486 (47.3%)	910 (41.5%)	576 (60.8%)	< 0.001
BMI (kg/m^2)	28.4 ± 5.6	28.8 ± 5.8	27.7 ± 4.8	< 0.001
SBP (mm Hg)	127.2 ± 18.3	125.4 ± 17.5	131.6 ± 19.2	< 0.001
DBP (mm Hg)	71.3 ± 10.8	72.3 ± 10.6	68.9 ± 10.8	< 0.001
PP (mm Hg)	56.0 ± 17.5	53.1 ± 15.9	62.6 ± 19.2	< 0.001
MBP (mm Hg)	93.7 ± 11.4	93.5 ± 11.4	94.0 ± 11.4	0.324
EPWV (m/s)	9.3 ± 2.0	8.7 ± 1.8	10.3 ± 2.1	< 0.001
TGs (mg/dL)	132.0 (86.0, 192.8)	125.0 (82.0, 185.0)	134.0 (93.5, 195.5)	0.003
TC (mg/dL)	196.0 ± 42.7	197.5 ± 42.0	192.6 ± 44.2	0.003
LDL-C (mg/dL)	114.8 ± 36.0	115.7 ± 35.2	112.9 ± 37.7	0.173
HDL-C (mg/dL)	54.1 ± 16.5	54.5 ± 16.8	53.0 ± 15.6	0.016
BUN (mg/dL)	14.3 ± 6.2	13.7 ± 5.4	15.7 ± 7.5	< 0.001
CR (mg/dL)	0.9 (0.7, 1.0)	0.9 (0.7, 1.0)	0.9 (0.8, 1.1)	< 0.001
UA (mg/dL)	5.5 ± 1.4	5.4 ± 1.3	5.6 ± 1.4	< 0.001
FPG (mg/dL)	98.0 (90.0, 110.0)	97.0 (89.0, 110.0)	101.0 (92.0, 118.0)	< 0.001
HbA1c (%)	5.7 (5.4, 6.0)	5.6 (5.3, 5.9)	5.8 (5.4, 6.2)	< 0.001
ALP (IU/L)	65.0 (53.0, 77.0)	65.0 (53.0, 77.0)	66.0 (54.0, 77.0)	0.301
Tca (mg/dL)	9.5 ± 0.4	9.4 ± 0.4	9.5 ± 0.4	0.008
Adj-Ca (mg/dL)	9.3 ± 0.3	9.2 ± 0.3	9.3 ± 0.3	< 0.001

AAC abdominal aortic calcification, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *PP* pulse pressure, *MBP* mean blood pressure, *ePWV* estimated pulse wave velocity, *TGs* triglycerides, *TC* total cholesterol, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *BUN* blood urea nitrogen, *CR* creatinine, *UA* uric acid, *FPG* fasting plasma glucose, *HbA1c* hemoglobin A1c, *ALP* alkaline phosphatase, *Tca* total calcium, *Adj-Ca* adjusted calcium

Table 2 Associations between ePWV (as a continuous variable) and covariates

	Correlation coefficient	*P* value
Age	.906	< 0.001
Smoking history	.033	0.023
Diabetes	.126	< 0.001
Hypertension	.302	< 0.001
SBP	.632	< 0.001
DBP	.104	< 0.001
PP	.602	< 0.001
MBP	.457	< 0.001
TGs	.044	0.013
TC	− .070	< 0.001
LDL-C	− .127	< 0.001
HDL-C	.046	0.009
BUN	.305	< 0.001
CR	.217	< 0.001
UA	.158	< 0.001
FPG	.171	< 0.001
HbA1c	.269	< 0.001
ALP	.103	< 0.001
Tca	.074	< 0.001
Adj-Ca	.157	< 0.001
AAC	.332	< 0.001

ePWV estimated pulse wave velocity, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *PP* pulse pressure, *MBP* mean blood pressure, *TGs* triglycerides, *TC* total cholesterol, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *BUN* blood urea nitrogen, *CR* creatinine, *UA* uric acid, *FPG* fasting plasma glucose, *HbA1c* hemoglobin A1c, *ALP* alkaline phosphatase, *Tca* total calcium, *Adj-Ca* adjusted calcium, *AAC* abdominal aortic calcification

Table 3 Assocaition between ePWV and AAC (categorical models)

	OR	95% CI	*P* value
Crude model	2.971	2.529–3.490	< 0.001
Model 1	2.503	2.109–2.970	< 0.001
Model 2	2.271	1.903–2.711	< 0.001
Model 3	1.962	1.612–2.389	< 0.001
Model 4	0.716	0.529–0.969	0.031

The OR was tested by viewing low ePWV as reference

Crude model: unadjusted; Model 1: adjusted for race, smoking, hypertension and diabetes; Model 2: adjusted for race, smoking, hypertension, diabetes, body mass index, triglycerides, total cholesterol, high-density lipoprotein cholesterol, blood urea nitrogen, creatinine, uric acid, fasting plasma glucose, hemoglobin A1c and adjusted calcium; Model 3: adjusted for variables included in Model 2 and systolic blood pressure and diastolic blood pressure; Model 4: adjusted for variables included in Model 3 plus age

variable, was no longer significantly positively associated with AAC (Tables 3), but when used as a continuous variable, ePWV still had a positive correlation with AAC (Tables 4). In addition, the ROC curve analysis indicated that ePWV, age, SBP, PP and MBP had certain diagnostic performance for AAC, among which ePWV and age had better performance (AUC = 0.690 and 0.708) (Fig. 3).

4 Discussion

For all we know, we found that in the age range of 40–80 years, ePWV did have an association with AAC but did not have predictive power beyond age for the first time. This study disclosed that there were statistical differences in several clinical features between non-AAC and AAC groups, and individuals with traditional cardiovascular metabolic risk factors were more likely to suffer from AAC. Furthermore, we also found that after adjusting for confounding variables, the higher ePWV was independently related to higher risk of AAC. While after adjusting for all confounding variables plus age, ePWV, when as a categorical variable, was no longer significantly positively associated with AAC, but when used as a continuous variable, ePWV still had a positive correlation with AAC. And we also found that ePWV had a moderate diagnostic performance for AAC.

The Study of Osteoporotic Fractures demonstrated that individuals who suffered from AAC were older, had higher percentages of smoking and diabetes and higher SBP than those without AAC [10]. In recent years, two articles published in the Nephrol Dial Transplant have also shown that participants with AAC were older and had higher percentage of diabetes than those without AAC, and among them, Chen et al. have shown that participants with AAC had higher levels of HbA1c and estimated glomerular filtration rate (eGFR), higher percentages of smoking and hypertension [34, 35], which was principally in accordance with our study. In addition, our study also found that participants with AAC had higher levels of ePWV and other metabolic parameters. In a word, the above studies showed that participants with traditional cardiovascular metabolic risk factors were more likely to suffer from AAC.

Moreover, previous study have shown that measured PWV, as a worthy marker of arterial stiffness, was positively correlated with age, SBP and length of AAC, and PWV was considered to be a useful predictor of AAC [17]. Similarly, Lioufas et al. also reported that higher PWV was related to elder, diabetes, SBP, ALP and presence of AAC [36]. The same was true of our study with ePWV.

And since the advent of ePWV, many studies showed that ePWV, similar to the predictive performance of PWV, could also independently predict subclinical and clinical cardiovascular diseases. For instance, HSU et al. found that ePWV was an independent risk factor for long-term cardiogenic and all-cause death of patients with cardiovascular diseases whether in univariable or

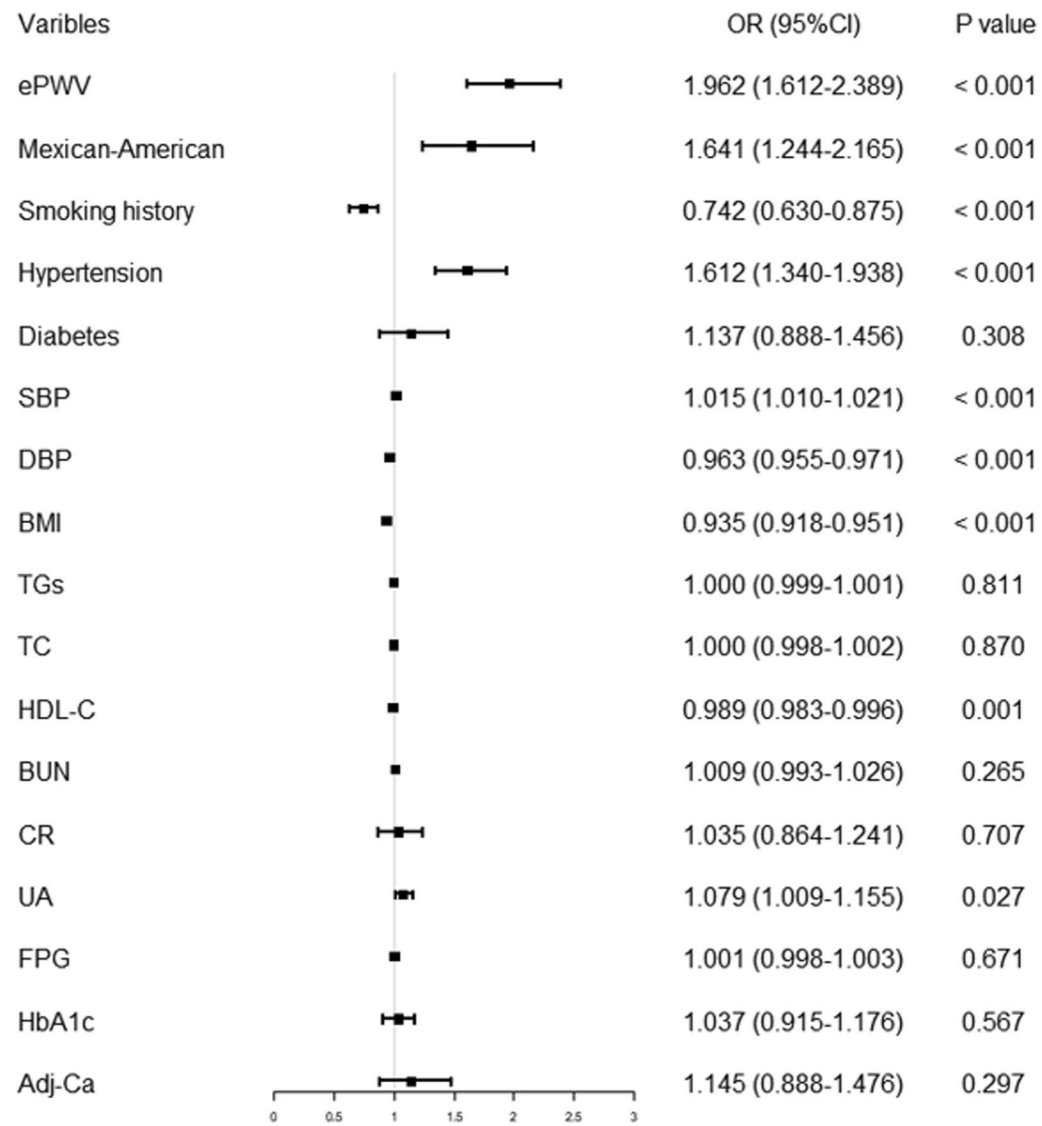

Fig. 2 Forest map of model 3. After adjusting for race, smoking, hypertension, diabetes, SBP, DBP, BMI, TGs, TC, HDL-C, BUN, CR, UA, FPG, HbA1c and Adj-Ca in Model 3, higher ePWV was still associated with a higher risk of AAC. The OR was tested by viewing low ePWV as reference. *ePWV* estimated pulse wave velocity, *AAC* abdominal aortic calcification, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BMI* body mass index, *TGs* triglycerides, *TC* total cholesterol; *HDL-C* high-density lipoprotein cholesterol, *BUN* blood urea nitrogen, *CR* creatinine, *UA* uric acid, *FPG* fasting plasma glucose, *HbA1c* hemoglobin A1c, *Adj-Ca* adjusted calcium, *OR* odds ratio, *CI* confidence interval

multivariable analysis, and they also found ePWV had higher predictive value for cardiac death than measured PWV [28]. Besides, Vishram-Nielsen et al. discovered that high ePWV, independent of Framingham Risk Score and systematic coronary risk evaluation, was associated with main end-point events including mortality and cardiovascular morbidity in multivariable cox regression analysis [24], which was basically consistent with a secondary analysis by Vlachopoulos et al., that is, in the Sprint population, ePWV is independent of Framingham risk score to predict the main outcome and all-cause death [21]. Moreover, the Kuopio Ischemic Heart Disease Cohort Study showed that higher ePWV was independently associated with the increased risk of stroke in middle-aged men [23]. And we also found that ePWV had some diagnostic value for AAC. Therefore, ePWV was worthy of being estimated for prediction of AAC and other cardiovascular diseases.

Although our study had achieved encouraging results, there were still several shortcomings. First of all, our study failed to determine the causal association between ePWV and AAC. In addition, due to the lack

Table 4 Association between ePWV and AAC (continuous models)

	β	95% CI	*P* value
Crude model	0.630	0.574–0.686	< 0.001
Model 1	0.570	0.510–0.630	< 0.001
Model 2	0.505	0.442–0.568	< 0.001
Model 3	0.606	0.529–0.684	< 0.001
Model 4	2.009	1.625–2.392	< 0.001

Crude model: unadjusted; Model 1: adjusted for race, smoking, hypertension and diabetes; Model 2: adjusted for race, smoking, hypertension, diabetes, body mass index, triglycerides, total cholesterol, high-density lipoprotein cholesterol, blood urea nitrogen, creatinine, uric acid, fasting plasma glucose, hemoglobin A1c and adjusted calcium; Model 3: adjusted for variables included in Model 2 and systolic blood pressure and diastolic blood pressure; Model 4: adjusted for variables included in Model 3 plus age

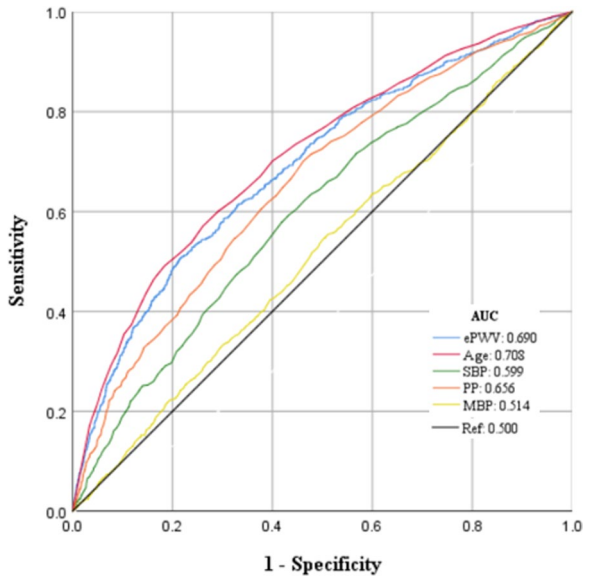

Fig. 3 Receiver operating characteristic (ROC) curve evaluating predictive effects of ePWV, age, SBP, PP and MBP on AAC

of PWV data, we could not compare the difference of predictive efficacy of ePWV and PWV for AAC, so it was impossible to know whether ePWV could replace PWV as a predictive biomarker for AAC. Moreover, we only considered some common confounding factors and might miss other potential risk factors, such as nutrition, diet, drugs and genetic susceptibility. Additionally, this study only included adults aged 40–80 from NHANES, and the weight of the survey sample was not taken into account in the analysis, so our findings might not be extended to other races and populations.

5 Conclusion

IN conclusion, our study showed that there was a significant correlation between ePWV and AAC among adults aged 40–80, which not only expanded the research field of ePWV, but also filled in the knowledge gap of the correlation study between ePWV and AAC, and provided new ideas for preventing and intervening premature cardiovascular diseases.

Abbreviations

PWV: Pulse wave velocity; AAC: Abdominal aortic calcification; ePWV: Estimated pulse wave velocity; ROC: Receiver operating characteristic; DXA: Dual-energy X-ray absorptiometry; MBP: Mean blood pressure; NHANES: National Health and Nutrition Examination Survey; FPG: Fasting plasma glucose; HbA1c: Hemoglobin A1c; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; BMI: Body mass index; PP: Pulse pressure; TGs: Triglycerides; TC: Total cholesterol; HDL-C: High-density lipoprotein cholesterol; BUN: Blood urea nitrogen; CR: Creatinine; UA: Uric acid; ALP: Alkaline phosphatase; Tca: Total calcium; Adj-Ca: Adjusted calcium; eGFR: Estimated glomerular filtration rate.

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Author Contributions

XXG and CZX designed the study, collected and analyzed the statistics, and wrote the manuscript. YQL made contribution to the writing. XXG coordinated and supervised data collection, and reviewed the manuscript. All authors read and approved the final manuscript.

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None.

Availability of Data and Materials

The data and materials used in this study are available on NHANES website.

Declarations

Ethical Approval and Informed Consent

The protocol was approved by the National Center for Health Statistics of the Center for Disease Control and Prevention Institutional Review Board (Protocol #2011-17), all participants provided written informed consent.

Consent for Publication

Not applicable.

Competing interest

The authors have no conflicts of interest to disclose.

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