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Association between vascular aging and cognitive function in Chinese adults

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Abstract

Background Vascular health has been associated with cognition but related evidence is limited in Chinese. The objective of this study was to examine the association of vascular aging assessed by arterial stiffness and blood pressure with cognitive function in an unselected Chinese population.

Methods In the Tianning Cohort ($N=5158$), indicators of arterial stiffness and blood pressure including carotid-femoral pulse wave velocity (cfPWV), ankle-brachial index (ABI), pulse pressure (PP), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were measured. Cognitive function was assessed using the Mini Mental State Examination (MMSE) questionnaire. We applied Poisson regression and logistic regression to examine the associations of vascular aging and blood pressure with cognitive function.

Results 76 (1.47%) participants had impaired cognitive function diagnosed by a MMSE score of less than 24 points. Participants with a higher level of PP were more likely to have a decreased score of MMSE (β=-0.0121, $P<0.001$ for log-transformed pulse pressure) and a higher risk of having impaired cognitive function (OR = 5.95, 95%CI: 2.02–17.79, $P<0.001$ for log-transformed PP). Per standard deviation increment in SBP was significantly associated with lower MMSE score (β=-0.0020, $P<0.001$) and impaired cognitive function (OR = 1.69, 95%CI: 1.38–2.06, $P<0.001$). No significant associations were found regarding other parameters.

Conclusions Blood pressure and hypertension were associated with cognitive function in Chinese adults. PP may be a potential predictor for impaired cognitive function.

Keywords Arterial stiffness, Blood pressure, Cognitive impairment, Chinese

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Introduction

As the population ages, more and more middle-aged and older adults have suffered from cognitive impairment including mild cognitive impairment (MCI) and dementia, which has already threatened public health and brought a tremendous economic burden to society worldwide [1]. Therefore, searching for more risk factors of cognitive impairment is of great significance for the prevention and management of this debilitating disorder. Recently, along with the in-depth study in vascular aging, we can see that it is not only related to increased risk of cardiovascular diseases [2] but also involved in the changes in cognitive function [3]. The stiffening and loss of recoil in the aorta would transmit excessive and damaging pulsatile load to the peripheral arteries of body organs. The brain is theoretically more vulnerable to pulsatile damage due to its low resistance and high flow characteristics [4]. Vascular aging can increase blood pressure and arterial stiffness, affecting left ventricular mass and blood flow [5], and potentially contributing to cognitive impairment [6]. Further, stiffer arteries can also impact the response to antihypertensive treatment [7, 8] with uncontrolled hypertension being the leading risk factor for dementia. Experimental studies have also demonstrated that vascular aging may disrupt the communication between vascular pericytes and neighboring brain cells, leading to cognitive impairment and central nervous system diseases [9–17]. In population studies, cognitive function has also been related to vascular aging assessed by various parameters of arterial stiffness and blood pressure such as carotid-femoral pulse wave velocity (cfPWV), pulse pressure (PP), ankle-brachial index (ABI), cardio-ankle vascular index (CAVI), augmentation index (AI), flow-mediated dilation (FMD), and carotid intima-media thickness (IMT) [18–27]. However, not all studies revealed the same result [26, 28] and most studies were mainly conducted in European populations with a relatively small sample size. To date, we are aware that there are few large-sample studies on the association between vascular aging and cognitive function in Chinese adults who have different risk profiles from Europeans [29]. Therefore, the main objective of this study was to examine the association of vascular aging assessed by arterial stiffness and blood pressure with cognitive function in a large population of Chinese adults.

Methods

Study participants

The Tianning Cohort is a community-based prospective cohort study, designed to search for new risk factors and potential therapeutic targets of cardiovascular disease (CVD) in Chinese adults. The protocols of the Tianning Cohort were approved by the Ethics Committee of Soochow University (approval No. ECSU-201800051) and performed in accordance with the Declaration of Helsinki. The study design, survey methods, and participant recruitment have been detailed elsewhere [30]. At baseline examination conducted between May and November 2018, a total of 5,199 participants were recruited from randomly selected communities after signing a written informed consent. They received face-to-face interviews, physical examinations, and collection of blood and urine samples. After excluding participants with missing data on vascular aging or cognitive function (N=41), 5158 participants were included in the current analysis.

Assessment of cognitive function

Cognitive function was assessed using the Mini-Mental State Examination (MMSE) questionnaire by a professional doctor The MMSE was a common test of cognitive function with established validity and reliability [31]. It assessed cognitive function in five components, including orientation, registration, attention and calculation, recall, and language. The total score ranges from 0 to 30 points, with a higher score denoting better cognitive function. A score of <24 points was recognized as the presence of cognitive impairment or dementia. This definition has been widely used in prior population studies [32–37].

Measurement of blood pressure

According to standard procedures, the blood pressure was measured three times by trained staff using a digital blood pressure measuring device (HBP-1320, Omron, Japan) with an appropriate cuff size, after the participants had been resting for at least five minutes in a relaxed sitting position. All participants were required to avoid exercise, smoking, and drinking alcohol and tea for at least 30 min before the measurement. The average of the three measurements was used as the levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP) for each participant. Mean arterial pressure (MAP) was calculated using the following formula: MAP = (SBP+DBP ×2)/3. PP was calculated by subtracting DBP from SBP.

Measurement of arterial stiffness

Arterial stiffness was assessed by cfPWV and ABI in our study. cfPWV was measured by an effective arterial stiffness analyzer AS-2000 equipment (Hong Kong Biomedical Holdings Co., Ltd., Hong Kong, China). After the participants rested for at least 15 min, a well-trained technician placed a pressure cuff on the carotid and femoral arteries of the participants in a supine position. The device used oscillometric cuff technology to measure the pulse waves of the carotid and femoral arteries at the same time. It estimated the path of travel by subtracting the distance of the carotid artery-sternal notch

from the femoral artery-sternal notch and automatically calculates cfPWV (cfPWV = path of travel / the time difference between pulse waves transmitted to the carotid artery and femoral artery). In the meantime, SBP from both brachial arteries and from both the dorsalis pedis and posterior tibial arteries was measured. The ABI value is determined by taking the higher pressure of the two arteries at the ankle, divided by the brachial arterial systolic pressure. In calculating the ABI, the higher of the two brachial systolic pressure measurements is used.

Collection of conventional risk factors

Data on age, sex, education level (years of under-education), disease history, and lifestyles were obtained by standard questionnaires administered by trained staff through face-to-face interviews. Using the key questions from the Global Adult Tobacco Survey (GATS), smoking status was classified as current, former, and never smoking. Current smoking was defined as having smoked at least 100 cigarettes in the subject's entire life, having smoked cigarettes regularly, and smoking currently. Former smoking was defined as having smoked at least 100 cigarettes in the subject's entire life, having smoked cigarettes regularly in the past, and not smoking currently. Never smoking was defined as never smoked or having smoked fewer than 100 cigarettes in their lifetime. Alcohol consumption was classified as current drinking or not. Current drinking was defined as having consumed alcohol ≥ 12 times in the past year and drinking currently. Physical activity was determined using the Global Physical Activity Questionnaire (GPAQ) which was developed by the WHO for physical activity surveillance in developing countries [38]. It collects information on physical activity at work, commuting, and recreational activities as well as sedentary behavior. The measured data were processed according to the GPAQ Analysis Guide, and MET-minutes per week values were calculated and used in data analysis. Sleep was assessed by a total score of the Pittsburgh Sleep Quality Index (PSQI), a widely used and well-validated measure of sleep quality [39]. Body weight (kg) and height (cm) were measured using a regularly calibrated stadiometer and balance-beam scale with participants wearing light clothes and no shoes by trained staff. Body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in meters (kg/m^2). Fasting glucose and blood lipids including total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were measured using commercial reagents (Siemens Healthcare Diagnostic Inc., Co Antrim, UK) at the Key Laboratory of Geriatric Disease Prevention and Translational Medicine of Jiangsu Province. The laboratory staff was unaware of the health status of participants.

Statistical analysis

The baseline characteristics of study participants were presented in participants with normal and impaired cognitive function, respectively. Parameters of arterial stiffness (cfPWV, ABI) and blood pressure (SBP, DBP, MAP, PP) were compared between the two groups using the Mann-Whitney U test and Student's t-test, respectively, considering their data distribution. Log transformation was applied to maximize the normality of data distribution of cfPWV, ABI, and PP, and the generated values were used in downstream analyses. To examine the association between vascular aging and cognitive function, we constructed a Poisson regression model in which MMSE total score was the dependent variable and each parameter (continuous or categorized as quartiles for cfPWV, ABI, SBP, DBP, MAP, and PP) was the independent variable individually, adjusting for age, sex, educational level, cigarette smoking, alcohol consumption, physical activity, sleep quality, fasting glucose, LDL-C, and HDL-C. The rationale for using the Poisson regression model was to account for the count data of the MMSE score. To ease data interpretation, the association between vascular aging and the risk of having impaired cognitive function was also similarly examined by constructing a binary logistic regression model with impaired cognitive function as the dependent variable. The false-discovery rate (FDR) approach was applied to correct potential multiple testing errors by adjusting for the total number of parameters of vascular aging. To examine whether arterial stiffness and blood pressure act synergistically on cognitive function, their interactive effect on cognitive function was examined in both additive scale and multiplicative scale using Poisson regression and logistic regression models, respectively. Receiver operating characteristics (ROC) curves were used to compare the prediction performance for impaired cognitive function.

Sensitivity analysis

Sex-specific associations of arterial stiffness and blood pressure with cognitive function were additionally examined. To examine whether disease history affects our results, participants with a disease history of hypertension, diabetes, and CVD were excluded, respectively. All statistical analyses were using R version 4.2.2. A two-tailed $P < 0.05$ was considered statistically significant.

Results

Characteristics of study participants

A total of 5158 participants (mean aged 50.9 ± 15.6 years, aged 18–96 years, 41.5% males) were included in the current study. Of them, 76 (1.47%) participants had impaired cognitive function diagnosed by a MMSE score of less than 24 points. The characteristics of study participants are shown in Table 1. Participants with impaired

Table 1 Characteristics of study participants according to the status of cognitive function

Characteristics	MMSE total score		*P*-value
	≥ 24	< 24	
No. of participants	5082	76	
Age, years	50.7 ± 15.6	65.0 ± 10.1	< 0.001
Sex, females (%)	2966 (58.4)	53 (69.7)	0.047
Educational level, years	10.8 ± 3.9	6.5 ± 3.1	< 0.001
Cigarette smoking, *n* (%)			0.317
Current smoking	1018 (20.0%)	13 (17.1%)	
Past smoking	310 (6.1%)	1 (1.3%)	
Never smoking	3754 (73.9%)	62 (81.6%)	
Current drinking, *n* (%)	946 (18.6)	14 (18.4)	1.000
Disease history, *n* (%)			
CVD	259(5.1)	4(5.3)	0.795
Hypertension	1603(31.5)	43(56.6)	< 0.001
Diabetes	365(7.2)	4(5.3)	0.657
MMSE total score, point	29.0 ± 0.4	19.0 ± 3.8	< 0.001
Body mass index, kg/m^2	24.06 ± 3.66	24.13 ± 3.75	0.882
Total cholesterol, mmol/L	1.77 ± 1.24	1.62 ± 1.05	0.277
Triglycerides, mmol/L	4.81 ± 0.94	4.60 ± 0.80	0.062
HDL cholesterol, mmol/L	1.22 ± 0.31	1.23 ± 0.24	0.769
LDL cholesterol, mmol/L	2.76 ± 0.79	2.60 ± 0.74	0.093
Glucose, mmol/L	5.12 ± 1.46	4.96 ± 0.84	0.352
Physical activity, MET-minutes/week	5090.5 ± 8052.5	2332.0 ± 3055.6	< 0.001
PSQI total score, point	3.7 ± 3.2	3.2 ± 3.5	0.160

Results were expressed with mean ± SD unless otherwise noted.

MMSE: Mini Mental State Examination; CVD: cardiovascular disease; HDL: high-density lipoprotein; LDL: low-density lipoprotein; PSQI: Pittsburgh Sleep Quality Index; SD: standard deviation;

cognitive function were more likely to be older, females, less educated, physically inactive, and had a history of hypertension than those without (all $P<0.05$). No significant difference in other variables listed was found between the two groups.

Association between arterial stiffness and cognitive function

The levels of parameters of arterial stiffness including cfPWV and ABI in participants with and without impaired cognitive function are shown in Fig. 1. We failed to observe a statistically significant group difference in cfPWV (median: 6.94 m/s vs. 6.72 m/s, $P=0.773$) and ABI (median: 1.08 vs. 1.11, $P=0.172$). Their associations with cognitive function were not significant in either the Poisson regression model or the logistic regression model (Table 2).

Association between blood pressure and cognitive function

The levels of SBP, DBP, MAP, and PP in participants with and without impaired cognitive function are shown in Fig. 1. The mean level of SBP (134.0 mmHg vs. 124.0 mmHg, $P<0.001$) and median level of PP (56.5 mmHg vs. 45.3 mmHg, $P<0.001$) were significantly higher in participants with impaired cognitive function than those without, whereas no group-difference in DBP (mean: 75.05 mmHg vs. 76.70 mmHg, $P=0.164$) and MAP (mean: 94.71 mmHg vs. 92.46 mmHg, $P=0.089$) was found. After adjusting for potential confounders collected, participants with a higher level of SBP (β=-0.0020, $P<0.001$ for per-SD increment) and PP (β=-0.0121, $P<0.001$ for log-transformed PP) were more likely to have a decreased score of MMSE (Table 3). Compared to participants with the lowest quartile, those with the highest quartile of SBP and PP had 0.0096 and 0.0055 points, respectively, of decrement in the MMSE score (Table 3).

Logistic regression revealed similar results (Table 3). A higher level of SBP (OR=1.69, 95%CI: 1.38–2.06, $P<0.001$ for per-SD increment) and PP (OR=5.95, 95%CI: 2.02–17.79, $P<0.001$ for log-transformed PP) was significantly associated with a higher risk of having impaired cognitive function (Table 3). Compared to participants with the lowest quartile of SBP, those with the highest had 4.39 times the risk of developing impaired cognitive function (OR=4.39, 95%CI: 2.13–10.26, $P<0.001$). Compared to participants with the lowest quartile of PP, those with the highest had 5 times the risk of developing impaired cognitive function (OR=5.00, 95%CI: 2.54–11.01, $P<0.001$). We failed to observe any statistically significant associations of DBP and MAP with cognitive function.

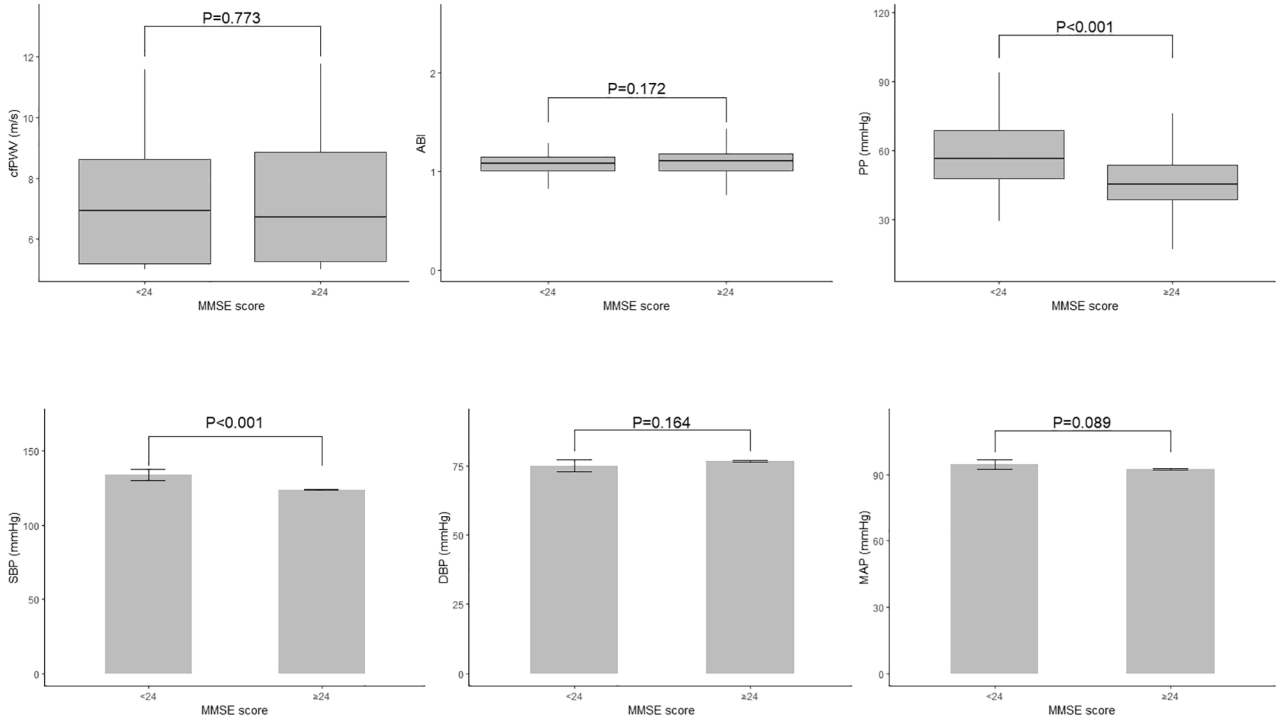

Fig. 1 The average levels of parameters of arterial stiffness and blood pressure according to cognitive function. **Legend**: Box plots show the median levels of cfPWV, ABI, and PP. Historical plots show the mean levels of blood pressure including SBP, DBP, and MAP. Impaired cognitive function was defined as an MMSE score of less than 24 points. MMSE: Mini-Mental State Examination; cfPWV: carotid–femoral Pulse Wave Velocity; ABI: Ankle Brachial Index; PP: Pulse Pressure; SBP: Systolic Blood Pressure; DBP: Diastole Blood Pressure; MAP: Mean Arterial Pressure

Table 2 Association between arterial stiffness and cognitive function

Parameters of arterial stiffness	MMSE score			Impaired cognitive function		
	β (SE)*	*P*-value	PDR-*P*	OR (95%CI) *	*P*-value	FDR-*P*
cfPWV						
Quartiles						
Q1	reference	-		reference	-	
Q2	-0.0015 (0.0019)	0.419		0.75 (0.37–1.46)	0.394	
Q3	-0.0010 (0.0019)	0.601		1.10 (0.60–2.05)	0.754	
Q4	-0.0027 (0.0019)	0.160		0.95 (0.50–1.80)	0.878	
Log-cfPWV	-0.0017 (0.0022)	0.434	0.651	0.94 (0.43–1.99)	0.879	0.994
ABI						
Quartiles						
Q1	reference	-		reference	-	
Q2	-0.0027 (0.0019)	0.150		1.59 (0.89–2.88)	0.121	
Q3	0.0000 (0.0018)	0.995		0.88 (0.45–1.68)	0.691	
Q4	-0.0000 (0.0019)	0.986		0.68 (0.32–1.38)	0.292	
Log-ABI	-0.0019 (0.0036)	0.605	0.726	1.00 (0.38–3.89)	0.994	0.994

*Adjusting for age, sex, educational level, cigarette smoking, alcohol consumption, physical activity, sleep quality, fasting glucose, and low- and high-density lipoprotein cholesterol

MMSE: Mini-Mental State Examination; cfPWV: carotid-femoral pulse wave velocity; ABI: ankle-brachial index; Q: quartiles; FDR: false-discovery rate

Interaction between arterial stiffness and blood pressure on cognitive function

Among the parameters of arterial stiffness and blood pressure, only PP and SBP seemed to be associated with cognitive function, as suggested by the above results. Further interaction analysis found that participants with elevated PP (≥60 mmHg) alone or complicated with elevated SBP (≥140 mmHg) were more likely to have impaired cognitive function (all $P<0.001$, Table 4), compared to those with normal PP and SBP. Although the

Table 3 Interaction between PP and SBP on cognitive function

Parameters of blood pressure	MMSE score			Impaired cognitive function		
	β (SE) *	*P*-value	PDR-*P*	OR (95%CI) *	*P*-value	PDR-*P*
SBP						
Quartiles						
Q1	reference	-		reference	-	
Q2	-0.0028 (0.0018)	0.129		1.93 (0.84–4.81)	0.134	
Q3	-0.0050 (0.0018)	0.006		2.55 (1.61–6.17)	0.026	
Q4	-0.0096 (0.0019)	< 0.001		4.39 (2.13–10.26)	< 0.001	
Per 1-SD	-0.0020 (0.0007)	0.008	0.024	1.69 (1.38–2.06)	< 0.001	0.003
DBP						
Quartiles						
Q1	reference	-		reference	-	
Q2	0.0031 (0.0018)	0.097		0.59 (0.30–1.12)	0.111	
Q3	-0.0001 (0.0018)	0.975		0.98 (0.55–1.75)	0.958	
Q4	0.0019 (0.0019)	0.300		0.53 (0.26–1.01)	0.062	
Per 1-SD	0.0011 (0.0007)	0.111	0.222	0.85 (0.67–1.07)	0.164	0.246
MAP						
Quartiles						
Q1	reference	-		reference	-	
Q2	0.0003 (0.0019)	0.869		0.68 (0.29–1.56)	0.361	
Q3	-0.0032 (0.0020)	0.107		1.11 (0.55–2.32)	0.779	
Q4	-0.0010 (0.0021)	0.613		1.02 (0.50–2.15)	0.955	
Per 1-SD	-0.0002 (0.0007)	0.818	0.818	0.84 (0.75–1.26)	0.089	0.178
PP						
Quartiles						
Q1	reference	-		reference	-	
Q2	0.0016 (0.0019)	0.400		0.84 (0.30–2.27)	0.737	
Q3	-0.0004 (0.0019)	0.833		2.11 (0.98–4.95)	0.068	
Q4	-0.0055 (0.0021)	0.009		5.00 (2.54–11.01)	< 0.001	
Log-PP	-0.0121 (0.0030)	< 0.001	0.006	5.95 (2.02–17.79)	< 0.001	0.003

*Adjusting for age, sex, educational level, cigarette smoking, alcohol consumption, physical activity, sleep quality, fasting glucose, and low- and high-density lipoprotein cholesterol

MMSE: Mini-Mental State Examination; SBP: systolic blood pressure; DBP: diastole blood pressure; MAP: mean arterial pressure; PP: pulse pressure; Q: quartiles; FDR: false-discovery rate

Table 4 Interaction between PP and SBP on cognitive function

Categories		Cases/*n*	MMSE score*		Impaired cognitive function*	
PP ≥ 60	SBP ≥ 140		β (SE)	*P*-value	OR (95%CI)	*P*-value
(-)	(-)	39/4053	reference	-	reference	-
(-)	(+)	3/356	0.0024 (0.0026)	0.358	0.87 (0.21–2.43)	0.824
(+)	(-)	9/213	-0.0064 (0.0035)	0.067	4.54 (2.04–9.09)	< 0.001
(+)	(+)	25/536	-0.0106 (0.0024)	< 0.001	5.04 (2.99–8.34)	< 0.001
PP ≥ 60 × SBP ≥ 140		76/5158	-0.0066 (0.0047)	0.157	1.27 (0.42–8.03)	0.742

*Adjusting for age, sex, educational level, cigarette smoking, alcohol consumption, physical activity, sleep quality, fasting glucose, and low- and high-density lipoprotein cholesterol

PP: Pulse pressure; SBP: systolic blood pressure; MMSE: Mini-Mental State Examination

estimated risk effect for participants with both conditions seemed to be higher than that for those with elevated PP alone (OR = 5.04 vs. 4.54), no statistically significant interaction was found (Supplementary Table S1). Similarly, PP had a better prediction performance for impaired cognitive function than SBP (area under the ROC curve: 0.714 vs. 0.662, *P* for difference = 0.009, Fig. 2).

Results of sensitivity analysis

The association between PP and cognitive function seemed stronger in females than males (Supplementary Tables S2-S3). After excluding participants with prevalent hypertension, diabetes, and CVD, respectively, our results did not change a lot (Supplementary Tables S4-S5).

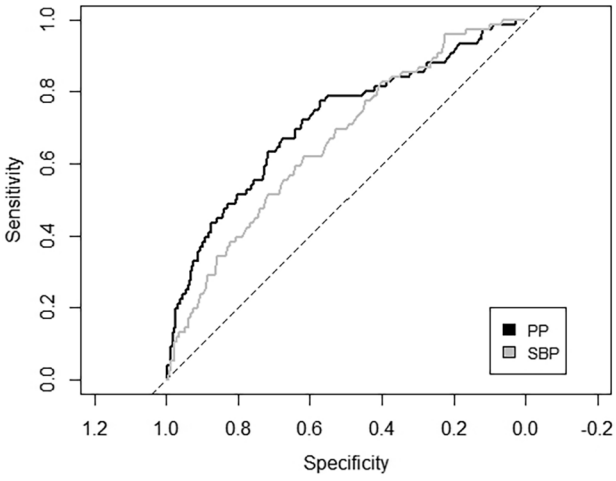

Fig. 2 ROC curves showing the prediction performance of PP and SBP for impaired cognitive function. **Legends**: The area under the ROC curve for PP was 0.714 (95%CI: 0.651–0.778), which was significantly higher than that for SBP (0.662, 95%CI: 0.603–0.722). The *P* value for the difference was 0.009. ROC: Receiver operating characteristic

Discussion

In this large population of Chinese adults participating in the Tianning Cohort, we examined the associations of vascular aging assessed by arterial stiffness and blood pressure simultaneously with cognitive function assessed by MMSE. Using an MMSE score of less than 24 points as the diagnosis of impaired cognitive function, about 1.47% of Chinese adults suffered from this disorder. PP and SBP were significantly associated with a higher risk of prevalent impaired cognitive function. These associations were independent of behavioral and metabolic factors. Our results indicated that blood pressure may deliver effects on cognitive function through mechanisms beyond behavioral and metabolic factors. Among the parameters of vascular aging PP could be a potential predictor for impaired cognitive function, as suggested by the best prediction performance.

In line with our study, the association between vascular aging and cognitive function has also been found in various studies. For example, a cross-sectional study including 698 middle-aged and elderly Spanish adults [24] found that ambulatory-measured PP was related to cognitive decline. A longitudinal study in Baltimore [19] and a 4-year community-based cohort study in China [40] revealed that PP was significantly associated with an increased risk of cognitive impairment. However, most previous studies only performed univariate analysis or adjusted sociodemographic variables such as age, gender, and education level as main covariates in relatively small populations, while ignoring behavioral factors, metabolic disease factors, etc. which may still be potential confounding factors. In our study, we conducted multivariate analysis and included all factors that may affect cognition as much as possible in a relatively large sample size, which greatly improves the validity and reliability of the results. Our study provided another initial evidence for the role of vascular aging in the process of cognitive decline. As far as we know, the possible mechanism lies in that PP is more pronounced in the brain compared to peripheral organs due to the absence of upstream vasoconstrictors that provide protection. Fortunately, microcerebral arteries can adjust to pulse pressure fluctuations through self-regulation. However, if this regulation is disrupted, it can result in microvascular damage and reduced cerebral blood flow, which may contribute to the occurrence of cerebral infarction and the development of white matter hyperintensities (WMH), ultimately leading to cognitive impairment [41, 42]. It was undeniable that few studies could specifically elucidate the mechanism, which still needs a large amount of basic experimental research to study. The association between elevated SBP and cognition disorders observed in our study was also consistently demonstrated by prior studies. Several systematic reviews and meta-analyses have shown that high SBP was associated with an increased risk of cognitive impairment and that anti-hypertensive treatment could reduce the risk of cognitive impairment [43, 44]. A prospective longitudinal study including 2777 participants found that hypertension was associated with a greater cognitive decline [45].

Unlike most of the existing research results [18, 27, 46, 47], we did not find a significant relationship between vascular aging measured by cfPWV and ABI and cognition. For example, a community-dwelling study including 388 individuals in Japan [46] and a case-control study in Shanghai, China [27] found that a low ABI may be a risk factor for cognitive impairment and a critical tool for predicting early cognitive impairment. A cross-sectional study including 4,086 participants from the Heinz Nixdorf Recall cohort study found a significant association between ABI and mild cognitive impairment [48]. Several studies like the Singapore longitudinal aging study [23] found a link between vascular health measured by cfPWV and cognitive function. Nevertheless, conflicting results were also found in some other studies. In addition to ours, the Sydney memory aging study [49] and the Rotterdam study [50] did not find a significant association between PWV and cognitive decline. One longitudinal study did not find a significant association between ABI and the risk for dementia [51]. Population characteristics and the complicated nature of the association between vascular health and cognition may cause these conflicting findings. In our study, almost all participants had good cognition with only 76 individuals suffering cognitive impairment. Such a relatively healthy population may be not powerful for the identification of the association between arterial stiffness and cognitive

function. Therefore, we additionally conducted a subgroup analysis based on age and discovered that the association between PP and cognitive function appeared to be more pronounced in participants aged over 50 compared to those under 50 (Supplementary Tables S5-S6). This age disparity suggests that the association between vascular aging and cognitive function may be underestimated in our study population. Further research is needed to thoroughly examine their association in diverse populations.

Although arterial stiffness and blood pressure increase with aging synchronously, whether the mechanisms behind these two vascular phenotypes act synergistically or in parallel is not known. Therefore, an interaction between arterial stiffness and blood pressure could be hypothesized. To our knowledge, few studies have examined the interactive effects of arterial stiffness and blood pressure on cognitive function. In our study, we failed to observe any significant multipliable or additive interactions, which indicated that the effects of arterial stiffness and blood pressure on cognition may be parallel. Among the parameters of arterial stiffness and blood pressure, PP may be a better predictor of cognitive impairment. Although a higher PP indicates a stiffer arterial, blood pressure, SBP and PP, in particular, may be more likely to affect blood flow in the brain. This may be the reason why we failed to observe the association between arterial stiffness and cognitive function. In our study, we found that the association between PP and cognitive function was more pronounced in females than males. This sex difference has also been observed in the Framingham Heart Study, where PP was found to be higher and more closely linked to arterial properties in females [52].

Some strengths deserve to be mentioned in our study. Compared with other small-sample studies, this study was one of the largest-scale cross-sectional population studies with good regional representation. Besides, in terms of methodology, we applied two regression models, Poisson and logistic, and adjusted confounding factors such as sociodemographic variables (age, gender, and educational level), lifestyle (cigarette smoking and alcohol consumption), behavioral factors (physical activity and sleep) and metabolic factors (fasting glucose and lipids). Furthermore, sensitivity analysis was performed to further ensure the robustness and reliability of the results by excluding participants with prevalent hypertension, diabetes, and CVD, respectively.

Limitations

There were also some limitations worth noting in the present study. First, due to the cross-sectional design, we could not explain a causal relationship between vascular aging or blood pressure and cognitive impairment. Second, although we used an unselected sample, the population in the Tianning Cohort was of the Han nationality and could not represent other ethnic/racial populations. Third, only the MMSE score was used to evaluate the cognitive level of individuals, the lack of specialized cognitive assessment equipment may have led to some patients with mild cognitive impairment being overlooked, which may have introduced some selection bias.

Conclusion

This large-scale cross-sectional population-based study provided initial evidence for the associations of vascular aging and hypertension with cognitive function in Chinese adults. PP may be used as a predictive indicator to recognize impaired cognition which provides an opportunity for early intervention.

Abbreviations

AI	Augmentation index
ABI	Ankle-brachial index
BMI	Body mass index
CVD	Cardiovascular disease
CAVI	Cardio-ankle vascular index
cfPWV	carotid-femoral pulse wave velocity
DBP	Diastolic blood pressure
FMD	Flow-mediated dilation
FDR	False-discovery rate
GATS	Global Adult Tobacco Survey
GPAQ	Global Physical Activity Questionnaire
HDL-C	High-density lipoprotein cholesterol
IMT	Intima-media thickness
LDL-C	Low-density lipoprotein cholesterol
MCI	Mild cognitive impairment
MMSE	Mini-Mental State Examination
MAP	Mean arterial pressure
PP	Pulse pressure
PSQI	Pittsburgh Sleep Quality Index
SBP	Systolic blood pressure

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-024-19700-6.

Supplementary Material 1

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Author contributions

SC and HZ wrote the manuscript. JZ performed data analysis. HJ, WF, XZ, YJ, and XY collected data. CM and HP designed the study and reviewed the manuscript.

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Data availability
The datasets analyzed during the current study are not publicly available because the Tianning Cohort study is still ongoing but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The protocols of the Tianning Cohort were approved by the Ethics Committee of Soochow University (approval No. ECSU-201800051). All participants signed a written informed consent.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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