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L-shaped association between ankle-brachial index and coronary heart disease in Chinese adults with hypertension

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Abstract

Background Prior research has established that an ankle-brachial index (ABI) ≤ 0.9 is positively correlated with cardiovascular events, including coronary heart disease (CHD). The present study aimed to elucidate the dose–response relationship between ABI and CHD within a hypertensive population.

Methods We conducted a cross-sectional analysis involving 10,900 hypertensive patients, with CHD as the primary outcome. A generalized additive model (GAM) and fitted smoothing curve were employed to assess linearity and delineate the dose–response association between ABI and CHD.

Results The cohort had a mean (SD) age of 68.3 (9.25) years, with 5129 (47.06%) being male. CHD was present in 552 (5.06%) participants. The fully adjusted odds ratio (OR) for CHD associated with ABI levels was 0.75 (95%CI 0.33–1.71). An L-shaped relationship between ABI and CHD was identified, with an inflection point at 1.07. Below this threshold, ABI showed a negative correlation with CHD (OR: 0.27; 95%CI 0.08–0.84), whereas above it, the association was not significant (OR: 3.08; 95%CI 0.60–15.80).

Conclusions In Chinese adults with hypertension, ABI exhibits a nonlinear, L-shaped association with CHD, with the inflection point at 1.07.

Keywords Coronary heart disease, Ankle-brachial index, Hypertension, L-shaped curve, Stratified analysis

Introduction

Hypertension is a major global public health challenge, which increases the risk of cardiovascular disease and premature death [1]. With the rapid urbanization and population aging in China, the prevalence of hypertension has increased significantly in recent years, reaching 44.7% among Chinese adults aged 35–75 in 2017 [2]. Based on available researches, there is a close and frequent connection between hypertension and coronary heart disease (CHD) [3–5]. When the systolic blood pressure is between 120 and 129 mmHg, the risk ratio of myocardial infarction is 1, and when the systolic blood pressure is more significant than 140 mmHg, the risk ratio is 2 [6]. A study by Stamler J et al. showed that the risk of death from CHD in hypertensive patients is 2.3 time [7]. Therefore, it is necessary to find a detection

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method to find early asymptomatic CHD in high-risk groups such as hypertension and formulate relevant treatment strategies to reduce the occurrence and development of CHD.

In 2017, European guidelines for diagnosis and treatment of peripheral vascular diseases pointed out that peripheral arterial diseases (PAD) could be indicated by ankle-brachial index (ABI) [8]. PAD is an atherosclerotic disease characterized by occlusion (blockage) or narrowing (narrowing) of the lumen of the peripheral artery [9]. As we all know, atherosclerosis is the main pathogenic factor of CHD. There are also studies showing that ABI is a simple and noninvasive method to measure subclinical atherosclerosis [10]. Therefore, we speculate that ABI can also be used as a non-invasive clinical detection method to detect CHD. However, most studies merely evaluated the relationship between PAD defined by ABI and CHD, these studies all presented that a low ABI level (< 0.9) was related to the increased risk of CHD [11–15]. However, using ABI < 0.9 to predict CHD has some limitations. Some typical examples were as follows: Weatherley et al. [16] conducted a study on African Americans to study the relationship between the level of the ABI and the risk of CHD, which results show that the risk of CHD not only increases exponentially when ABI < 1.0 but also decreases continuously when ABI > 1.0. The Honolulu Heart Project results [17] shew that a low ABI (< 0.8) increases the risk of CHD in older adults. The above studies showed the level of ABI for predicting the risk of CHD is different. So far, the true dose–effect relationship between ABI and CHD has not been determined.

Therefore, using the data from the Chinese H-type hypertension registration study (CHHR), this study explores the association of the optimal range of ABI with the risk of CHD in a large cross-sectional analysis of Chinese adults with hypertensive.

Methods

Study design and participants

Data analyzed in this study was the baseline of the ongoing China H-type Hypertension Registry Study (registration number: ChiCTR1800017274). The method of data collection and the exclusion criteria have been described previously [18]. This study aimed to establish a population-based H-type hypertension registry cohort and explore the prevalence, treatment status, and related factors that influence the prognosis of H-type hypertension. In summary, this study is a real-world observational study with baseline data collected from March to August 2018 in Wuyuan County, Jiangxi Province, China. The variables collected encompassed socio-demographic characteristics, physical examination findings, blood biochemical indices, and histories of disease and medication use. Eligible participants were adults aged 18 years and older with hypertension, defined as a seated, resting systolic blood pressure (SBP) ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg at screening, or those on antihypertensive medications. Exclusion criteria included neurological abnormalities, inability to follow the study protocol for follow-up, plans to relocate shortly, and patients deemed unsuitable for inclusion or long-term follow-up by study physicians. Study was approved by the Ethics Committees of the Biomedical Institute of Anhui Medical University. All participants provided written informed consent.

A total of 14,234 hypertensive patients were included in the baseline of this study. Patients were excluded if they had missing ABI data ($n = 3328$). Finally, 10,906 subjects were analyzed (Figure S1).

Clinical characteristics and laboratory measurements

Baseline information on demographic sociological variables, alcohol habits, smoking habits, medication use, and past medical history was collected by trained medical staff during a standardized questionnaire. Anthropometric parameters indicators such as hip-circumference (HC), waist circumference (WC), height, and weight were also collected by trained medical staff at baseline. With participants standing, waist circumference (WC) was measured midway between the lower edge of the costal arch and the iliac crest's upper edge to the nearest 0.1 cm. Seated systolic/diastolic blood pressure (SBP/DBP) was measured by trained technicians in triplicate after a 10 min rest, using an automatic sphygmomanometer (Omron; Dalian, China). The three readings were averaged. BMI was calculated by weight/height^2 (kg/m^2).

Participants fasted for at least 8 h before blood collection. Samples of blood were drawn from the antecubital vein by a trained phlebotomist. Total triglycerides, total- and high-density lipoprotein (HDL)-cholesterols, creatinine, plasma homocysteine, and fasting plasma glucose (FPG) was measured using automatic clinical analyzers (Beckman Coulter). The renal function was calculated as the chronic kidney disease (CKD) epidemiology collaboration of estimated glomerular filtration rate (eGFR ml/min /1.73 m^2) [19].

Ankle-brachial index measurement

Ankle-brachial index (ABI) is the ratio of systolic blood pressure (SBP) measured at the ankle artery and brachial artery [8]. In this study, we used the Omron Colin BP-203RPE III device (Omron Health Care) to measure the ABI of all subjects and measured it in a supine position after 10 min of rest [20]. At the same time, we calculate the ABI value of each leg and then take the lowest ABI value as the exposure variable of the final analysis [21].

Definition of coronary heart disease

The diagnoses of CHD were obtained from the questionnaire. The questionnaire on stroke includes the following questions "Have you ever had a CHD," if so, when the CHD occurred, the symptoms at that time, what treatment methods, and whether there are medical records, including discharge summaries and imaging pictures [22].

Statistical analysis

Baseline characteristics are presented as the mean (standard deviation, SD) or number (percentage), as appropriate. To study the linear trends of baseline characteristics of ABI index quartile, chi-square test of linear trend was used for categorical variables, and one-way analysis of variance of linear trend was used for continuous variables.

To investigate the association between ABI and CHD, we employed multivariate logistic regression to estimate odds ratios and confidence intervals. We constructed four models: Model 1 was unadjusted; Model 2 was adjusted for age and sex; Model 3 was further adjusted for serum homocysteine, fasting plasma glucose, total cholesterol, triglyceride, LDL-C, eGFR; and Model 4 was additionally adjusted for diabetes mellitus, antihypertensive drugs, glucose-lowering drugs, lipid-lowering drugs, antiplatelet drugs. The adjusted covariates are clinical risk factors related to CHD and potential confounding factors, and the potential confounding factors are that the effect estimates value changed by more than 10% [23]. In addition, a fitting generalized additive model (GAMs) and a fitted smoothing curve was performed to test for linearity and explore the shape of the dose–response relation of ABI with CHD. To evaluate potential effect modification, we performed stratified analyses. We assessed whether the relationship between ABI and CHD was stable across subgroups defined by sex, BMI, age, smoking, drinking, use of antihypertensive drugs, and diabetes mellitus in this study.

Data was analyzed using the Empower (R;www.empowerstats.com; X&Y Solutions, Inc, Boston, MA, USA) and the statistical package (R) (http://www.R-project.org, The R Foundation). We employed the 'mgcv' package for fitting generalized additive models (GAMs). Most importantly, statistical significance was regarded as a two-sided p value < 0.05.

Results

Study populations

The mean age was 63.86 ± 9.25 years among the 10,906 enrolled individuals with hypertensive; 5134 (47.08%) were men. The mean ABI was 1.09 ± 0.10. There were 553 (5.07%) participants had CHD in the current study. Table 1 presents the baseline demographic characteristics and biochemical data of the participants based on quartiles of ABI levels. Concerning the BMI, fasting plasma glucose, AST, ALT, diabetes mellitus, glucose-lowering drugs, antiplatelet drugs, lipid-lowering drugs, and antihypertensive drugs, there were no significant differences among the above three groups. Participants with the highest ABI in Q4 (ABI ≥ 1.15) were more likely than the other three counterparts to be older and males. SBP, heart rate, serum homocysteine, total cholesterol, triglyceride, HDL-C, LDL-C, eGFR, and the prevalence of stroke and CHD were significantly higher, whereas DBP, current smokers, current drinkers, serum uric acid and eGFR were lower in participants with the lowest ABI (ABI < 1.05) than the rest counterparts.

Association of ABI with CHD

Overall, there was an L-shaped association between ABI and CHD (Fig. 1). ORs and 95% CIs of CHD according to tertiles of ABI levels are summarized in Table 2. Per 1 unit increment in ABI, the odds ratios (OR) of the prevalence of CHD was 0.75 (95%CI 0.33–1.71) after adjusting for age, sex, BMI, SBP, DBP, pulse, current smoking, current drinking, serum homocysteine, fasting plasma glucose, total cholesterol, triglyceride, LDL-C, eGFR, diabetes mellitus, antihypertensive drugs, glucose-lowering drugs, lipid-lowering drugs, antiplatelet drugs. The decrease of CHD prevalence rate is not significant with the increase of ABI. When ABI is used as the quartile of classification variable, full adjusted OR of CHD for participants in quartile 1, quartiles 3 and quartile 4 of ABI level, respectively, were 1.35 (95%CI 1.03–1.76), 1.19 (95%CI 0.91–1.56) and 1.22(95%CI 0.94–1.60) compared with those in the quartile 2 (P for trend = 0.343). The result of multiple logistic regression analysis is consistent with the fitting curve, which indicates that ABI is nonlinear related to CHD. Association of ABI with CHD was evaluated binary logistic regression model, which figured out the turning point was 1.07. On the left of the turning point, ABI is negatively associated with CHD (OR: 0.27; 95%CI 0.08–0.84), while the prevalence of CHD does not decrease with the increase of ABI level on the right of turning point (OR: 3.08; 95%CI 0.60–15.80) (Table 3).

Stratified analyses by important covariables

According to the inflection point of ABI, we divided the participants into two groups. Additional exploratory analyses were performed. In the stratified analyses, the ABI level associations with CHD in the different subgroups were consistently the same (Fig. 2). These subgroups included sex (males vs. females), BMI (< 25 vs. ≥ 25 kg/m^2), age (< 65 vs. ≥ 65 years), current smoking

Table 1 Clinical characteristics of the study population according to ABI

Characteristics	Total	ABI				*p* value
		Q1 (< 1.05)	Q2 (1.05–1.09)	Q3 (1.10–1.14)	Q4 (≥ 1.15)	
N	10,906	2689	2484	2823	2910	
Males, *N* (%)	5134 (47.08)	1092 (40.61)	1039 (41.83)	1367 (48.42)	1636 (56.22)	< 0.001
Age, year	63.86 ± 9.25	64.58 ± 10.33	63.09 ± 9.25	63.15 ± 8.82	64.55 ± 8.46	< 0.001
BMI, kg/m^2	23.59 ± 3.81	23.54 ± 3.89	23.62 ± 3.46	23.71 ± 4.32	23.48 ± 3.47	0.124
Current smoking, *N* (%)	2871 (26.33)	699 (26.00)	589 (23.71)	763 (27.03)	820 (28.18)	0.002
Current drinking, *N* (%)	2472 (22.67)	498 (18.53)	516 (20.78)	687 (24.34)	771 (26.49)	< 0.001
SBP, mmHg	148.48 ± 17.78	149.79 ± 19.36	148.45 ± 17.61	147.84 ± 17.06	147.92 ± 17.03	< 0.001
DBP, mmHg	89.02 ± 10.74	88.57 ± 11.10	89.51 ± 10.42	89.50 ± 10.42	88.54 ± 10.95	< 0.001
Heart rate, bpm	76.32 ± 14.18	77.92 ± 15.66	76.47 ± 13.30	75.80 ± 13.46	75.23 ± 14.01	< 0.001
Laboratory data						
Serum homocysteine, μmol/L	18.00 ± 11.04	18.50 ± 11.92	17.78 ± 11.16	17.73 ± 10.87	17.97 ± 10.19	0.042
Fasting plasma glucose, mmol/L	6.17 ± 1.60	6.19 ± 1.65	6.14 ± 1.54	6.15 ± 1.61	6.17 ± 1.60	0.649
Total cholesterol, mmol/L	5.14 ± 1.11	5.23 ± 1.14	5.15 ± 1.10	5.15 ± 1.09	5.05 ± 1.11	< 0.001
Triglyceride, mmol/L	1.78 ± 1.25	1.81 ± 1.18	1.84 ± 1.37	1.78 ± 1.26	1.70 ± 1.18	< 0.001
HDL-C, mmol/L	1.59 ± 0.44	1.61 ± 0.44	1.61 ± 0.43	1.60 ± 0.45	1.55 ± 0.42	< 0.001
LDL-C, mmol/L	2.99 ± 0.81	3.09 ± 0.84	3.01 ± 0.79	2.98 ± 0.79	2.89 ± 0.80	< 0.001
AST, U/L	26.78 ± 16.70	26.81 ± 24.19	26.58 ± 14.45	26.87 ± 14.05	26.82 ± 11.60	0.924
ALT, U/L	20.44 ± 16.95	20.34 ± 23.18	20.32 ± 14.41	20.71 ± 15.68	20.38 ± 12.84	0.807
Serum uric acid,, mmol/L	415.20 ± 120.84	412.12 ± 121.03	405.46 ± 120.49	413.87 ± 120.87	427.64 ± 119.97	< 0.001
eGFR, mL/min/1.73 m^2	88.67 ± 20.38	87.41 ± 22.75	90.51 ± 19.47	89.93 ± 18.90	87.03 ± 19.98	< 0.001
Comorbidities, *N* (%)						
Stroke	708 (6.49)	203 (7.55)	133 (5.35)	186 (6.59)	186 (6.39)	0.016
CHD	553 (5.07)	158 (5.88)	100 (4.03)	136 (4.82)	159 (5.46)	0.014
Diabetes mellitus[a]	1238 (11.35)	319 (11.86)	278 (11.19)	327 (11.58)	314 (10.79)	0.610
Medication use, *N* (%)						
Antihypertensive drugs	7159 (65.65)	1790 (66.59)	1643 (66.14)	1844 (65.32)	1882 (64.67)	0.441
Glucose-lowering drugs	573 (5.25)	138 (5.13)	124 (4.99)	156 (5.53)	155 (5.33)	0.832
Lipid-lowering drugs	381 (3.49)	103 (3.83)	82 (3.30)	103 (3.65)	93 (3.20)	0.543
Antiplatelet drugs	429 (3.93)	128 (4.76)	93 (3.74)	106 (3.75)	102 (3.51)	0.080

Data are the mean ± SD, or number (percentage)

BMI body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *CHD* coronary heart disease, *eGFR* estimated glomerular filtration rate, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *ALT* alanine aminotransferase, *AST* aspartate aminotransferase, *eGFR* estimated glomerular filtration rate

[a] Diabetes mellitus was defined as self-reported physician diagnosis of diabetes or fasting plasma glucose concentration ≥ 7.0 mmol/L or use of glucose-lowering drugs. Table 1 Clinical characteristics of the study population according to ABI

(no vs. yes), current drinking (no vs. yes), and diabetes mellitus (no vs. yes).

Discussion

In this study of Chinese adult hypertensive patients with an average age of 63.86 years, we observed a nonlinear relationship between the level of ABI and the prevalence of CHD. Specifically, an independent negative correlation between ABI level and the prevalence of CHD was identified only in the subgroup with ABI ≤ 1.07, after adjusting for risk factors associated with cardiovascular disease. Furthermore, this L-shaped association between ABI and CHD was consistent across various subgroups.

Although numerous studies have investigated the correlation between ABI-defined PAD and the incidence of CHD, the findings have been inconsistent. Sarangi et al. [11] selected 182 inpatients over 45 years of age with one or more traditional risk factors for PAD (ABI < 0.9) to explore the relationship between CHD and PAD, revealing a positive correlation. Amer et al.[12] conducted a case–control study on the severity of ABI and CHD among elderly Egyptian participants, indicating a positive correlation with CHD severity for ABI < 0.9.

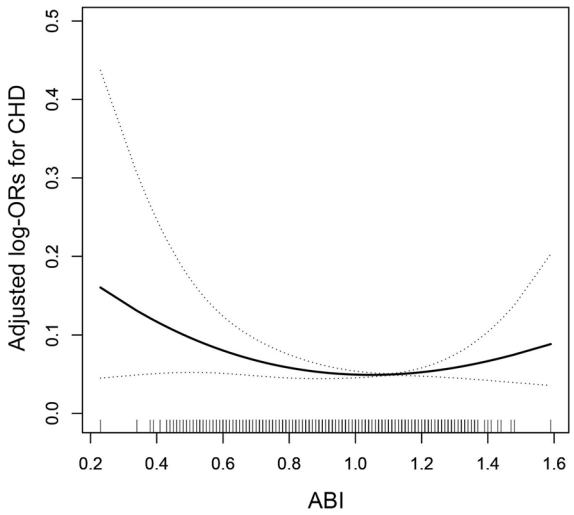

Fig. 1 Association between ABI and the prevalence of CHD. A nonlinear association between ABI and the prevalence of CHD was found ($p < 0.05$). The solid line and dashed line represent the estimated values and their corresponding 95% confidence interval. Adjustment factors included age, sex, BMI, SBP, DBP, heart rate, current smoking, current drinking, serum homocysteine, fasting plasma glucose, total cholesterol, triglyceride, LDL-C, eGFR, diabetes mellitus, antihypertensive drugs, glucose-lowering drugs, lipid-lowering drugs, antiplatelet drugs

Filippella et al. [13] evaluated the relationship between ABI and CHD in a cohort of 969 patients with type 2 diabetes, concluding that patients with ABI < 0.9 were at a higher risk of CHD. However, Weatherley et al. [16] included 12,186 white persons and African–Americans with the data of the Atherosclerosis Risk In Communities (ARIC) study to explore the relationship between different ABI values and the risk of CHD, the median follow-up was 13.1 years. In Caucasians, after adjusting for age and occupational center, the hazard ratio (HR, 95% CI) for CHD risk in patients with PAD (ABI < 0.90) was 2.81 (1.77–4.45) for males and 2.05 (1.20–3.53) for females. In African Americans, the HR for males was 4.86 (2.76–8.47), and for females, it was 2.34 (1.26–4.35). The risk of CHD increased exponentially with decreasing ABI as a continuous variable in all race-gender subgroups. The association between ABI and relative risk of CHD was similar in males and females across both racial groups. In Caucasian males, for every 0.10 decrease in ABI, the risk of CHD increased by 25% (95% CI 17–34%); in Caucasian females, for every 0.10 decrease in ABI, the risk of CHD increased by 20% (8–33%); in African American males, for every 0.10 decrease in ABI, the risk of CHD increased by 34% (19–50%); and in African American females, for every 0.10 decrease in ABI, the risk of CHD increased by 32% (17–50%). These significant findings indicate that the risk of CHD is closely associated with ABI levels, with a more pronounced effect observed in certain demographic groups. Abbott et al. [17] using data from the Honolulu Heart Program, included 2863 Japanese American men over 70 years old and found that an ABI value of less than 0.8 was nearly related to an increased risk of CHD after a follow-up of 3–6 years.

The aforementioned studies highlight that while PAD, as defined by a low ABI, is associated with an increased risk of CHD events, the intrinsic value of ABI itself is often underappreciated. Given that atherosclerosis is a systemic condition, the presence of lesions in one vascular bed frequently suggests that other arterial territories have undergone analogous pathological changes [24]. Consequently, the ABI can be employed as an index to gauge the extent of arteriosclerosis within the coronary vasculature. However, due to the variability in arterial position and length, the ABI value that defines arterial stiffness exhibits heterogeneity. Furthermore, the ABI values predictive of coronary heart disease risk differ

Table 2 Association between ABI and the prevalence of CHD

ABI	*N*	Events (%)	CHD *OR* (95%CI)			
			Model 1	Model 2	Model 3	Model 4
Continuous	10,906	553 (5.07)	0.79 (0.36, 1.74)	0.63 (0.28, 1.41)	0.79 (0.36,1.74)	0.75 (0.33, 1.71)
Quartiles						
Q1 (< 1.05)	2689	158 (5.88)	1.49 (1.15, 1.92)	1.36 (1.05, 1.76)	1.34 (1.03,1.74)	1.35 (1.03, 1.76)
Q2 (1.05–1.10)	2484	100 (4.03)	1	1	1	1
Q3 (1.10–1.15)	2823	136 (4.82)	1.21 (0.93, 1.57)	1.21 (0.93, 1.58)	1.21 (0.93, 1.58)	1.19 (0.91, 1.56)
Q4 (≥ 1.15)	2910	159 (5.46)	1.38 (1.07, 1.78)	1.29 (1.00, 1.67)	1.26 (0.97, 1.64)	1.22 (0.94, 1.60)
P for trend			0.102	0.163	0.205	0.343

Model 1: Unadjusted

Model 2: Adjusted for age, sex

Model 3: Further adjusted for serum homocysteine, fasting plasma glucose, total cholesterol, triglyceride, LDL-C, eGFR

Model 4 Additionally adjusted for diabetes mellitus, antihypertensive drugs, glucose-lowering drugs, lipid-lowering drugs, antiplatelet drugs

Table 3 Threshold effect of ABI on CHD

ABI	*N*	Events (%)	CHD *OR* (95%CI)			
			Model 1	Model 2	Model 3	Model 4
Continuous	10,906	553 (5.07)	0.79 (0.36, 1.74)	0.63 (0.28, 1.41)	0.79 (0.36,1.74)	0.75 (0.33, 1.71)
Inflection point						
≤ 1.07	6806	215 (5.24)	0.30 (0.08, 0.83)	0.29 (0.10, 0.86)	0.33 (0.11,0.99)	0.27 (0.08, 0.84)
> 1.07	4100	338 (4.97)	4.54 (0.97, 21.32)	4.15 (0.86, 19.95)	3.37(0.69, 16.37)	3.08 (0.60, 15.80)
P for log likelihood ratio test			0.014	0.021	0.044	0.024

Model 1: Unadjusted

Model 2: Adjusted for age, sex

Model 3: Further adjusted for serum homocysteine, fasting plasma glucose, total cholesterol, triglyceride, LDL-C, eGFR

Model 4 Additionally adjusted for diabetes mellitus, antihypertensive drugs, glucose-lowering drugs, lipid-lowering drugs, antiplatelet drugs

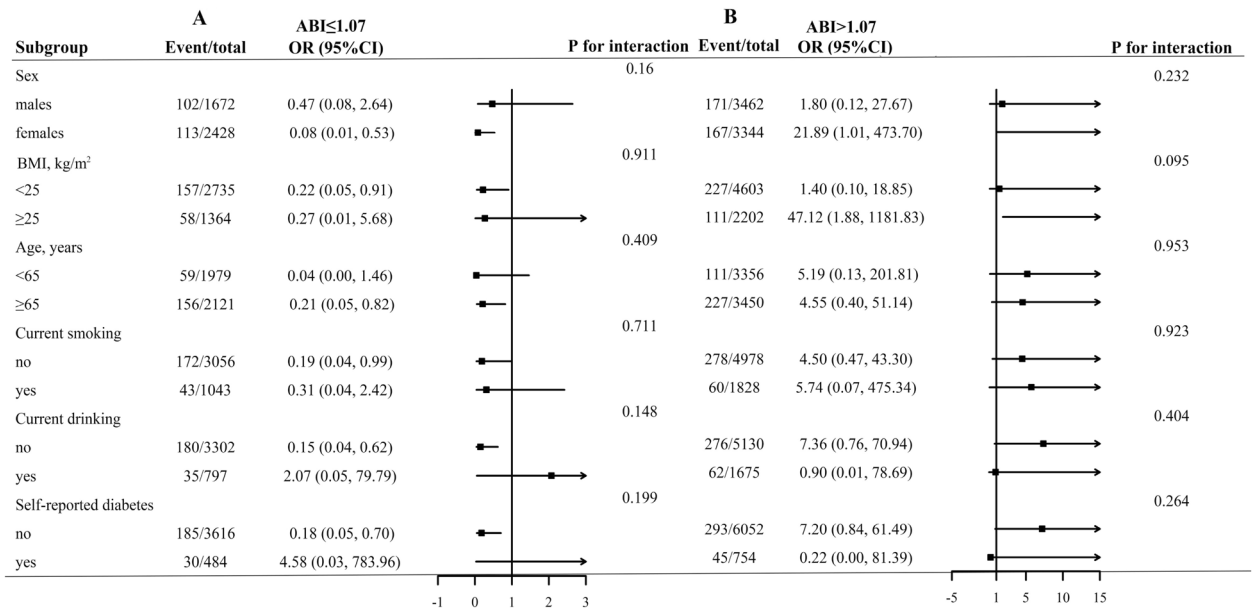

Fig. 2 Stratified analysis for the CHD and ABI in various subgroups divided by 1.07. (**A** ABI ≤ 1.07 μmol/lL, **B** ABI > 1.07 μmol/lL). *Each subgroup analysis adjusted for age, sex, BMI, SBP, DBP, heart rate, current smoking, current drinking, serum homocysteine, fasting plasma glucose, total cholesterol, triglyceride, LDL-C, eGFR, diabetes mellitus, antihypertensive drugs, glucose-lowering drugs, lipid-lowering drugs, antiplatelet drugs except for the stratifying variable

among study populations with varying disease states. This study aims to highlight this critical aspect and contributes to the management of atherosclerotic risk in hypertensive patients by recognizing the nonlinear relationship between ABI levels and CHD prevalence, particularly in the subgroup with ABI ≤ 1.07 after adjusting for cardiovascular risk factors.

We can get some new viewpoints from the results of this study: 1. the pathological mechanism of PAD and CHD is closely related to atherosclerosis, and these two diseases are just different clinical manifestations of atherosclerosis [25]. Then ABI can be used as a detection method to predict the risk of CHD. Because of the heterogeneity of diseases, the critical value of ABI may be different. 2. The prevalence of PAD in general population is low [26], and the prevalence of PAD in the hypertensive population in this study is only 3.22%, so it is not accurate to use ABI as a classification variable, which will make us unable to find the risk of CHD in high-risk groups such as hypertension in time. 3. The results of this study show that ABI is negatively correlated with CHD in patients with ABI ≤ 1.07, while ABI is not related to CHD in patients with ABI > 1.07, which is inconsistent with the results of Weatherley et al. Their research shows

that ABI is negatively correlated with CHD. The reason for the inconsistent ABI inflection point may be related to research design, research population, and race.

Limitations

The current study has several limitations. First, the cross-sectional nature of this study does not allow us to evaluate causal associations between ABI value and CHD in Chinese hypertensive patients. Second, we only measured the ABI value once at baseline, and multiple measurements may be more accurate. Third, we conducted in Chinese population, and it remains to be discussed whether the results can be extended to other populations.

Future directions

Although our cross-sectional study provides valuable insights, it is not robust enough to alter clinical practice guidelines. The evidence from this study suggests that future large-scale randomized controlled trials are necessary to confirm our findings.

Clinical practical significance

The results do underscore the importance of closely monitoring ABI values in hypertensive patients, which may contribute to more nuanced risk stratification and management strategies. This study's findings indicate that ABI values below 1.07 are associated with a higher prevalence of CHD, particularly in the context of cardiovascular risk factors. Therefore, these findings should prompt clinicians to consider ABI as a potential marker for coronary artery disease in their assessment of hypertensive patients. The implications for future research are clear: there is a need for further investigation into the ABI's role in cardiovascular risk assessment, especially in different demographic and disease state populations, to better inform clinical decision-making.

Conclusions

In this cross-sectional study, we identified a significant inverse correlation between ABI levels and CHD prevalence specifically within the subgroup with ABI $\leq$ 1.07, highlighting the potential clinical relevance of ABI threshold in CHD risk assessment.

Abbreviations

ABI	Ankle-brachial index
BMI	Body mass index
CHD	Coronary heart disease
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
Hcy	Homocysteine
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
eGFR	Estimated glomerular filtration rate
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
SD	Standard deviation
CI	Confidence interval
OR	Odds ratio
ANOVA	One-way analysis of variance
GAM	Generalized additive model

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s40001-025-02342-8.

Supplementary Material 1
Supplementary Material 2
Supplementary Material 3
Supplementary Material 4

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Author contributions

Xiaoshu Cheng and Huihui Bao devised the study concept and designed the study. Wei Zhou, Yumeng Shi, Chao Yu, Tao Wang, and Lingjuan Zhu conducted the data collection and analysis and drafted the manuscript. Wei Zhou, Chao Yu, Huihui Bao and Xiaoshu Cheng critically reviewed and revised the manuscript for important intellectual content. All authors reviewed the manuscript.

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Availability of data and materials

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Written informed consent was obtained from the participants.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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