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A cross-sectional study of the ambulatory central artery stiffness index in patients with hypertension

Xuezhai Zeng, MD[a], Na Jia, MD[a], Deping Liu, MD[a,*], Lijuan Wang, MD[b], Zhujin Xu, MD[c], Yan Zhang, MD[d], Hua Wang, MD[a], Ruisheng Zhang, MD[a], Jihong Zhou[a], Chunyu Pan[a], Yu Gan, MB[a], Weiwei Qiao[a]

Abstract

The present study aimed to investigate the characteristics of the ambulatory central artery stiffness index (AcASI) and its related factors. The association between AcASI and the left ventricular mass index (LVMI), and other factors related to atherosclerosis were explored.

Patients with primary hypertension were enrolled into this study. Ambulatory central artery blood pressure (CABP) and ambulatory brachial artery blood pressure (BABP) were assessed using a Mobil-O-Graph NG hemomanometer, whereas AcASI and the ambulatory arterial stiffness index (AASI) were determined. LVMI was assessed by echocardiography.

A total of 136 patients with primary hypertension were enrolled from May 2011 to January 2013 in Beijing Hospital. AcASI was significantly associated with AASI ($r = 0.879$, $P < .001$). AcASI was significantly lower than AASI (0.422 ± 0.302 vs 0.482 ± 0.270; $P < .001$). AcASI increased with age, ambulatory brachial mean blood pressure (MBP), and fasting glucose. AcASI was significantly associated with office pulse pressure (PP), ambulatory brachial PP, ambulatory central PP, and pulse wave velocity (PWV). AcASI, but not AASI, was significantly associated with LVMI. Receiver operator characteristic analysis indicated that AcASI and AASI could may be a predictor of left ventricular hypertrophy (LVH). Multiple regression analysis indicated that AcASI, chronic kidney disease, and hypertension course were associated with LVMI, but AASI was not.

AcASI, which is obtained from ambulatory CABP monitoring, could be a new marker for the evaluation of atherosclerosis. AcASI may be stronger associated with LVH than AASI.

Abbreviations: AASI = ambulatory arterial stiffness, AcASI = ambulatory central artery stiffness, BABP = brachial artery blood pressure, CABP = central artery blood pressure, DBP = diastolic blood pressure, IMT = intima-media thickness, LVH = left ventricular hypertrophy, LVMI = left ventricular mass index, MBP = mean blood pressure, PP = pulse pressure, PWV = pulse wave velocity, SBP = systolic blood pressure.

Keywords: ambulatory arterial stiffness index, ambulatory blood pressure, central arterial blood pressure, hypertension, left ventricular mass index

1. Introduction

Atherosclerosis is a complication of hypertension, and also a risk factor for the development of cardiovascular disease (CVD).[1] Early studies have indicated that structural or functional changes of the artery wall occur before marked stenosis or occlusion arises. Early functional screening for large arteries is important in patients with hypertensive to prevent the formation of arterial lesions. Pulse wave velocity (PWV) is a sensitive marker for the evaluation of arterial stiffness in early stage CVD and is also a predictive factor for left ventricular hypertrophy (LVH). The 2007 ESH/ESC guidelines for the management of hypertension proposed PWV as the gold-standard method for the evaluation of arterial stiffness.[2] However, this analysis requires specialized equipment, which limit its use in clinical and research. Therefore, a simpler method for the evaluation of atherosclerosis in patients is needed.

Changes in systolic blood pressure (SBP) and diastolic blood pressure (DBP) may reflect arterial stiffness to some extent. Ambulatory arterial stiffness index (AASI) is a parameter detected from ambulatory blood pressure monitoring for the evaluation of arterial stiffness. AASI may reflect general arterial elasticity, and the relationship between SBP and DBP in various physiological conditions.[3] Recent studies showed that AASI is associated with hypertension-related organ damage, such as intima-media thickness (IMT), microalbuminuria, and LVH,[4–7] and is valuable for the prediction of CVD-linked events.[8–12]

Peripheral blood pressure (PBP) differs from CABP; PBP does not indicate the changes in CABP.[13] Some studies have suggested that the association between target organ damage and ambulatory CABP is stronger than that with ambulatory BABP.[14,15]

LVH is a notable type of hypertensive-related target organ damage and is also an independent risk factor for chronic heart

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[a] Department of Cardiology, [b] Department of Nutrition, [c] Department of Radiology, [d] Special Care Ward, Beijing Hospital, National Center of Gerontology, Beijing, China.

[*] Correspondence: Deping Liu, Department of Cardiology, Beijing Hospital, National Center of Gerontology, No. 1 Dahua Road, Dongdan, Dongcheng District, Beijing, China (e-mail: lliudeping@263.net).

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failure, coronary artery disease (CAD), and cardiac arrest.[16] LVMI is the main indicator of LVH. No studies have been carried out to investigate the association between LVH and ambulatory central artery stiffness index (AcASI). In the present study, the features of AcASI in patients with hypertension, the factors influencing AcASI, and the association with other atherosclerotic factors will be explored. The association between AcASI, AASI, and LVH will be also analyzed.

2. Materials and methods

2.1. Study design and population

This cross-sectional study was conducted in a retrospective manner. From May 2011 to January 2013, the participants that were diagnosed with primary hypertension in Beijing Hospital were enrolled. The inclusion criteria of patients included[1]: aged >18 years old[2]; patients who had CABP and BABP were measured by a Mobil-O-Graph NG hemomanometer[3]; and LVMI was assessed by echocardiography. Patients with secondary hypertension, cardiomyopathy, pericardial disease, valvular heart disease, congenital heart disease, atrial fibrillation, infectious diseases, or malignant tumors were excluded. The Ethics Committee of Beijing Hospital approved this study.

Hypertension was defined as untreated average SBP ≥140 mm Hg or DBP ≥90 mm Hg under untreated conditions from 3 measurements conducted on ≥2 separate days. Those with SBP/DBP<140/90 mm Hg were still considered to have hypertension if prior diagnosis of hypertension and was documented and were currently under antihypertensive treatment. For ambulatory blood pressure monitoring (ABPM), the thresholds for hypertension were determined as a 24-hour average blood pressure (BP) ≥130/80 mm Hg, daytime average BP ≥135/85 mm Hg, or nighttime average BP ≥120/70 mm Hg.[17]

2.2. General clinical data collection

General clinical data comprised the medical history, physical examination findings, and laboratory data of patients. Medical history data included a history of hypertension, diabetes, cardiovascular disease and renal disease, a history of smoking and medication, and family history. Sex, age, height, and weight were also collected.

2.3. ABPM

ABPM was performed using a noninvasive, validated device (Mobil-O-Graph PWA, Model S/N: CP0178, IEM, Stolberg, Germany). Analysis was carried out using the oscillometric method (ARCSolver algorithm) with an upper-arm blood pressure cuff.[18] The standard cuff, inflated to just above the diastolic pressure, is used to record the brachial artery waveforms. Then, a generalized transfer function is applied to the averaged waveform to generate a corresponding aortic waveform, which is scaled to the recorded brachial diastolic and mean pressures.[19] The monitored parameters for each time point were SBP, DBP, and pulse rate. During the 24-hour period, the device was set to record readings of each parameter every 30 minutes from 06:00 to 21:59 and every 60 minutes from 22:00 to 05:59; ≥80% rate of successful readings was considered to be valid. The central arterial augmentation index (AIx), central PP, and PWV were also to be recorded.

2.4. AASI and AcASI

AASI is a novel index of vascular stiffness by computing the slope of diastolic on systolic pressure (β) from 24-hour ambulatory recordings.[3,20] AASI and AcASI were calculated from a simple linear regression analyses. For AASI, 24-hour SBP was set as the independent variable and DBP was set as the dependent variable. AASI was defined as 1−β. For AcASI, 24-hour central SBP was set as the independent variable and 24-hour central DBP was set as the dependent variable. AcASI was defined as 1−β.

2.5. Assessment of LVM and LVMI

Echocardiography for the assessment of LVM and LVMI was conducted following the recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging[21] by an experienced cardiologist. The cardiologist was blinded to the group designations and BP data of each patient. The GE Vivid E9 Ultrasound System (GE Healthcare, Boston, MA) was used for two-dimensional, M-mode, and color Doppler echocardiography. Parasternal long-axis cross-sectional images were collected to measure the left atrial diameter (LAD), interventricular septal thickness (IVST), posterior wall thickness (PWT), and the left ventricular end-diastole diameter (LVEDD). Left ventricular ejection fraction (LVEF) was estimated using the M-mode curve at the level of chordae tendineae. Body surface area (BSA) was calculated using the formula[22]: BSA (m^2) = 0.0057 × height (cm) + 0.0121 × weight (kg) +0.0882 (male), BSA (m^2) = 0.0073 × height (cm) + 0.0127 × weight (kg) − 0.2106 (female). LVM and LVMI were calculated based on the modified Devereux formula,[23] where LVM (g) = 0.8 × {1.04[(IVST + PWT + LVIDd)^3 − LVIDd^3]} + 0.6, and LVMI (g/m^2) = LVM/BSA. LVH was defined as LVHI ≥115 g/m^2 for men and LVHI ≥95 g/m^2 for women.[24]

2.6. Statistical methods

Statistical analysis was performed using SPSS 16.0 software (SPSS Inc., Chicago, IL) and MedCalc v.15.0 software (MedCalc. Software bvba, Ostend, Belgium). Quantitative data were presented as the mean ±SD ($\bar{x} \pm s$). A *t* test was used to compare means. Count data were compared via a Chi-square test. Pearson's coefficient was used for correlation analysis. A *Z* test (Delong method) was chosen for the comparison of the correlation coefficient by using MedCalc software. Receiver operator characteristics (ROC) analysis was used for the evaluation of LVH using different markers of atherosclerosis. Logistic regression analysis was used to test independent factors of AcASI and LVMI. $P < .05$ was considered to indicate significant differences.

3. Results

After evaluating the data efficacy, 136 patients with primary hypertension were finally enrolled. The mean patient age was 55.4 ± 14.1 years with a range of 18 to 91 years; 98 participants were male (72.1%). The mean BMI was 26.2 ± 3.5 kg/m^2. Among these participants, 89 of them (65.4%) were taking antihypertensive drugs. The number of patients and type of drugs was shown as follows: ACE (n = 16), ARB (n = 39), CCB (n = 48), β-receptor blocker (n = 24), and diuretics (n = 12). Forty-one participants were prescribed one drug, whereas 48 participants received 2 or more drugs. Females were older with a longer course of hypertension than males in the present study. The incidence of

Table 1

General characteristics of participants.

	All participants (n = 136)	Men (n = 98)	Women (n = 38)
Age, y	55.8 ± 14.2	52.7 ± 13.6**	63.7 ± 12.8
Body height, cm	169.5 ± 8.4	173.4 ± 5.6**	159.3 ± 5.0
Body weight, kg	75.5 ± 13.6	78.8 ± 13.5**	67.1 ± 9.9
BMI, kg/m^2	26.2 ± 3.5	26.1 ± 3.6	26.4 ± 3.2
Diabetes n, %	40 (29.4)	27 (27.6)	13 (34.2)
Chronic kidney disease n, %	11 (8.1)	8 (8.2)	3 (7.9)
CAD n, %	27 (19.9)	14 (14.3)*	13 (34.2)
Stroke history n, %	12 (8.8)	5 (5.1)*	7 (18.4)
Smoking history n, %	63 (46.4)	58 (59.2)**	5 (13.2)
Dyslipidemia n, %	69 (50.7)	49 (50.0)	20 (52.6)
Hypertension duration, y	6.7 ± 10.2	4.9 ± 8.1**	11.4 ± 13.2
Uric acid, mmol/L	352.9 ± 88.0	366.8 ± 86.0**	317.4 ± 83.7
Creatinine, μmol/L	83.1 ± 32.4	86.4 ± 32.4	74.6 ± 31.3
Fasting glucose, mmol/L	6.3 ± 2.1	6.2 ± 1.7	6.7 ± 2.9
Cholesterol, mmol/L	4.7 ± 1.1	4.7 ± 1.1	4.6 ± 1.2
Triglyceride, mmol/L	2.1 ± 1.4	2.1 ± 1.3	2.1 ± 1.7
LDL-C, mmol/L	2.8 ± 0.9	2.8 ± 0.8	2.6 ± 1.0
HDL-C, mmol/L	1.1 ± 0.2	1.0 ± 0.2	1.1 ± 0.3

Values are represented as n (%) or mean ± SD.

BMI = body mass index, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol.

* $P < .05$.

** $P < .01$, compared with the women group.

smoking and uric acid levels of males were higher than females, but the incidence of stroke in males was lower (Table 1).

The levels of office BP, ambulatory CABP, and ambulatory BABP were shown in Table 2. The office PP, ambulatory BAPP, and ambulatory CAPP in males were lower than in females. The office DBP, ambulatory brachial DBP, and ambulatory central DBP in males were higher than in females. The central artery AIx, PWV, AASI, and AcASI in females were higher than in males.

The results of echocardiography were shown in Table 3. LVM and LVEDD in males were significantly higher than in females. There were no differences in LVMI between the 2 sexes. Fifteen patients (11%) were considered to have LVH, in which 8 patients were male and 7 were female.

Table 3

Echocardiographic measurements.

	All (n = 136)	Men (n = 98)	Women (n = 38)
LA diameter, mm	35.8 ± 4.8	35.6 ± 4.6	36.2 ± 5.2
LVEDD, mm	46.3 ± 4.3	47.0 ± 4.3*	44.5 ± 3.9
IVST, mm	10.6 ± 1.6	10.7 ± 1.5	10.3 ± 1.6
PWT, mm	10.4 ± 1.2	10.5 ± 1.2	10.2 ± 1.4
LVEF, %	66.1 ± 6.1	65.4 ± 6.6*	67.7 ± 4.2
LVM, g	173.1 ± 48.3	178.7 ± 48.8*	158.7 ± 44.4
LVMI, g/m^2	88.3 ± 25.2	88.3 ± 25.1	88.4 ± 25.6
LVH n, %	15 (11.0%)	8 (8.2%)	7 (18.4%)

Values are represented as n (%), mean ± SD.

IVST = interventricular septal thickness, LA = left atrial, LVEDD = left ventricular end-diastolic diameter, LVEF = left ventricular ejection fraction, LVH = left ventricular hypertrophy, LVM = left ventricular mass, LVMI = left ventricular mass index, PWT = posterior wall thickness.

* $P < .05$.

** $P < .01$, compared with the female.

The distribution of AcASI and AASI was all normal and the correlation between these 2 factors was significant ($r = 0.879$, $P < .001$). The level of AASI was significantly higher than that of AcASI (0.482 ± 0.270 vs 0.422 ± 0.302; $P < .001$). AASI and AcASI changed according to age, height, weight, heart rate (HR), and brachial ambulatory MBP (Fig. 1). Correlation analysis showed that AASI increased with age, ambulatory brachial MBP, and fasting glucose, but decreased with height. AcASI increased with age, ambulatory brachial MBP, and fasting glucose, but decreased with height and HR (Table 4). Correlation analysis showed that the AcASI was significantly associated with and AASI ($r = 0.88$, 95% CI, 0.83–0.91), brachial ambulatory PP ($r = 0.64$, 95% CI, 0.53–0.73), central ambulatory PP ($r = 0.65$, 95% CI, 0.54–0.61), central AIx ($r = 0.49$, 95% CI, 0.35–0.61), and PWV ($r = 0.50$, 95% CI, 0.36–0.61). LVMI was significantly associated with AcASI ($r = 0.34$, 95% CI, 0.18–0.48), AASI ($r = 0.27$, 95% CI, 0.10–0.42), brachial ambulatory PP ($r = 0.44$, 95% CI, 0.29–0.57), central ambulatory PP ($r = 0.34$, 95% CI, 0.18–0.48), central AIx ($r = 0.08$, 95% CI, 0.18–0.48), and PWV ($r = 0.26$, 95% CI, 0.09–0.41).

Logistic regression analysis indicated that AcASI and AASI had substantial collinearity. Thus, AcASI and AASI were respectively

Table 2

Blood pressure, HR, and arteriosclerosis parameters.

	All (n = 136)	Men (n = 98)	Women (n = 38)
Office brachial SBP, mm Hg	137.7 ± 19.5	136.8 ± 20.4	140.1 ± 17.0
Office brachial DBP, mm Hg	82.3 ± 14.2	84.3 ± 13.0*	77.2 ± 16.2
Office brachial PP, mm Hg	55.4 ± 16.0	55.5 ± 15.1**	63.0 ± 16.2
24 h average brachial SBP, mm Hg	123.3 ± 13.7	123.3 ± 13.0	123.2 ± 15.5
24 h average brachial DBP, mm Hg	80.5 ± 10.7	82.3 ± 9.9**	74.3 ± 10.4
24 h average brachial PP, mm Hg	42.5 ± 11.2	40.0 ± 9.5**	48.9 ± 12.9
24 h average aortic SBP, mm Hg	114.7 ± 12.2	115.6 ± 11.8	112.4 ± 13.1
24 h average aortic DBP, mm Hg	82.0 ± 11.1	84.6 ± 10.2**	75.5 ± 10.9
24 h average aortic PP, mm Hg	32.7 ± 7.8	31.0 ± 7.2**	37.0 ± 7.8
HR, bpm	70.1 ± 9.0	71.5 ± 9.1**	66.5 ± 7.8
Central artery AIx, %	24.4 ± 8.5	20.7 ± 6.2**	33.9 ± 5.8
PWV, m/s	8.1 ± 2.0	7.7 ± 1.9**	9.1 ± 1.8
AASI	0.482 ± 0.270	0.452 ± 0.283*	0.559 ± 0.217
AcASI	0.422 ± 0.302	0.377 ± 0.304**	0.536 ± 0.270

Values are represented as n (%), mean ± SD.

AASI = ambulatory artery stiffness index, AcASI = ambulatory central artery stiffness index, AIx = augmentation index, BMI = body mass index, DBP = diastolic pressure, HR = heart rate, PP = pulse pressure, PWV = pulse wave velocity, SBP = systolic pressure.

* $P < .05$.

** $P < .01$, compared with the nontreatment group.

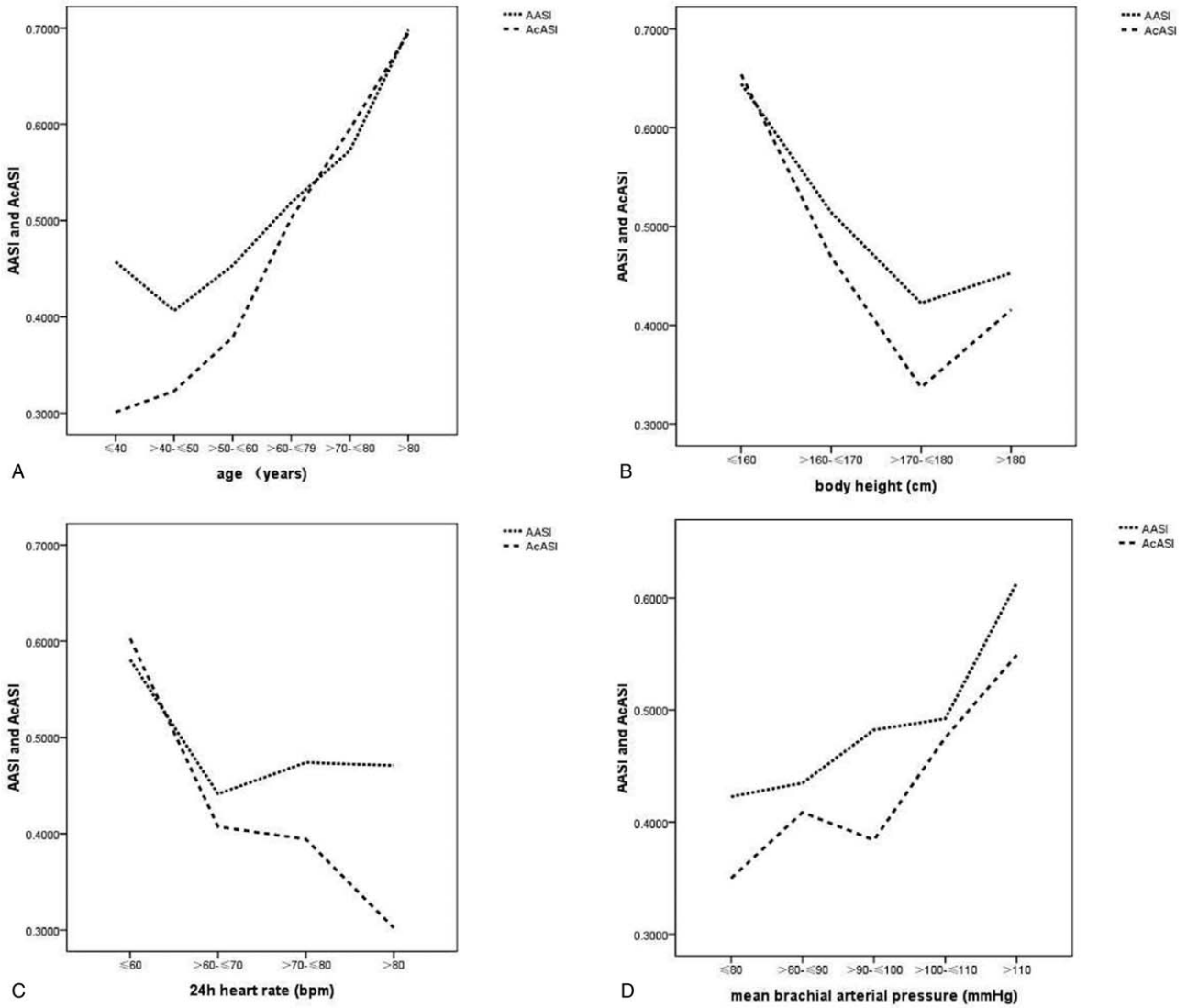

Figure 1. The correlation analysis of AASI and AcASI. The dense dash line indicated AASI and the loose dash line indicated AcASI. AcASI increased with age, ambulatory brachial mean BP and decreased with height and HR. AASI increased with age, ambulatory brachial mean BP and decreased with height. AASI = ambulatory artery stiffness index, AcASI = ambulatory central artery stiffness index, BP = blood pressure, HR = heart rate.

analyzed to determine the LVMI; the data were adjusted for the confounding variables. Age, sex, weight, diabetes, CAD, incidence of stroke, chronic kidney disease, hyperlipidemia, hypertension course, and antihypertensive drug usage were included in the analyses. The results showed that AcASI (B coefficient = 15.883, 95% CI, 1.960–29.806, $P = .027$), chronic kidney disease (B coefficient = 23.793, 95% CI, 8.488–39.098, $P = .003$), and hypertension course (B coefficient = 0.492, 95% CI, 0.029–0.955, $P = .037$) were the independent factors of LVMI, whereas AASI was not (B coefficient = 14.816, 95% CI, −0.803to 30.405, $P = .063$).

Table 4

The correlation between AASI, AcASI, and general clinical data.

	AASI	AcASI
Age	0.242**	0.367**
Body height	−0.216*	−0.258**
Body weight	−0.100	−0.121
Brachial ambulatory mean BP	0.191*	0.181*
HR	−0.051	−0.257**
Fasting glucose	0.220*	0.237*
Uric acid	−0.070	−0.033
Cholesterol	−0.020	−0.111

* $P < .05$.
** $P < .01$.
AASI = ambulatory artery stiffness index, AcASI = ambulatory central artery stiffness index, BP = blood pressure, HR = heart rate.

ROC analysis showed that AASI and AcASI were associated with the LVH (Fig. 2). *Z* test by using Delong method indicated that the discriminatory difference in the area under the curve of AASI and AcASI was 0.755 and 0.807, respectively ($P = .106$). AcASI did not show superiority than AASI.

4. Discussion

Central BP is not consistent with peripheral BP. Different size of arteries with different elasticity may lead to different arterial resistance and blood velocity. The overlapping of reflection wave

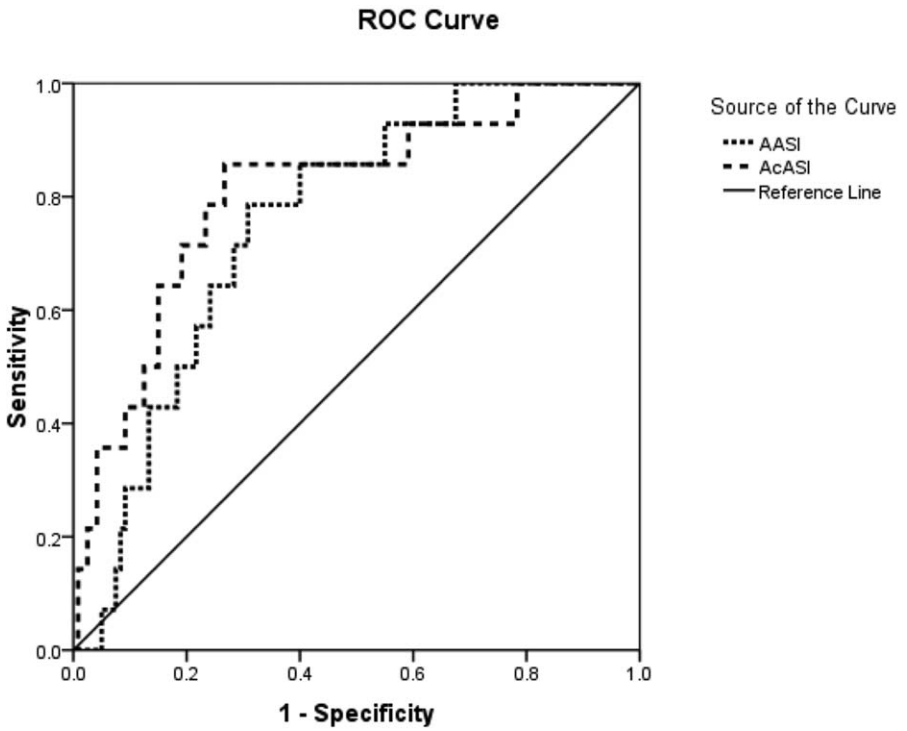

Figure 2. ROC analysis of AASI and AcASI in predicting LVH. ROC analysis evaluating the ability of AASI and AcASI index to predict the presence of LVH. The discriminatory difference in area under the curve of AASI and AcASI was 0.755 and 0.807 respectively, which indicated those 2 curves were not related. AASI = ambulatory artery stiffness index, AcASI = ambulatory central artery stiffness index, LVH = left ventricular hypertrophy.

in branchial artery is earlier than central artery. Therefore, systolic BP and pulse pressure (PP) is gradually increased from central artery to peripheral arteries.

The value of brachial artery pressure as a predictor of future cardiovascular events is well reported. Recently, many reports have investigated ASAI.[13,19,25] For the evaluation of arterial structure and function, PP, PWV, and central artery AIx may also provide insight into the elasticity and stiffness of large arteries.[26] PWV analysis is considered the gold-standard method to evaluate arterial stiffness. PP varies throughout the arterial tree, resulting in a gradient between the central and peripheral pressure. Increasing evidence showed that assessing central pressure may improve the identification and management of patients with a high risk of developing cardiovascular disease.[1,9,13] AcASI could be obtained directly from the same analysis, in addition to PP and PWV. To the best of our knowledge, rare studies on AcASI have been carried out until now.

Factors such as age, HR, height, and nocturnal BP fall influence central pressure. Some studies investigated central pressure and its related factors.[18,19,25] Previous studies show that sex, height, and HR are the factors linked to pulse wave reflection; height is a key factor related to arterial hemodynamic variations between genders.[27] The present study showed that female AcASI was higher than males, but deceased with height and HR. The distance from the reflection wave to the central artery and the time of reflection is shorter in females and the shorter people, and has also been linked to increased central artery AIx. Slow HR leads to the prolongation of the cardiac cycle and left ventricular ejection time, which may cause increased amplitude of overlapping of the reflection wave in the systolic phase. The present study indicated that brachial AASI was not associated with HR. This may be due to slow HR, and augmented central BP and brachial BP. With age, arterial stiffness, the degree of atherosclerosis progression, PWV, augmentation of central artery pressure and AIx will increase. The present study showed that AcASI increased with age and brachial ambulatory MBP, and was significantly associated with ambulatory brachial PP, ambulatory central PP, central Aix, and PWV. These results suggest AcASI as a potential indicator of arterial stiffness.

Many studies have indicated that, apart from cardiac damage, increased AASI is related to a high degree of damage to various target organs[11,12,28] in primary hypertension. Some studies found that AASI also could predict mortality in cardiovascular disease.[6,15] An 8.2-year follow-up study in Portugal found that AASI could predict long-term cardiovascular events and stroke, but not coronary events.[12] However, no studies into AcASI and its predictive value in cardiovascular disease have been conducted. Therefore, whether AcASI could predict short- or long-term or cardiac events require further study.

LVH is a common complication of hypertension. The association between LVH and AcASI was explored in our study. The results showed that AcASI and AASI were significantly associated with LVH. On the contrary, regression analysis showed that AcASI was significantly associated with LVMI, but AASI was not. Thus, we proposed that the majority of our participants who received antihypertensive treatment that may be a considered as confounding variable, which led to false negative results of LVH.

The limitations of this study are considered as follows. Firstly, the sample size was relatively small. Secondly, there was a notable difference in age between the gender groups. Thirdly, short- and

long-term follow-up analyses were not carried out. Finally, the majority of participants were administered antihypertensive drugs, which may be impact on CABP and BABP. Therefore, further study is needed in the future.

5. Conclusion

In conclusion, the present study proposed that AcASI, which is derived from ambulatory CABP, could be a new marker of arterial stiffness. AcASI may be more related with LVH than AASI. However, further study is required in the future.

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Author contributions

Conceptualization: Xuezhai Zeng, Deping Liu, Yu Gan.
Data curation: Zhujin Xu, Yan Zhang, Ruisheng Zhang, Jihong Zhou, Chunyu Pan, Weiwei Qiao.
Formal analysis: Xuezhai Zeng.
Methodology: Na Jia, Hua Wang, Ruisheng Zhang.
Project administration: Xuezhai Zeng.
Resources: Lijuan Wang.
Software: Xuezhai Zeng, Na Jia.
Supervision: Deping Liu.
Visualization: Lijuan Wang.
Writing – original draft: Xuezhai Zeng.
Writing – review and editing: Na Jia.
Deping Liu orcid: 0000-0001-5887-4120.

References

[1] Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. J Am Coll Cardiol 2010;55:1318–27.
[2] Mancia G, De Backer G, Dominiczak A, et al. 2007 Guidelines for the management of arterial hypertension: the Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Eur Heart J 2007;28:1462–536.
[3] Li Y, Wang JG, Dolan E, et al. Ambulatory arterial stiffness index derived from 24-hour ambulatory blood pressure monitoring. Hypertension 2006;47:359–64.
[4] Leoncini G, Ratto E, Viazzi F, et al. Increased ambulatory arterial stiffness index is associated with target organ damage in primary hypertension. Hypertension 2006;48:397–403.
[5] Chen HH, Sun YS, Liu ML. [Association between ambulatory arterial stiffness index with left ventricular mass index in the elderly hypertensive patients]. Zhonghua Xin Xue Guan Bing Za Zhi 2016;44:750–3.
[6] Garcia-Garcia A, Gomez-Marcos MA, Recio-Rodriguez JI, et al. Relationship between ambulatory arterial stiffness index and subclinical target organ damage in hypertensive patients. Hypertens Res 2011;34:180–6.
[7] Kalaycioglu E, Gokdeniz T, Aykan AC, et al. Ambulatory arterial stiffness index is associated with impaired left atrial mechanical functions in hypertensive diabetic patients: A speckle tracking study. Anatol J Cardiol 2015;15:807–13.
[8] Dolan E, Thijs L, Li Y, et al. Ambulatory arterial stiffness index as a predictor of cardiovascular mortality in the Dublin Outcome Study. Hypertension 2006;47:365–70.
[9] Hansen TW, Li Y, Staessen JA, et al. Independent prognostic value of the ambulatory arterial stiffness index and aortic pulse wave velocity in a general population. J Hum Hypertens 2008;22:214–6.
[10] Bastos JM, Bertoquini S, Polonia J. Prognostic significance of ambulatory arterial stiffness index in hypertensives followed for 8.2 years: its relation with new events and cardiovascular risk estimation. Rev Port Cardiol 2010;29:1287–303.
[11] Xu TY, Li Y, Wang YQ, et al. Association of stroke with ambulatory arterial stiffness index (AASI) in hypertensive patients. Clin Exp Hypertens 2011;33:304–8.
[12] Kilinc F, Pekkolay Z, Demircan F, et al. Association of clinical and laboratory parameters with ambulatory arterial stiffness index in acromegaly patients. Pak J Med Sci 2018;34:37–42.
[13] McEniery CM, Yasmin , McDonnell B, et al. Central pressure: variability and impact of cardiovascular risk factors: the Anglo-Cardiff Collaborative Trial II. Hypertension 2008;51:1476–82.
[14] Protogerou AD, Argyris AA, Papaioannou TG, et al. Left-ventricular hypertrophy is associated better with 24-h aortic pressure than 24-h brachial pressure in hypertensive patients: the SAFAR study. J Hypertens 2014;32:1805–14.
[15] Weber T, Wassertheurer S, Schmidt-Trucksass A, et al. Relationship between 24-hour ambulatory central systolic blood pressure and left ventricular mass: a prospective multicenter study. Hypertension 2017;70:1157–64.
[16] Oh JY, Allison MA, Barrett-Connor E. Different impacts of hypertension and diabetes mellitus on all-cause and cardiovascular mortality in community-dwelling older adults: the Rancho Bernardo Study. J Hypertens 2017;35:55–62.
[17] Liu LS. [2010 Chinese guidelines for the management of hypertension]. Zhonghua Xin Xue Guan Bing Za Zhi 2011;39:579–615.
[18] Wassertheurer S, Kropf J, Weber T, et al. A new oscillometric method for pulse wave analysis: comparison with a common tonometric method. J Hum Hypertens 2010;24:498–504.
[19] Wilkinson IB, McEniery CM, Cockcroft JR. Central blood pressure estimation for the masses moves a step closer. J Hum Hypertens 2010;24:495–7.
[20] Berukstis A, Jarasunas J, Daskeviciute A, et al. How to interpret 24-h arterial stiffness markers: comparison of 24-h ambulatory Mobil-O-Graph with SphygmoCor office values. Blood Press Monit 2019;24:93–8.
[21] Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440–63.
[22] Sun NL, Chen JW, Wang JG, et al. Expert consensus on diagnosis and treatment of hypertension complicated with left ventricular hypertrophy in Asia. Chin J Hypertens 2016;26:619–27.
[23] Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450–8.
[24] 2013 Practice guidelines for the management of arterial hypertension of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC): ESH/ESC Task Force for the Management of Arterial Hypertension. J Hypertens 2013;31:1925–38.
[25] Schillaci G, Pucci G. Is ambulatory arterial stiffness index a marker of large-artery stiffness? Evidence from intervention studies. Hypertens Res 2015;38:799–801.
[26] Butlin M, Qasem A. Large Artery Stiffness Assessment Using SphygmoCor Technology. Pulse (Basel) 2017;4:180–92.
[27] Huang P, Liang YL, Xu Y, et al. Height is a key determinant in arterial function in older aged subjects. Chin J Med Guide 2010;12:735–9.
[28] Wohlfahrt P, Krajcoviechova A, Jozifova M, et al. Large artery stiffness and carotid flow pulsatility in stroke survivors. J Hypertens 2014;32:1097–103.