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A Comparison of Oscillometrically Measured Ankle-to-Brachial Mean Arterial Pressure Ratio and Ankle-Brachial Index in Predicting Cardiovascular Events and All-Cause Mortality

ABSTRACT

Background: The oscillometrically measured ankle-brachial index (omABI), which is determined by the ratio of ankle to brachial systolic blood pressure measured through oscillography, has been demonstrated as a robust predictor of cardiovascular events. However, the reliability of mean arterial pressure measured by oscillography may be higher than that of systolic blood pressure based on the principle of oscillographic oscillation. We aimed to compare the predictive value of oscillometrically measured ankle-to-brachial mean arterial pressure ratio (omMAPR) and omABI for cardiovascular events and all-cause mortality.

Methods: The observation cohort consisted of a total of 37 803 employees from the Chinese Kailuan Group who underwent limb blood pressure measurements during their participation in physical examination between 2010 and 2017.

Results: After an average follow-up period of 3 years, a total of 589 cardiovascular events and 570 cases of all-cause mortality were observed. The predictive performance of omMAPR was found to be slightly superior to omABI in terms of cardiovascular events (*C*-statistics: 0.55 vs. 0.51, $P < .001$) and all-cause mortality (*C*-statistics: 0.60 vs. 0.55, $P < .001$). After adjusting for confounders, within a specific range (omMAPR ≤ 1.06 or omABI ≤ 1.12), each 0.1-unit increase in omMAPR was associated with reductions of 14% (HR = 0.86, 95% CI: 0.77-0.96) and 23% (HR = 0.77, 95% CI: 0.70-0.84) in cardiovascular events and all-cause mortality, respectively, while each 0.1-unit increase in omABI was associated with reductions of 12% (HR = 0.88, 95% CI: 0.79-0.97) and 22% (HR = 0.78, 95% CI: 0.72-0.85) in cardiovascular events and all-cause mortality, respectively. However, once out of that range (omMAPR > 1.06 or omABI > 1.12), neither omMAPR nor omABI was significantly associated with cardiovascular events or all-cause mortality.

Conclusion: Both omMAPR and omABI within specific ranges (omMAPR ≤ 1.06 or omABI ≤ 1.12) were independent predictors for cardiovascular events and all-cause mortality. Moreover, omMAPR exhibited a slightly superior predictive ability compared to omABI in relation to cardiovascular events and all-cause mortality. The trial registration number is ChiCTR-TNRC-11001489.

Keywords: Ankle-to-brachial mean arterial pressure ratio, ankle-brachial index, cardiovascular events, all-cause mortality

INTRODUCTION

The ankle-brachial index (ABI) is a reliable and noninvasive indicator used to assess the extent of atherosclerosis in the lower limbs, calculated by comparing the systolic blood pressure (SBP) at the ankle with that at the brachial artery.[1] However, the presence of atherosclerosis is a systemic lesion, and it has also been demonstrated that a reduced ABI significantly elevates the risk of cardiovascular events and all-cause mortality. In a meta-analysis comprising 43 prospective cohort studies, an ABI below 0.9 was associated with a 152% increased risk of all-cause mortality, a 194% increased risk of cardiovascular mortality, a 117% increased risk of cerebrovascular events, and a 128% increased risk of myocardial infarction when compared to individuals with a normal ABI range of 0.9-1.3.[2]

ORIGINAL INVESTIGATION

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The oscillometric method is currently the primary approach for obtaining ABI, which involves synchronizing blood pressure measurements of the limbs. However, unlike the mercury sphygmomanometer, which estimates systolic blood pressure (SBP) and diastolic blood pressure (DBP) by identifying the onset and disappearance of Korotkoff sounds, oscillometry calculates blood pressure based on the principle of oscillographic oscillation, where the peak point of oscillation corresponds to mean arterial pressure (MAP). Systolic blood pressure and DBP are estimated by a specific formula based on patent protection.[3] Therefore, the reliability of MAP measured by oscillography may be higher when compared to SBP and DBP. The purpose of this study was to compare the predictive value of oscillometrically measured ankle-to-brachial mean arterial pressure ratio (omMAPR) and oscillometrically measured ABI (omABI) for cardiovascular events and all-cause mortality.

METHODS

Detailed information on the Kailuan Study (The trial registration number: ChiCTR-TNRC-11001489; Date of registration: 30 August 2011) has been described previously.[4] In brief, the Kailuan study, which includes 101510 Chinese adults (81110 men and 20 400 women) aged 18-98 years, was established when detailed information on health status and lifestyle was collected in 2006-2007. Participants receive a health examination biennially to update information on potential risk factors and to ascertain newly diagnosed diseases. Of those, 37 869 participants underwent synchronized blood pressure measurement of the limbs between 2010 and 2017. After an exclusion of those who did not participate in any health examination (n = 66), the remaining 37 803 participants were included in the final statistical analysis.

The authors declare that they do not use artificial intelligence (AI)-assisted technologies (such as large language models, chatbots, or image creators) in the production of submitted work.

The methods of questionnaire surveys, anthropometric measurements, blood pressure measurements, and blood sample detection were referred to the published literature[4] of our research group.

HIGHLIGHTS

- omMAPR within specific ranges (≤1.06) was an independent predictor for cardiovascular events and all-cause mortality.
- omABI within specific ranges (≤1.12) was an independent predictor for cardiovascular events and all-cause mortality.
- omMAPR exhibited a slightly superior predictive ability compared to omABI for both cardiovascular events and all-cause mortality.
- Once out of the range (omMAPR > 1.06 or omABI > 1.12), neither omMAPR nor omABI was associated with cardiovascular events or all-cause mortality.

The BP-203RPEIII network arteriosclerosis detection device (Omron Health Medical Co. Ltd., Dalian, China) was utilized for simultaneous measurement of bilateral ankle and brachial blood pressure, including MAP. The ambient temperature was maintained within the range of 22-25°C. The participants reclined on a flat bed. The blood pressure cuffs were applied to the upper arms and ankles of the lower limbs. The airbag marker of the upper arm cuff was aligned with the brachial artery, and the bottom of the cuff was positioned 2-3 cm away from the elbow joint. The lower extremity cuff airbag sign was located on the medial side of the lower extremity, and the lower cuff margin was 1-2 cm distant from the medial malleolus. After a minimum of 5 minutes of rest in the supine position, the measurements were conducted. The measurements were replicated, and the subsequent value was adopted as the ultimate outcome.[5] The omABI was automatically derived by calculating the ankle-to-brachial systolic blood pressure ratio. Similarly, the omMAPR was determined by computing the ankle-to-brachial mean arterial pressure ratio. In this study, the minimum value of omABI and omMAPR from both sides was used for analysis.

The primary outcomes included a composite endpoint of myocardial infarction, cerebral infarction, and cerebral hemorrhage, collectively referred to as cardiovascular events. Additionally, all-cause death was considered another primary outcome. The initiation of follow-up was determined by the time of synchronized blood pressure measurement. Follow-up continued until December 31, 2017, or until the occurrence of cardiovascular events or death. Outcome events were identified through the government health care system and further confirmed by trained researchers who reviewed medical records from hospitals every 6 months.[4,6]

Incident myocardial infarction was diagnosed according to the criteria of the fourth universal definition based on clinical symptoms, changes in the serum concentrations of cardiac enzymes and/or biomarkers, and electrocardiogram results.[7] Stroke was diagnosed according to the World Health Organization criteria, based on symptoms, clinical signs, images obtained by computed tomography or magnetic resonance imaging, and other diagnostic reports.[8] Hypertension was defined as SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg, or a history of hypertension, or taking antihypertensive agents.[9] Diabetes was defined as fasting blood glucose ≥ 7.0 mmol/L, or a history of diabetes, or taking antidiabetic agents.[10]

Statistical analysis was performed using the SAS software (Version 9.2, SAS Institute, Cary, NC). All statistical tests were two-sided, and $P < .05$ was considered statistically significant. The Kolmogorov–Smirnov method was used to test normality for continuous variables. Continuous variables with a normal distribution were expressed as the mean and standard deviation. The serum concentrations of the C-reactive protein and triglyceride were expressed as median and quartiles due to their skewed distribution. Categorical variables were described as percentages. The C-statistics of omMAPR and omABI were computed and compared for the prediction of cardiovascular events and all-cause mortality.

The non-linear relationships between omMAPR and omABI and the risk of cardiovascular events and all-cause mortality were investigated by employing restricted cubic spline curves after adjusting for age, sex, alcohol consumption status [never and past, or current (i.e., ≥once/day)], smoking status [never and past, or current (i.e., ≥once/day)], physical exercise [none, occasionally, or frequently (i.e., ≥once/week)], family history of cardiovascular disease (yes/no), history of myocardial infarction (yes/no), history of stroke (yes/no), SBP, body mass index, and serum concentrations of total cholesterol, high-density lipoprotein cholesterol, glucose, uric acid, and C-reactive protein. Multiple Cox proportional hazard models were used to estimate hazard ratios and 95% CI of cardiovascular events and all-cause mortality for every 0.1-unit increase in omMAPR and omABI within the defined ranges after adjusting for the above confounders.

RESULTS

A total of 37 803 employees were included in the final statistical analysis, with an average age of 48.4 ± 12.6 years, and males accounted for 72.4% of the sample. The mean values of omABI and omMAPR at baseline were recorded as 1.09 ± 0.10 and 0.99 ± 0.08, respectively. The prevalence of hypertension, diabetes, myocardial infarction, and stroke was 38.7%, 14.4%, 0.99%, and 1.88%, respectively. The baseline presentation of the means or percentages for other covariates is displayed in Table 1.

After an average follow-up period of 3 years, a total of 589 cases of cardiovascular events and 570 cases of all-cause mortality events were observed. The results presented in Table 2 demonstrated that the baseline omMAPR exhibited slightly superior *C*-statistics compared to omABI for predicting both cardiovascular events and all-cause mortality (cardiovascular events: 0.55 vs. 0.51, $P < .001$; all-cause mortality: 0.60 vs. 0.55, $P < .001$).

Restricted cubic spline analysis revealed a J-shaped relationship between omMAPR and both cardiovascular events and all-cause mortality, even after adjusting for age, sex, and other potential confounding factors. Before approaching the inflection point (omMAPR = 1.06) of the curve, a gradual decline in the risk of cardiovascular events and all-cause mortality was observed with baseline omMAPR. However, no significant association was observed between omMAPR and cardiovascular events or between omMAPR and all-cause mortality beyond that inflection point. Similarly, a J-shaped curve was observed in the association between omABI and cardiovascular events as well as all-cause mortality. The risk of cardiovascular events or all-cause mortality gradually decreased with baseline omABI until reaching an inflection point at omABI equal to 1.12. However, beyond this inflection point, there was no significant association between baseline omABI and the occurrence of cardiovascular events or all-cause mortality (Figure 1).

After adjusting for the aforementioned confounders, multivariate Cox regression analysis revealed that each 0.1-unit increase in omMAPR was associated with a 14% decrease in the risk of cardiovascular events [hazard ratio (HR) = 0.86, 95% CI: 0.77-0.96)] and a 23% decrease in the risk of all-cause mortality (HR = 0.77, 95% CI: 0.70-0.84) among participants with an omMAPR equal to or below 1.06 (Table 3).

Participants with an omMAPR equal to or below 1.06 were further categorized into 4 subgroups based on their omMAPR values: <0.80, 0.80-0.89, 0.90-0.99, and 1.00-1.06. The incidence rate of cardiovascular events and all-cause mortality exhibited a gradual increase in association with decreasing baseline omMAPR. After adjusting for the aforementioned confounding factors, participants with an omMAPR below 0.80 exhibited a 78% increased risk of cardiovascular events (HR = 1.78, 95% CI: 1.13-2.81) compared to those with an omMAPR ranging from 1.00 to 1.06. However, there was no significant increase in the risk of cardiovascular events among individuals with an omMAPR between 0.80 and 0.89 or between 0.90 and 0.99. The risk for all-cause mortality increased by 168% (HR = 2.68, 95% CI: 1.83-3.92), 47% (HR = 1.47, 95% CI: 1.06-2.03), and 33% (HR = 1.33, 95% CI: 1.07-1.66) in participants with an omMAPR below 0.80, between the range of 0.80 and 0.89, and between the range of 0.90 and 0.99, respectively, when compared to those with an omMAPR between the range of 1.00 and 1.06 (Table 4).

Table 1. Baseline Characteristics

Variables	Values
Age (years)	48.4 ± 12.6
Men	27 365 (72.4%)
omABI	1.09 ± 0.10
omMAPR	0.99 ± 0.08
Systolic blood pressure (mm Hg)	131 ± 19.0
Diastolic blood pressure (mm Hg)	82.1 ± 10.9
Body mass index (kg/m^2)	25.0 ± 3.44
Current smoker	11 867 (31.4%)
Current drinker	10 184 (26.9%)
Exerciser	21 253 (56.2%)
Family history of cardiovascular disease	1 076 (2.85%)
History of myocardial infarction	376 (0.99%)
History of stroke	712 (1.88%)
Hypertension	14 646 (38.7%)
Diabetes mellitus	5 455 (14.4%)
Total cholesterol (mmol/L)	4.93 ± 1.47
Triglyceride (mmol/L)	1.29 (0.87-2.05)
Low density lipoprotein (mmol/L)	2.73 ± 1.06
High density lipoprotein (mmol/L)	1.47 ± 0.77
Fasting blood glucose (mmol/L)	5.85 ± 2.14
Serum uric acid (μmol/L)	316 ± 95.8
C-reactive protein (mg/L)	1.00 (0.43-2.20)

Table 2. The *C*-Statistics (95% CIs) of omMAPR and omABI

Outcome	omMAPR	omABI	*P*
Cardiovascular event	0.55 (0.53-0.57)	0.51 (0.48-0.53)	<.001
All-cause mortality	0.60 (0.58-0.62)	0.55 (0.52-0.57)	<.001

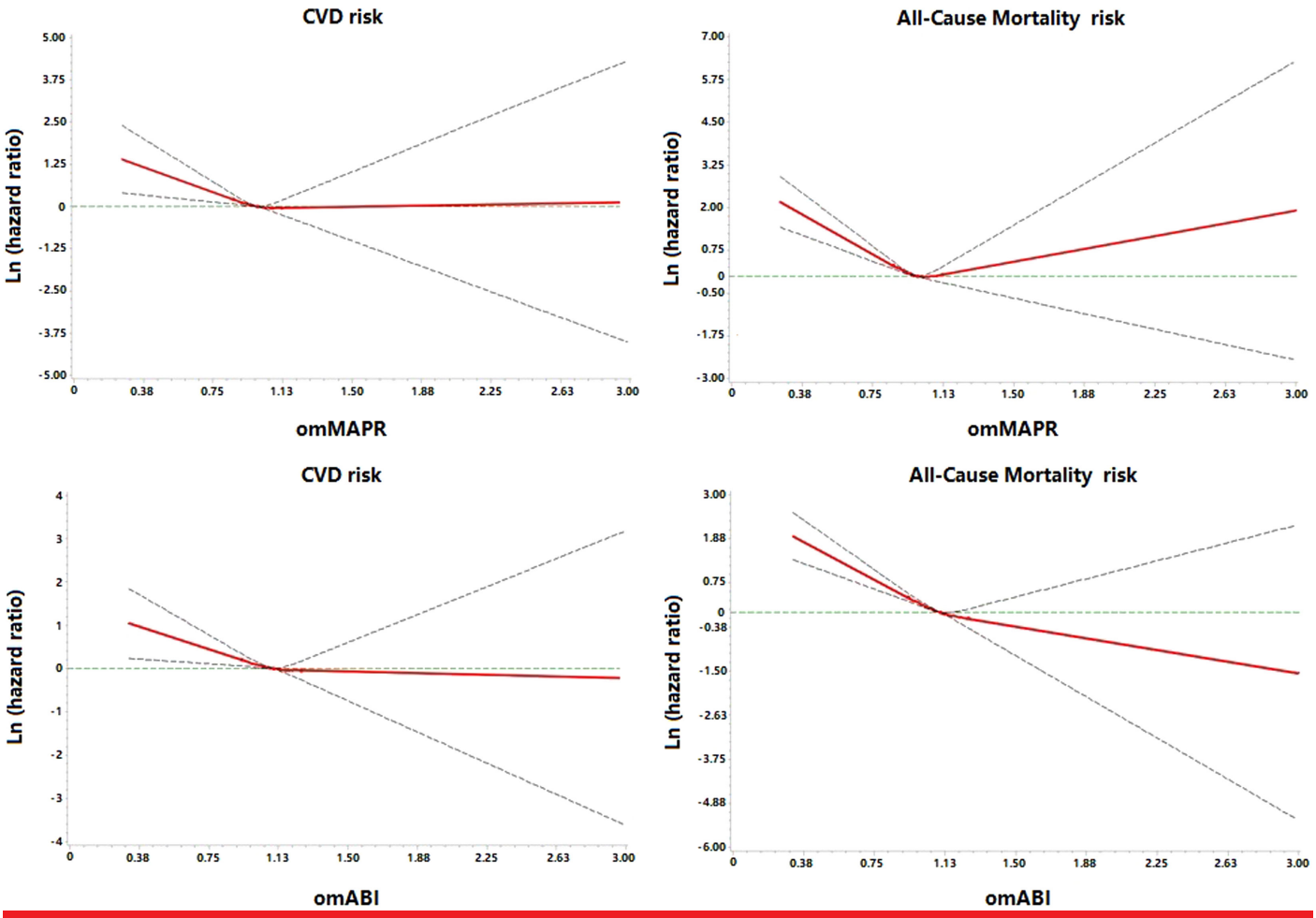

Figure 1. Adjusted restricted cubic spline for the hazard of an incident cardiovascular event and all-cause death by omMAPR (A, B) and omABI (C, D). Figure 1A and Figure 1B are the adjusted restricted cubic splines for the hazards of incident cardiovascular events and all-cause death by omMAPR. It showed a J-shaped curve for the relationship between omMAPR and cardiovascular events and between omMAPR and all-cause death after adjustment for age, sex, alcohol consumption status, smoking status, physical exercise, family history of cardiovascular disease, history of myocardial infarction, history of stroke, SBP, body mass index, and serum concentrations of total cholesterol, high-density lipoprotein cholesterol, glucose, uric acid, and C-reactive protein. Before the inflection point (omMAPR = 1.06) of the curve, the risk of cardiovascular events and all-cause death decreased gradually with the increase of omMAPR, but there was no significant association between omMAPR and cardiovascular events and between omMAPR and all-cause death after that inflection point. (C, D) It also showed a J-shaped curve for the relationship between omABI and cardiovascular events and between omABI and all-cause death after adjustment for the above confounders. The risk of cardiovascular events and all-cause death gradually declined with the increase of omABI prior to the inflection point (omABI = 1.12), but there was no significant association between omABI and cardiovascular events and between omABI and all-cause death after the inflection point.

After adjusting for the aforementioned confounding factors, each 0.1-unit increase in omABI was found to be associated with a 12% reduction in the risk of cardiovascular events (HR = 0.88, 95% CI: 0.79-0.97) and a 22% reduction in the risk of all-cause mortality (HR = 0.78, 95% CI: 0.72-0.85) among participants with an omABI equal to or below 1.12 (Table 3).

Participants with an omABI equal to or below 1.12 were further categorized into 4 subgroups based on their omABI values: <0.80, 0.80-0.89, 0.90-0.99, and 1.00-1.12. The incidence rate of cardiovascular events and all-cause mortality exhibited a gradual increase in association with decreasing baseline omABI. After adjusting for the aforementioned confounding factors, participants with an omABI below 0.80 exhibited a 68% increased risk of cardiovascular events (HR = 1.68, 95% CI: 1.03-2.75) compared to those with an omABI ranging from 1.00 to 1.12. However, there was no significant increase in the risk of cardiovascular events among individuals with omABI values between 0.80 and 0.89 or between 0.90 and 0.99. Compared to participants with an omABI ranging from 1.00 to 1.12, those with an omABI below 0.80 and between 0.80 and 0.89 exhibited a significantly elevated risk of all-cause mortality, with HRs (95% CI) of 2.67 (1.86-3.84) and 1.84 (1.15-2.94), respectively (Table 5).

Table 3. The Hazard Ratios (HRs) and 95% CIs of Cardiovascular Event and All-cause Mortality for Each 0.1-Unit Increase in omMAPR and omABI

	omMAPR ≤ 1.06	omABI ≤ 1.12
Cardiovascular event		
Case / n	524 / 32 213	357 / 23 580
HR (95% CI)[§]	0.86 (0.77-0.96)**	0.88 (0.79-0.97)*
All-cause mortality		
Case / n	520 / 32 213	366 / 23 580
HR (95% CI)[§]	0.77 (0.70-0.84)**	0.78 (0.72-0.85)**

[§]Adjusted for age, sex, systolic blood pressure, body mass index, smoking status [never and past, or current (i.e., ≥ once/day)], alcohol consumption status [never and past, or current (i.e., ≥ once/day)], physical exercise [none, occasionally, or frequently (i.e., ≥ once/week)], family history of cardiovascular disease (yes/no), history of myocardial infarction (yes/no), history of stroke (yes/no), total cholesterol, high-density lipoprotein, uric acid, and C-reactive protein. Compared with reference, **P < .01, *P < .05.

DISCUSSION

In this study, the predictive value of both omMAPR and omABI for cardiovascular events and all-cause mortality was observed, with omMAPR demonstrating a slightly superior performance compared to omABI. Within a specific range (omMAPR ≤ 1.06 or omABI ≤ 1.12), each 0.1-unit increase in omMAPR was associated with reductions of 14% and 23% in cardiovascular events and all-cause mortality, respectively, while each 0.1-unit increase in omABI was associated with reductions of 12% and 22% in cardiovascular events and all-cause mortality, respectively. The subgroup analysis also revealed that participants with an omMAPR <0.8 had a 78% increased risk of cardiovascular events and a 168% increased risk of all-cause mortality compared to those with an omMAPR between 1.00 and 1.06. Similarly, participants with an omABI <0.8 had a 68% increased risk of cardiovascular events and a 167% increased risk of all-cause mortality compared to those with an omABI between 1.00 and 1.12.

The findings of previous studies have consistently demonstrated that an ABI below 0.90 is significantly associated with an elevated risk of cardiovascular events and mortality, even when upper limits for a normal ABI are not specified.[11-15] However, in contrast to prior research, the results of this study provide further evidence that omMAPR slightly outperforms omABI in predicting both cardiovascular events and all-cause mortality.

Additionally, no significant associations were observed between either omMAPR or omABI and the incidence of cardiovascular events or all-cause mortality beyond the inflection point in this particular study. Conversely, the results from the Strong Heart Study, which followed 4393 participants for 8 years, showed that compared with a normal ABI (0.90-1.40), a high ABI (>1.40) was still associated with a 77% (HR = 1.77, 95% CI: 1.48-2.13) increased risk of all-cause mortality and a 109% (HR = 2.09, 95% CI: 1.49-2.94) increased risk of cardiovascular mortality.[16] The study included American Indian participants, who were older and exhibited a significantly higher prevalence of hypertension, diabetes, and obesity. These conditions are known to contribute to increased arterial incompressibility, resulting in a pseudo-elevation of ABI. Therefore, a higher ABI than normal may, to some extent, represent the presence of these risk factors, which may account for the increased cardiovascular risk. While the prevalence of hypertension, diabetes, and obesity in our study was comparable to that observed in the general population, thereby minimizing potential confounding effects. Moreover, it is worth noting that the duration of follow-up for this study was limited to 3 years, which might have led to an underestimation of the results.

In addition to omABI and omMAPR, the ankle's percentage of MAP (%MAP) and upstroke time (UT) have also been reported to exhibit superior predictive accuracy for both peripheral arterial disease (PAD) and all-cause mortality compared to ABI.[17,18] Moreover, the combination of high ankle %MAP (>45%) and low ABI (<0.9) has also been validated to enhance the predictive accuracy for both PAD and all-cause mortality.[19,20] Theoretically, obtaining omMAPR is easier compared to %MAP and UT. However, the majority of electronic blood pressure monitors currently available for purchase only

Table 4. The Hazard Ratios (HRs) and 95% CIs of Cardiovascular Event and All-cause Mortality for Different omMAPR Groups in the Range of Less Than or Equal to 1.06

	omMAPR			
	<0.80	0.80-0.89	0.90-0.99	1.00-1.06
Cardiovascular event				
Case / n	26 / 556	58 / 3206	282 / 16 827	158 / 11 624
Incidence / 1000 person-years	16.2	6.33	5.35	4.28
HR (95% CI)[§]	1.78 (1.13-2.81)*	1.19 (0.86-1.65)	1.14 (0.92-1.39)	Ref.
All-cause mortality				
Case / n	45 / 556	64 / 3206	276 / 16 827	135 / 11 624
Incidence / 1000 person-years	27.0	6.92	5.18	3.62
HR (95% CI)[§]	2.68 (1.83-3.92)**	1.47 (1.06-2.03)*	1.33 (1.07-1.66)*	Ref.

[§]Adjusted for age, sex, systolic blood pressure, body mass index, smoking status [never and past, or current (i.e., ≥ once/day)], alcohol consumption status [never and past, or current (i.e., ≥ once/day)], physical exercise [none, occasionally, or frequently (i.e., ≥ once/week)], family history of cardiovascular disease (yes/no), history of myocardial infarction (yes/no), history of stroke (yes/no), total cholesterol, high-density lipoprotein, uric acid, and C-reactive protein.
Compared with reference, **P < .01, *P < .05.

Table 5. The Hazard Ratios (HRs) and 95% CIs of Cardiovascular Event and All-cause Mortality for Different omABI Groups in the Range of Less Than or Equal to 1.12

	omABI			
	<0.80	**0.80-0.89**	**0.90-0.99**	**1.00-1.12**
Cardiovascular event				
Case / n	21 / 369	14 / 748	74 / 4194	248 / 18 269
Incidence / 1000 person-years	20.9	6.35	6.02	4.27
HR (95% CI)[§]	1.68 (1.03-2.75)*	1.15 (0.65-2.01)	1.24 (0.94-1.65)	Ref.
All-cause mortality				
Case / n	44 / 556	23 / 748	64 / 4194	235 / 18 269
Incidence / 1000 person-years	41.8	10.4	5.15	4.01
HR (95% CI)[§]	2.67 (1.86-3.84)**	1.84 (1.15-2.94)*	1.23 (0.91-1.65)	Ref.

[§]Adjusted for age, sex, systolic blood pressure, body mass index, smoking status [never and past, or current (i.e., ≥ once/day)], alcohol consumption status [never and past, or current (i.e., ≥ once/day)], physical exercise [none, occasionally, or frequently (i.e., ≥ once/week)], family history of cardiovascular disease (yes/no), history of myocardial infarction (yes/no), history of stroke (yes/no), total cholesterol, high-density lipoprotein, uric acid, and C-reactive protein. Compared with reference, $^{**}P < .01$, $^{*}P < .05$.

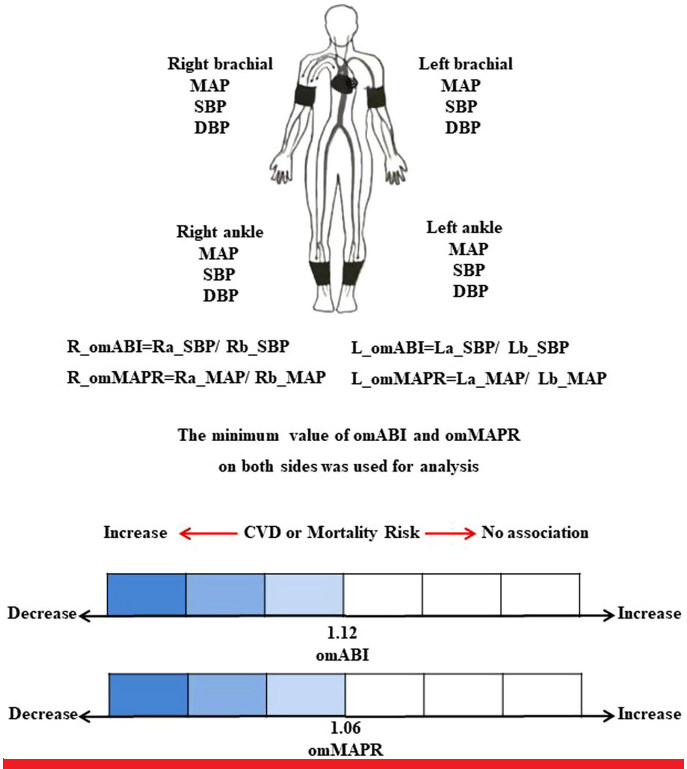

Figure 2. Measurements, flow chart, and main results of the study. The omABI was derived by calculating the ankle-to-brachial systolic blood pressure ratio and the omMAPR was determined by computing the ankle-to-brachial mean arterial pressure ratio. The minimum value of omABI and omMAPR on both sides was used for analysis. The main results of the study showed that within a specific range (omMAPR ≤ 1.06 or omABI ≤ 1.12), the risk of cardiovascular events and all-cause mortality exhibited a gradual increase in association with decreasing baseline omMAPR or omABI. However, once out of that range (omMAPR > 1.06 or omABI > 1.12), neither omMAPR nor omABI was associated with cardiovascular events or all-cause mortality.

provide SBP and DBP measurements, lacking the capability to measure MAP. Consequently, the clinical application of omMAPR may be limited due to its reliance on synchronous extremities blood pressure measuring devices.

The strengths of our study included its prospective cohort study design, the implementation of standardized data collection protocols, the utilization of a large sample size, and the achievement of almost complete follow-up for cardiovascular events and all-cause mortality. This was made possible by the Municipal Social Insurance's comprehensive collection of medical records encompassing the entire population in the Kailuan community. In addition, more potential confounders, such as health behaviors, history of cardiovascular disease, and biochemical indexes, were included in the multivariable analysis, which made the results more credible.

Study Limitations

The limitations of our study should be discussed. First, the study participants were limited to the employees of Kailuan Group, in which male accounted for three-quarters, so the generalizability of the findings to other populations should be interpreted with caution. Second, the relatively limited duration of the follow-up period may result in an underestimation of the findings. Third, due to the limited availability of relevant data on PAD, we encountered challenges in assessing the predictive value of omMAPR and omABI in patients with PAD. Fourth, the baseline parameters such as history of malignancy, chronic inflammatory diseases, reproductive system abnormalities, and social support are also important to determine cardiovascular risk. However, because these data were not available in this study, we could not perform relevant adjustments or subgroup analyses.

CONCLUSION

In conclusion, both omMAPR and omABI within specific ranges (omMAPR ≤ 1.06 or omABI ≤ 1.12) emerged as independent predictors for cardiovascular events and all-cause mortality. Moreover, omMAPR exhibited a slightly superior

predictive ability compared to omABI in relation to cardiovascular events and all-cause mortality. Finally, the measurement of omMAPR and omABI with the main results of this study can be helpful in screening individuals at high cardiovascular risk (Figure 2).

Ethics Committee Approval: The study was performed according to the guidelines of the Helsinki Declaration and was approved jointly by the Ethics Committee of the Kailuan General Hospital (Decision number: [2006] Approval No. 5; Decision date: January 5 2006).

Informed Consent: Written informed consent was obtained from all the participants. All methods were performed in accordance with the relevant guidelines.

Peer-review: Externally peer-reviewed.

Author Contributions: Design and revision of the work – W.C.; Acquisition and analysis – Z.H., C.Z.; Interpretation of data – S.W.; Drafting the work – C.J. All authors read and approved the final manuscript.

Declaration of Interests: The authors have no conflicts of interest to declare.

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